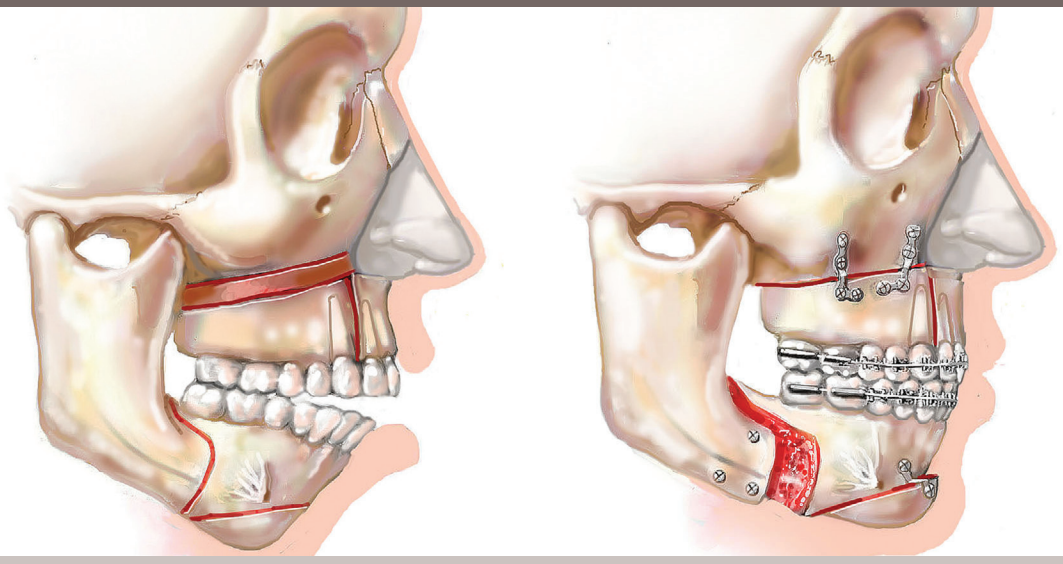
JEFFREY C. POSNICK, DMD, MD

ORTHOGNATHIC SURGERY:

PRINCIPLES & PRACTICE

JEFFREY C. POSNICK, DMD, MD



ORTHOGNATHIC SURGERY:
PRINCIPLES & PRACTICE

JEFFREY C. POSNICK, DMD, MD
Clinical Professor of Surgery and Pediatrics
Georgetown University
Washington, DC

Clinical Professor of Orthodontics
University of Maryland, School of Dentistry
Baltimore, Maryland

Adjunct Professor of Oral and Maxillofacial Surgery
Howard University College of Dentistry
Washington, DC

Director, Posnick Center for Facial Plastic Surgery
Chevy Chase, Maryland

ELSEVIER

ELSEVIER
SAUNDERS

3251 Riverport Lane
St. Louis, Missouri 63043

ORTHOGNATHIC SURGERY: PRINCIPLES & PRACTICE 978-1-4557-2698-1
Copyright © 2014 by Jeffrey C. Posnick. Published by Saunders, an imprint of Elsevier Inc.

No part of this publication may be reproduced or transmitted in any form or by any means, electronic or mechanical, including photocopying, recording, or any information storage and retrieval system, without permission in writing from the publisher. Details on how to seek permission, further information about the Publisher's permissions policies and our arrangements with organizations such as the Copyright Clearance Center and the Copyright Licensing Agency, can be found at our website: www.elsevier.com/permissions.

This book and the individual contributions contained in it are protected under copyright by the Publisher (other than as may be noted herein).

Notices

Knowledge and best practice in this field are constantly changing. As new research and experience broaden our understanding, changes in research methods, professional practices, or medical treatment may become necessary.

Practitioners and researchers must always rely on their own experience and knowledge in evaluating and using any information, methods, compounds, or experiments described herein. In using such information or methods they should be mindful of their own safety and the safety of others, including parties for whom they have a professional responsibility.

With respect to any drug or pharmaceutical products identified, readers are advised to check the most current information provided (i) on procedures featured or (ii) by the manufacturer of each product to be administered, to verify the recommended dose or formula, the method and duration of administration, and contraindications. It is the responsibility of practitioners, relying on their own experience and knowledge of their patients, to make diagnoses, to determine dosages and the best treatment for each individual patient, and to take all appropriate safety precautions.

To the fullest extent of the law, neither the Publisher nor the authors, contributors, or editors, assume any liability for any injury and/or damage to persons or property as a matter of products liability, negligence or otherwise, or from any use or operation of any methods, products, instructions, or ideas contained in the material herein.

Library of Congress Cataloging-in-Publication Data

Posnick, Jeffrey C., author.
Orthognathic surgery : principles & practice / Jeffrey C. Posnick.
p. ; cm.
Includes bibliographical references and index.
ISBN 978-1-4557-2698-1 (hbk. : alk. paper)
I. Title.
[DNLM: 1. Orthognathic Surgical Procedures–methods. 2. Malocclusion–surgery. 3. Maxillofacial Abnormalities–surgery. 4. Orthognathic Surgery–methods. WU 600]
RK501
617.6′9–dc23

2013024047

Unless otherwise indicated, medical illustrations throughout the work © by Elsevier Inc.

Vice President and Publisher: Linda Duncan
Executive Content Strategist: Kathy Falk
Senior Content Development Specialist: Brian Loehr
Publishing Services Manager: Julie Eddy
Senior Project Manager: Celeste Clingan
Design Direction: Brian Salisbury

Printed in China
Last digit is the print number: 9 8 7 6 5 4 3 2 1

Working together to grow libraries in developing countries
www.elsevier.com • www.bookaid.org

To my wife, best friend and Soul mate Patti, and our two sons,
Joshua and David who have grown into fine young men.

Jeffrey C. Posnick

Foreword

When I was asked by Jeff Posnick to write a foreword to his new book on orthognathic surgery I said yes, without hesitation. I have known and respected Jeff since he was an enthusiastic, curious, and very bright student at the Harvard School of Dental Medicine, class of 1977. These formative years as a student were instrumental in the subsequent development of his interests and career in craniofacial and orthognathic surgery. Jeff would attend the Children's Hospital Craniofacial Clinic on many a Friday afternoon where he would observe Joseph Murray, John Mulliken, and me interacting with complex craniofacial patients, their families, our students and residents and the rest of our team. From the beginning, I liked Jeff personally, and I developed a relationship with him because he was very attentive and asked many probing questions. He did this not to show off (as Harvard students have been known to do) but to satisfy his inherent curiosity and eagerness to learn. To this day, Jeff will occasionally call to discuss a case and ask for advice, not because he does not have his own ideas or because he does not know what to do, but to check with someone else to see if that person has something to add. This is consistent with his desire to obtain the best information to help each patient and to educate himself. When you give Jeff advice, he always has probing follow-up questions to test your knowledge and recommendations. I have very much enjoyed these interactions over the years, even when our opinions have differed. I also admire Jeff's persistent "sense of wonder" during his long career.*

Never having written a Foreword before, I considered the role of the Foreword and Foreword writer. I was surprised to find that most texts in surgical disciplines have a Preface or an Introduction written by the author, telling how the author became interested in the subject of the book and describing how the book came about. The Preface or Introduction may also contain a summary of the contents of the book. The less common Foreword, on the other hand, is a short introductory statement written by someone other than the author. The writer of a Foreword may be an expert in the field, an author of a similar book and may have a relationship with the author. Presumably, good things will be said about the book, and the author of the Foreword will tell the reader why reading the book is worthwhile. In this respect, the Foreword may be helpful to the publisher for the purpose of marketing.

The more I began thinking about the task at hand, the more onerous it seemed to become. There was no doubt in my mind that this book would be a major contribution to the field, as was Jeff Posnick's 2-volume book: *Craniofacial and Maxillofacial Surgery in Children and Adolescents*, Philadelphia, WB Saunders, 2000. Dr. Paul Tessier wrote in his Foreword to that book: "Thanks go to Dr. Posnick for his overall work and to the publisher for accepting such an abundance of images for printing. As we approach the year 2000 (which has no quantitative reality), this book is already a landmark in craniofacial surgery." M. Michael Cohen Jr. wrote a second foreword calling it a "tour de force" and noting that Jeff wrote 40 of 45 chapters, making it an unusual single-authored book relative to the primary subject. Well, *Orthognathic Surgery: Principles and Practice* is equally a "landmark" and a "tour de force" and there is no use in trying to say something clever about it. Anyone who reads this book will find that it speaks for itself: "*Res ipsi loquitur*."

As with *Craniofacial and Maxillofacial Surgery in Children and Adolescents, Orthognathic Surgery: Principles and Practice* is a single-authored, 2-volume set and therefore has a consistent format, writing style, and "personality" not usually achieved in a multi-authored and edited textbook. This makes it easier and more pleasant to read. The book is divided into seven sections: Basic Principles and Concepts; Planning, Surgical Technique, and Complications; Classic Patterns and Presentations of Dentofacial Deformities; Frequently Seen Malformations with Dentofacial Deformity; Cleft Jaw Deformities; Post-Traumatic Dentofacial Deformities; and Frequent Aesthetic Considerations in the Dentofacial Deformity Patient. Dr. Posnick wrote 39 of the 40 chapters. The first and only invited chapter is the wonderfully informative and entertaining contribution by Jeff's long-term friend and colleague, M. Michael Cohen Jr.: "*The New Perspectives on the Face.*"

Jeff Posnick is meticulous and pays obsessive attention to detail. Therefore, each chapter includes comprehensive background material presented with a scholarly review of the pertinent literature. The relevance of this background to the overall treatment planning, execution, and outcome of orthognathic surgery is revealed and all this is supported by the incredible, well-documented, and beautifully illustrated material from Jeff's personal experience and practice. This presentation allows the reader to benefit from Jeff's thinking and his triumphs, challenges, and difficulties.

It is not the role of the Foreword writer to summarize the book. However, I would like to describe the highlights of just two chapters to support my laudatory comments above. Chapter 2 is an account of the pioneers in orthodontics, oral and maxillofacial surgery, plastic surgery, and

*Mulliken JB: A Sense of Wonder, *Plast Reconstr Surg* 110:1353-1359, 2002.

craniofacial surgery. Jeff Posnick painstakingly chronicles the critical advances in these specialties that brought us to our current state. The chapter reads like an exciting novel. Not only is the history documented in referenced detail, but also anecdotes of personal relationships between these great leaders and personal communications regarding their thinking, ideas, triumphs, and tribulations are described. The chapter ends by thanking the pioneers for their contributions and an appeal to future generations of surgeons to take up the challenge of creating their own innovations.

Chapter 28 on hemifacial microsomia (HFM), a deformity in which I am particularly interested, is another example of the quality of this text. Jeff Posnick and I have some disagreements in this area, particularly regarding the natural progression of the deformity and timing of treatment. We also disagree about the potential benefits of operative correction during growth, i.e., in the mixed dentition stage. Nevertheless, this chapter is one of the most comprehensive treatises on the condition, what is known of the etiopathogenesis and all the significant issues related to the care of patients with this variable, and in my opinion, progressive facial asymmetry that you will find in one location. He has reviewed the pertinent literature, and presented and critically evaluated the available data. By doing this, he implies the importance of understanding the natural history of the deformity and the patterns of growth in the management of these patients. Jeff Posnick is also correctly cautious and skeptical about the use of distraction osteogenesis for early correction. My experience is somewhat different. However, Jeff presents the facts as he sees them, and the conclusions are debatable but fair.

Much has been written on the subject of orthognathic surgery from its history, basic biology and physiology of the operations, descriptions of the techniques, peer-reviewed outcome studies, to review articles and textbooks. The challenge in writing about a common subject is to bring new insights and information to the readers; to say something new or significant and not to simply say what has already been said. Jeff Posnick meets this challenge in *Orthognathic Surgery: Principles and Practice.* It is comprehensive, well referenced, data supported, and scholarly. Also of note is the amazing number of quality color illustrations, a credit to Jeff and to the commitment of the publisher.

I started this project on a beautiful, early summer weekend in Boston, thinking I would skim the chapters quickly for a few hours to get a feel for the book. Not by choice or plan, however, I spent the entire weekend reading the book; I could not put it down. I suspect the readers will have the same experience. This text should be required reading for all surgeons interested in orthognathic surgery.

Leonard B. Kaban, DMD, MD

Walter C. Guralnick Professor and Chairman,
Department of Oral & Maxillofacial Surgery
Massachusetts General Hospital
Harvard School of Dental Medicine
Boston, Massachusetts

Preface

The treatment of dentofacial deformities has come a long way since 1897 when Vilray Blair, with Edward Angle's coaxing, completed bilateral body osteotomies under chloroform anesthesia to setback a prognathic mandible and establish an improved occlusion. The 70-minute operation conducted at the Baptist Hospital in St. Louis, Missouri, also included placement of a custom gutta-percha interocclusal splint and application of intermaxillary fixation.

The field of orthognathic surgery advanced by small increments over the next 6 decades until Hugo Obwegeser executed what has now become the three classic orthognathic procedures: Le Fort I (maxillary) osteotomy with down-fracture and disimpaction; intraoral sagittal split ramus osteotomies of the mandible; and the intraoral oblique osteotomy of the chin. His published results in the 1950s and presentations throughout the 1960s disseminated this early work. The animal model research carried out by William Bell confirmed the safety of these osteotomies and set the stage for refinements in orthognathic procedures by practicing surgeons. During this same timeframe, Hans Luhr boldly challenged standard thinking of osteotomy and fracture healing and stabilization techniques with his concepts of rigid metal plate and screw fixation. Simultaneously, Paul Tessier's imaginative introduction of craniofacial surgery energized thinking concerning the reconstruction of all head and neck conditions.

Today, knowledge of how to safely improve the quality of life for the individual with a dentofacial deformity is extensive. The object is no longer limited to achieving short-term improved occlusion. Currently, the triad of improved quality of life by achieving long-term dental health, enhanced facial aesthetics, and an open airway represent standard thinking. There still remain limitations relating to the uneven geographic distribution of experienced dedicated clinicians and the financial barriers to the correction of dentofacial deformities. However, the value of treatment to improve lives is undisputed.

The last comprehensive textbook on the subject—*Surgical Correction of Dentofacial Deformities* edited by Bell, Proffit, and White (1980)—had a major impact on patient care and remains a landmark in the field. Since then, other published texts have been useful but not comprehensive. After setting the outline for this project, my initial intention was to have experts in the field make contributions. I soon realized this was impractical if a consistent and comprehensive level of cohesive knowledge on the subject was to be compiled in a timely manner. In writing this single-authored text (the exception being a chapter contribution by M. Michael Cohen Jr.), I enlisted the help of clinicians from a spectrum of specialties to read each chapter for accuracy, adequacy of depth, and readability. This included critiques from academicians, clinicians in practice, past surgical fellows, and residents in training. They came from a spectrum of specialties, including oral and maxillofacial surgery, orthodontics, periodontics, prosthedontics, speech pathology, otolaryngology/head and neck surgery, plastic surgery, anesthesiology, medical genetics, sleep medicine, radiology, psychology and psychiatry, and pathology. I am grateful for their suggestions, as each brought a different perspective and individual criticism. By clarifying current knowledge on the subject, I hope this text encourages quality care and further advances in the field.

I would also like to thank my patients who have allowed the use of their case studies as teaching instruments. The presentation of clinical problems and real-life solutions remains an invaluable way to convey this knowledge. Their contributions will no doubt minimize treatment errors and optimize results for future patients.

Jeffrey C. Posnick, DMD, MD

Director, Posnick Center for Facial Plastic Surgery
Chevy Chase, Maryland, USA
JPosnick@DrPosnick.com

Orthognathic Surgery Videos

Go to www.ElsevierOrthognathicSurgery.com to view the videos

INTRA-NASAL PROCEDURES

Video 1: Septoplasty and reduction of the inferior turbinates carried out through Le Fort I downfracture

Video 2: Septoplasty and reduction of the inferior turbinates carried out through Le Fort I downfracture

Video 3: Recontouring of the nasal floor, pyriform rims and anterior nasal spine region carried out during Le Fort I osteotomy

Video 4: Recontouring of the nasal floor, pyriform rims and anterior nasal spine region carried out during Le Fort I osteotomy

AWAKE INTUBATION TECHNIQUES

Video 5: Awake fiberoptic naso-tracheal intubation in patient with TMJ ankylosis

ORTHOGNATHIC PROCEDURES: STEP-BY-STEP APPROACH

Patient 1

Video 6-1: Patient preparation and draping

Video 6-2: Sagittal split ramus osteotomies: incision placement, dissection, and the cortical cuts

Video 6-3: Maxillary Le Fort I osteotomy

Video 6-4: Septoplasty and reduction of the inferior turbinates

Video 6-5: Removal of impacted maxillary wisdom teeth

Video 6-6: Continuing the Le Fort I osteotomy: removal of bony interferences, placement of the intermediate splint, and securing intermaxillary fixation

Video 6-7: Achieving the desired vertical midface dimension

Video 6-8: Placement of titanium plate and screw fixation across Le Fort I osteotomy

Video 6-9: Recontouring of the nasal floor, pyriform rims and anterior nasal spine region

Video 6-10: Release of IMF—confirming the correct occlusion-removal of the intermediate splint-securing of the final splint

Video 6-11: Osseous genioplasty

Video 6-12: Splitting of each sagittal ramus osteotomy of the mandible and removal of impacted mandibular wisdom teeth

Video 6-13: Securing of intermaxillary fixation through the final splint

Video 6-14: Insertion of the transbuccal trocar, seating of the proximal segment, removal of bony interferences, and placement of bicortical screw fixation

Video 6-15: Release of intermaxillary fixation and confirmation of correct occlusion

Video 6-16: Wound closure

Patient 2

Video 7-1: Patient preparation and draping

Video 7-2: Sagittal split ramus osteotomies: Incision placement, dissection, and cortical cuts

Video 7-3: Maxillary Le Fort I osteotomy

Video 7-4: Septoplasty and reduction of the inferior turbinates

Video 7-5: Continuing the Le Fort I osteotomy: removal of bony interferences, placement of the intermediate splint, and securing intermaxillary fixation

Video 7-6: Achieving the desired vertical midface dimension

Video 7-7: Recontouring of the nasal floor, pyriform rims, and anterior nasal spine region

Video 7-8: Placement of titanium plate and screw fixation across the Le Fort I osteotomy

Video 7-9: Release of IMF-confirming the correct occlusion-removal of the intermediate splint-securing of the final splint

Video 7-10: Osseous genioplasty

Video 7-11: Splitting of each sagittal ramus osteotomy of the mandible

Video 7-12: Securing of intermaxillary fixation through the final splint

Video 7-13: Insertion of the transbuccal trocar, seating of the proximal segment, removal of bony interferences, and placement of bicortical screw fixation

Video 7-14: Release of intermaxillary fixation and confirmation of the correct occlusion

Video 7-15: Wound closure

Video 7-16: Anterior neck rejuvenation

GRAFT HARVESTING

Video 8: Harvesting cancellous bone through the anterior iliac crest

Video 9: Harvesting a corticocancellous block graft from the anterior iliac crest

Video 10: Harvesting a rib cartilage graft

OSSEOUS GENIOPLASTY

Video 11: Osseous genioplasty with horizontal advancement

Video 12: Osseous genioplasty with horizontal advancement

SELECTED PROCEDURES

Video 13: Anterior neck rejuvenation: cervical flap elevation, fat removal, vertical platysma muscle plication

Video 14: Cleft orthognathic surgery for the BCLP deformity

Video 15: Monobloc and facial bipartition osteotomies for the reconstruction of craniosynostosis syndromes

1 The New Perspectives on the Face+

M. MICHAEL COHEN JR.

- What Constitutes a Face?
- Facial Perspectives and Stages in the Life Cycle
- Evolutionary Considerations
- Craniofacial Growth and Development
- Dysmorphic Faces
- The Outer Limits in Facial Surgery
- Making Blind People See Again
- Prosopagnosia
- The New Psychiatric Genetics
- Artistic Perspective

• **Figure 1-1** Two fish faces. *Copyright © Shutterstock.com.*

What Constitutes a Face?

Structural Definitions

There are several ways to view faces. Figure 1-1 shows two fish.[14] If two eyes and a mouth define a face, then fish have faces. They may have one or two nostrils, which are not connected to the mouth. However, water entering the nostrils does bathe the olfactory mucosa. Fish lack an ear canal, but the inner ear is present. Weber's bones connect the swim bladder to the inner ear, and sound is transmitted.[10]

Another definition requires evolutionary transformation of the skull, in which a face is recognizable in mammals, but not in fish, amphibians, or reptiles (Fig. 1-2). The jaw is suspended from the braincase in reptiles. Mammals, however, have three ear ossicles, a secondary palate separating the airway passage from the mouth, and vertical positioning of the dentary.[35]

Behavioral Definitions

All animals communicate with each other in various ways by tactile, chemical, visual, and acoustical signaling. Insects have heads, but do they have faces? Paper wasps *(Polistes dominulus)* signal their status to each other by the number of black splotches on their yellow faces (Fig. 1-3).[41]

Some define faces by the presence and use of facial muscles, which do not exist in fish, amphibians, reptiles, or birds. In contrast, mammals can suckle and later chew, supported by a muscular tongue and movable lips and cheeks (Fig. 1-4). Often, the muscles in the ears can change positions to aid in hearing; a movable nose for smelling and touching, and facial hair, the *vibrissae,* are associated with musculature and serve as tactile organs.[10]

For some, a behavioral definition must include facial expressions, which are found in various primates (Fig. 1-5), including humans (Fig. 1-6).[10]

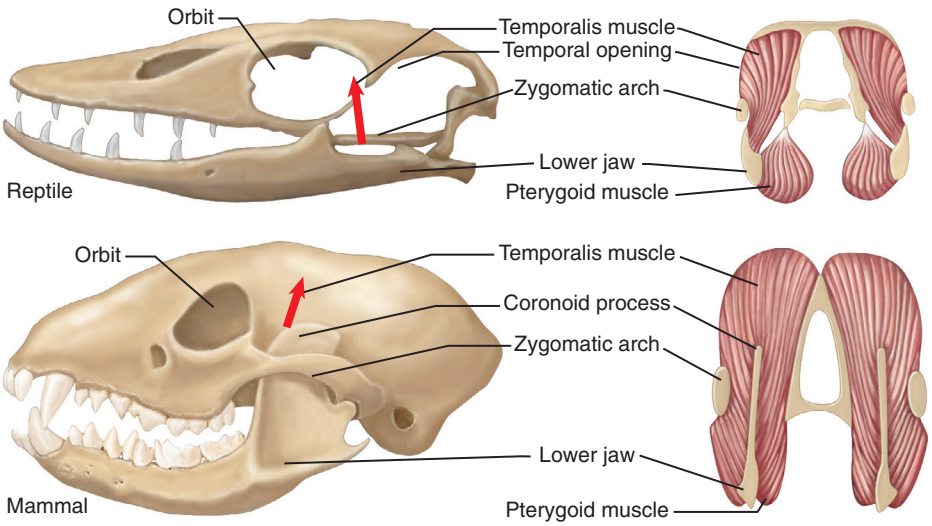
Symbolic Definitions

The term *symbolism* has been defined in many different ways. Here, I define *symbolic transformation* as the ambiguous representation of two things by association or resemblance. Two examples are shown in Figures 1-7 and 1-8.[10]

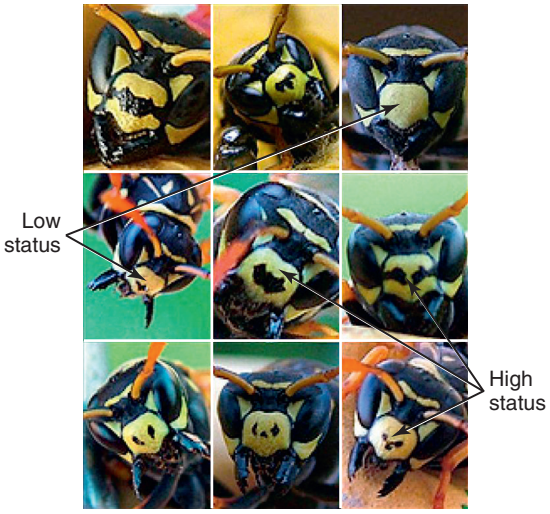
Facial Perspectives and Stages in the Life Cycle

Box 1-1 summarizes all possible perspectives from which the face can be described; Box 1-2 lists all the stages in the

+This chapter is dedicated to my Fellow Angela M. Castro, MD.



• **Figure 1-2** Transformation of a mammalian-like reptile to a mammalian skull. Lateral and cross-sectional views of the skulls. *Based on Radinsky, 1987.*

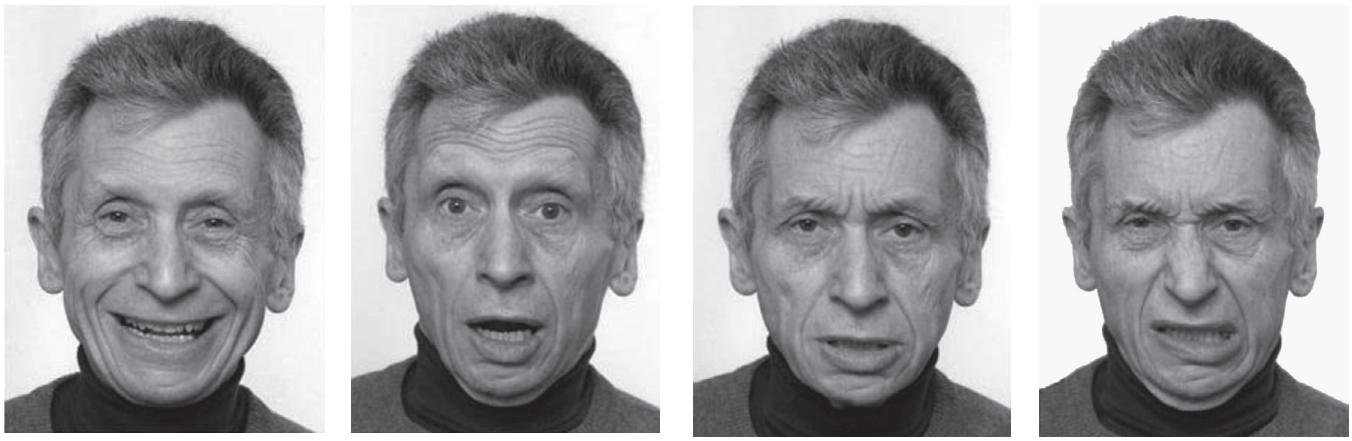


Paper wasps *(Polistes dominulus)*

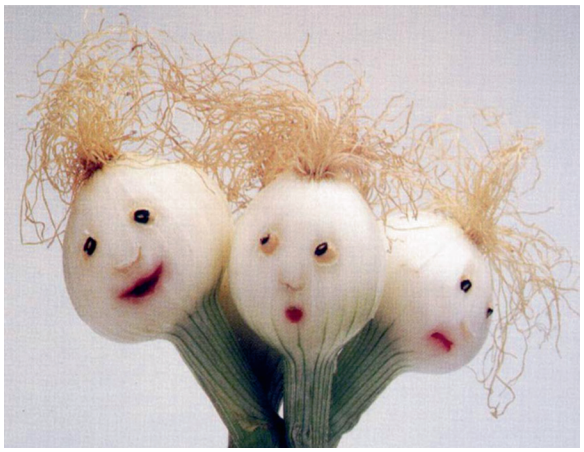
• **Figure 1-3** Wasp faces signal their status to each other by the number of black splotches on their yellow faces. The more blotches, the higher the status. *Copyright 2004 Elizabeth Tibbetts. Modified by M. Michael Cohen Jr.*

• **Figure 1-4** Mammals can suckle and later chew, supported by a muscular tongue and movable lips and cheeks.

• **Figure 1-5** Rhesus monkey facial expressions. **From left to right,** a bared-teeth display, a scream, an open-mouth threat, and a relaxed, open-mouth face (play face). *From Parr and Heintz, 2009.*



• **Figure 1-6** Human facial expressions. **A,** Happiness. **B,** Surprise. **C,** Anger. **D,** Disgust. *Based on Cohen, 2006.*



• **Figure 1-7** Gossiping scallions. *From Elffers, 1997.*

• **Figure 1-8** *All Is Vanity*. The mirror is also a skull. House of Art, New York.

• BOX 1-1

Perspectives on the Face

Evolutionary
Developmental
Anatomic
Genetic
Surgical
Psychological
Sociocultural
Artistic

• BOX 1-2

The Face in the Life Cycle

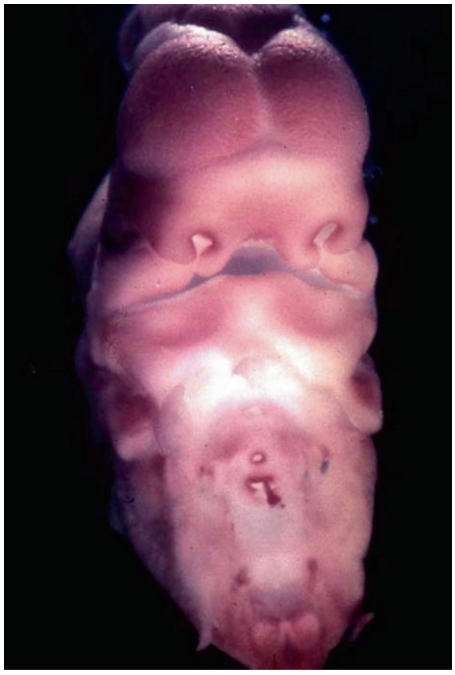
Intrauterine development
Birth
Infancy
Childhood
Adolescence
Adulthood
Old age
Death

life cycle; and Box 1-3 lists the origin of some craniofacial components. Figure 1-9 shows embryonic facial development at approximately 42 to 44 days.[25] Figure 1-10 shows skull molding in a newborn. Figure 1-11 shows the face of a small child, and Figure 1-12 shows a painting, *The Stages of Human Life*, by Hans Baldung Grien. In this allegory, a young woman, an old woman, and a dead woman are linked by their hands and arms.[4] The dead woman holds an hourglass timer, indicating that life is over.

• BOX 1-3

Origins of Some Craniofacial Components

Neural Crest Origin	Mesodermal Origin
Facial connective tissue	Facial muscles
Facial bones	Endothelial lining of blood vessels
Nasal capsule	Chondrocranium
Frontal bone	Parietal bones
Metopic suture	Coronal suture
Sagittal suture	Lambdoid suture
Temporal squama	Temporal bones (except squama)
	Occipital bone (somites plus squama)



• **Figure 1-9** Embryo of about 42 to 44 days. The developing face has a frontal area with bulging cerebral hemispheres; a nasodorsal center with nasoseptal and nasozygomatic portions; nasal pits delineated by nasal ridges with premaxillary, medionasal, and latero-nasal portions; maxillary primordia; interpremaxillary depression; a premaxillary–maxillary junction; and a lower jaw. *Courtesy of Jan E. Jirásek, Prague.*

• **Figure 1-10** Molding of the head caused by compression during passage through the birth canal. *From Cohen, 2006.*

Evolutionary Considerations

Evolution of the Mammalian Brain

The evolution of the mammalian brain is based on the development of the neocortex (Fig. 1-13), which also resulted in a cranial shape that was different than that of the dinosaurs. The coexistence of dinosaurs and mammals and their competition during the Mesozoic era made mammalian neocortical development possible.[5]

Dinosaurs were large and had long life spans of about 100 years. In contrast, mammals were very small and had short life spans, which allowed them to evolve into many different species rapidly. Possibly, there were 50 generations of mammals during one dinosaur generation.

Mammals were nocturnal and hunted at night. With the development of the neocortex, mammals had an active lifestyle as well as acute hearing and smelling, which made insect prey easier to come by and made escape from predators easier as well. The end of the dinosaur era allowed mammals to evolve larger body forms.

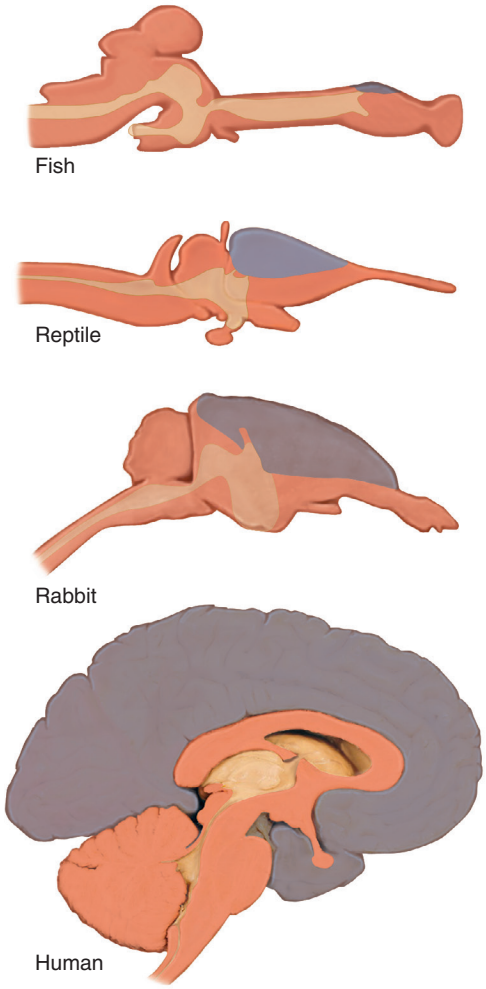
Molecular Components in Primate Brain and Craniofacial Evolution

The gene *Microcephalin (MCPH1)* was first identified in its mutant form in which it causes primary microcephaly, but the normal gene was then adaptively found to be important in regulating brain size, and it continues to evolve in humans.[18]

Homozygous *ASPM* mutations also cause microcephaly. The normal gene may regulate neural stem cell proliferation

• **Figure 1-11** Baby's face with relatively large head, prominent eyes, and a diminutive face. *From Cohen, 2006.*

• **Figure 1-12** *The Stages of Human Life* (1530-1545), by Hans Baldung Grien, oil on wood, 61 × 151 cm, Prado Museum, Madrid. *From Buendia, 1989. See text for details. Musceo del Prado, Madrid, Spain. Photo credit: Bridgeman-Giraudon/Art Resource, NY.*



• **Figure 1-13** Stages in the evolution of the brain. A comparison of the brain size of the fish, reptile, rabbit, and human. As the cerebral cortex increases in size, the cranium becomes larger and more rounded. *Based on Campbell, 1970.*

and/or differentiation, possibly by mediating spindle-cell assembly during cell division.[31]

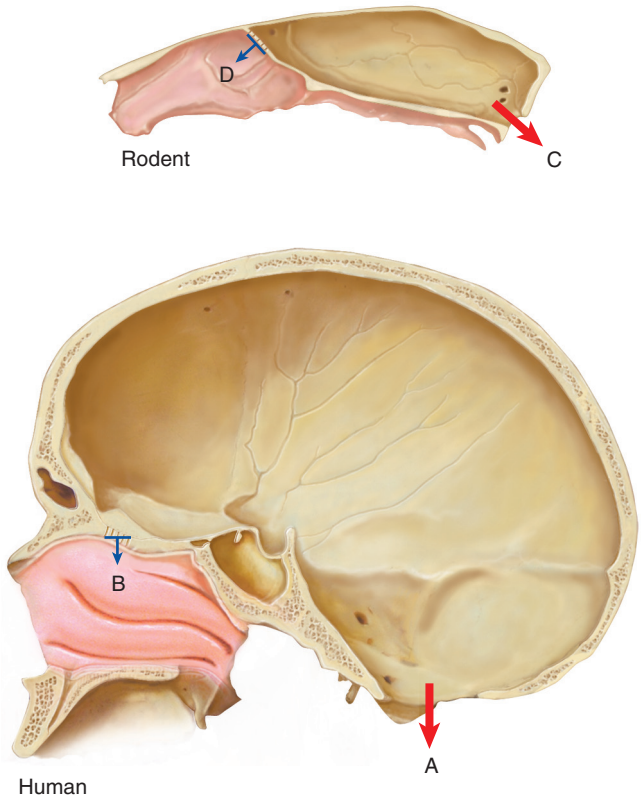
SIGLEC11, a gene involved in sialic acid biology, is expressed in high concentration in microglial cells in the human brain, but only occasionally in the cells of chimpanzees.[24]

The *Ret finger protein-like 1,2,3* (*RFPL 1,2,3*) genes on chromosome 22 are evolutionary forces that play a role in neocortical development.[3]

GTF2IRD1—a gene that, when mutated, causes craniofacial anomalies—has been shown in its normal form to be a regulatory determinant of craniofacial development.[39]

Comparison of Different Skulls

Figure 1-14 shows the orientation of the foramen magnum and the anterior cranial base in a rodent and a human. The foramen magnum is posteriorly placed in the rodent and vertically placed in the human. The anterior cranial floor and the cribriform plate face forward in the rodent and are vertically placed in the human.[16] Figure 1-15 shows a dog's



• **Figure 1-14** Orientation of the foramen magnum and the anterior cranial base in a human and a rodent. The foramen magnum is vertically placed in the human *(A)* and posteriorly placed in the rodent *(C)*. The anterior cranial floor and the cribriform plate face downward in the human *(B)* and forward in the rodent *(D)*. *Based on Enlow, 1968.*

skull compared to a Chevrolet Corvette in contrast to a human skull compared to a camper, indicating an expanded, upright forehead above the face.[17]

In Figure 1-16, expansion of the brain and a reduction in human facial prominence results in a flat facial profile compared with a smaller brain and a more protruding gorilla face, resulting in a sloping facial angle.[10] Figure 1-17 compares the growth pattern of a fetal chimp with that of a human fetus. Both fetuses look similar. However, compared with the adult chimp, the adult human more closely resembles its own fetal pattern. Neoteny—a slowdown in the growth rate with a delay in maturation—has occurred in human evolution.[23]

Comparison of *Homo sapiens, Homo neanderthalensis,* and *Homo floresiensis*

Evidence now shows that there are three separate species of hominins. Figure 1-18 compares the skulls of *Homo neanderthalensis* with *Homo sapiens*. The braincase of modern humans is relatively shorter, and the forehead is rounder, higher, and has a nearly vertical slope. Neanderthals have large brow ridges, projecting midface, elongated skull, occipital protuberance, a skull capacity 10% greater than that of modern humans, and a distinctive bony labyrinth not found in humans.[27] DNA sequence comparisons show that Neanderthals fall outside the variation of modern humans. Molecular divergence provides a date of over 500,000 years.[28]

Figure 1-19 contrasts the phenotypes of the different species of hominins. *Homo neanderthalensis* has an elongated skull with a larger endocranial capacity than in humans; a distinctive bony labyrinth not found in humans (not shown); a robust skeleton with a barrel-shaped ribcage; a long superior pubic ramus; long clavicles; thick, bowed femoral shafts; large patellas; and large, round, terminal phalanges (fingers). *Homo floresiensis* has a tiny skull with a small endocranial capacity (380 cm^3 to 430 cm^3), a chinless mandible, a diminutive body (~1 m in height), long arms in relation to the legs (arms hang almost to the knees), unusual shoulders, a wider pelvis than in humans, and large feet (more than 7½ inches long and out of proportion with the short lower limbs, flat feet, and a stubby great toe).[10]

The time spans of the three different species of hominins are (1) *Homo sapiens* from 500,000 years ago to the present time; (2) *Homo neanderthalensis* from 500,000 years ago until 30,000 years ago; and (3) *Homo floresiensis* from 95,000 years ago until 12,000 years ago. Thus, from 95,000 to 30,000 years ago, three different species of hominins occupied the earth at the same time.

Craniofacial Growth and Development

Comparative Skull Size with Age

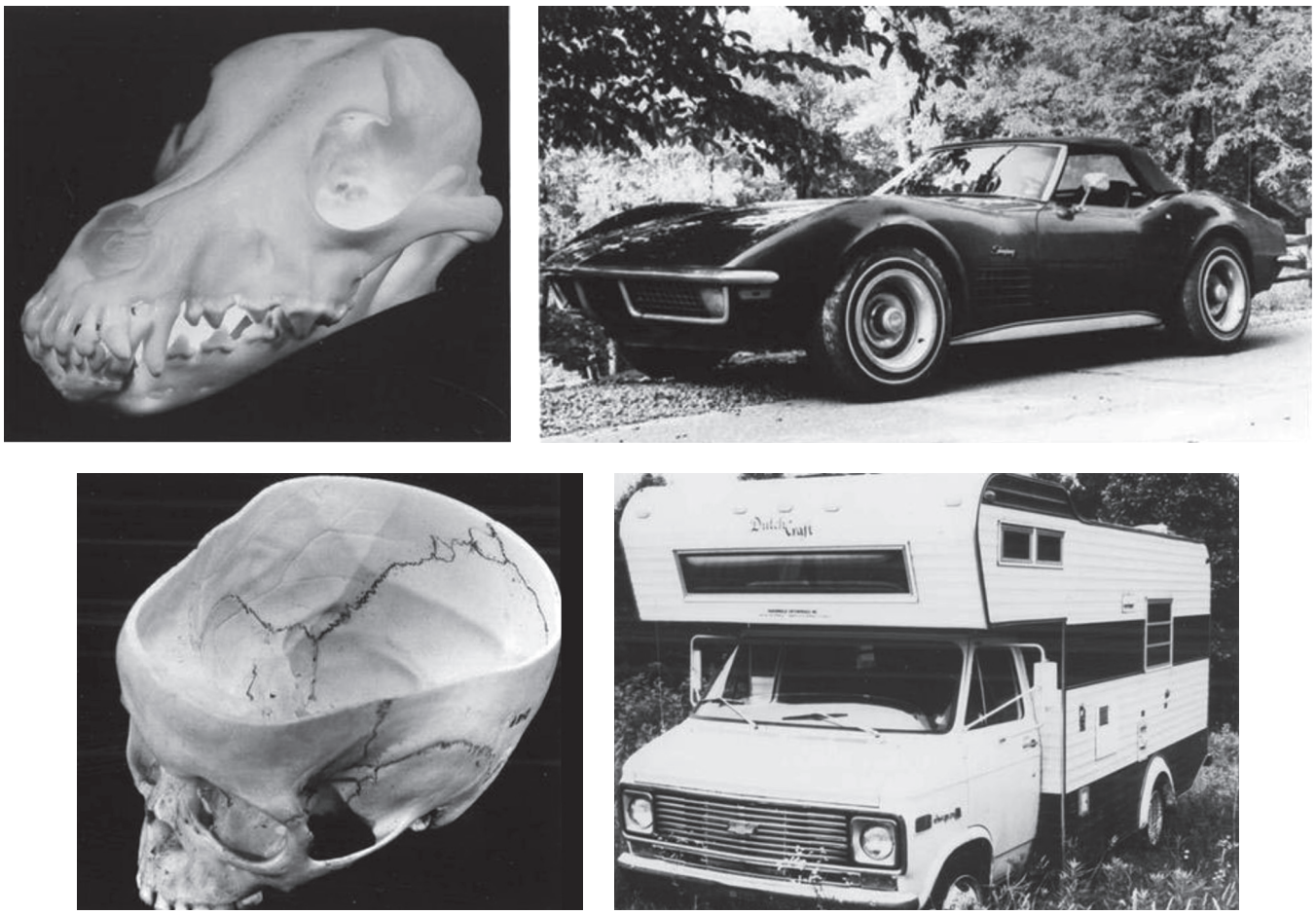
In the newborn skull (Fig. 1-20), the cranium and the orbits are relatively large, and the face is diminutive. In early childhood, the cranium and the orbits remain relatively large, but the eruption of the primary dentition enlarges the facial skeleton. In the adult skull, the facial skeleton is well-developed with relatively less prominence of the orbits and cranium. The ratio of the cranial volume to the facial volume changes during growth, and these ratios appear in Figure 1-20 under the skulls.[10]

Bone Modification with Age

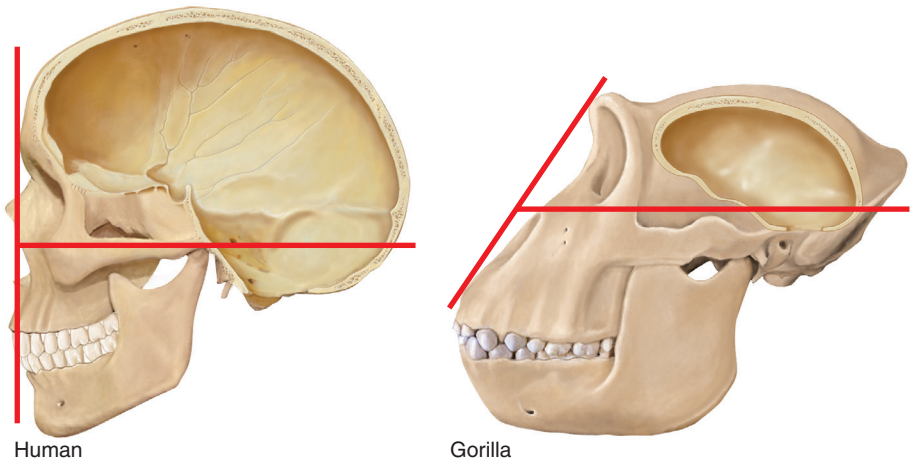
Bone modification of the craniofacial skeleton occurs normally with age (summarized in Table 1-1). Table 1-2 lists some examples of abnormal alterations in the craniofacial skeleton with age.[10]

Alterations in the Face with Age

The changing proportions of head size to body size are illustrated in Figure 1-21. From the 2-month-old embryo to the 22-year-old adult, the relative head size decreases significantly, but it is greatest in the fetus and the infant. Figure 1-22 illustrates line drawings of the facial profile showing alterations from 5 months of age to adulthood, with striking changes in the nose and chin with age.[10] The



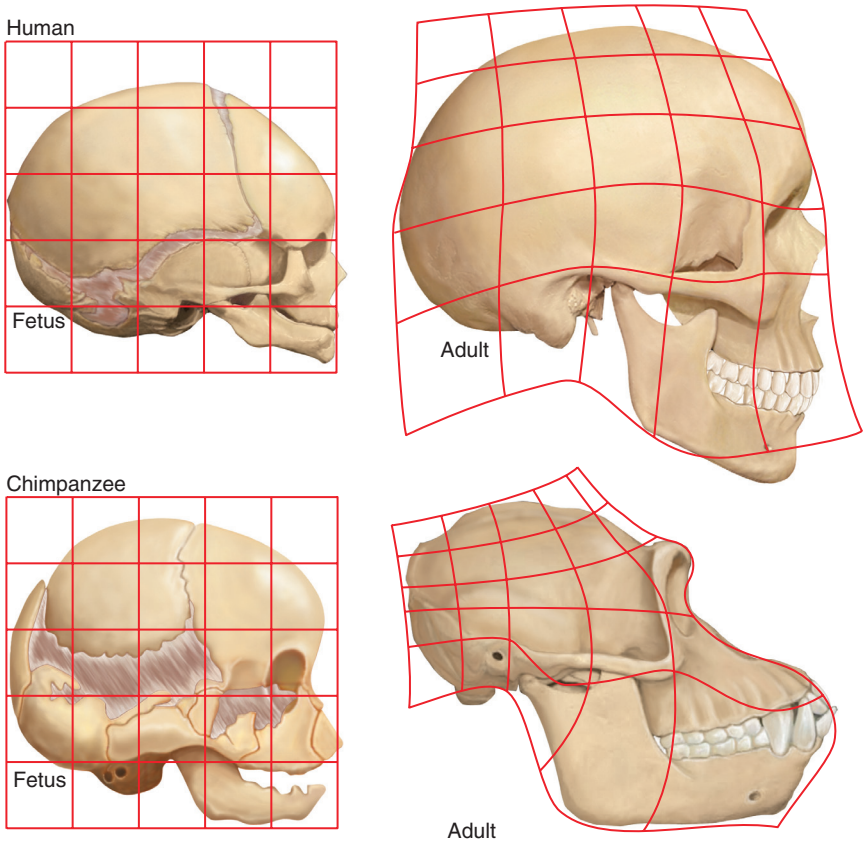
• **Figure 1-15** **Top,** Lateral view of a dog skull compared to a Chevrolet Corvette; the face is anterior to the cranium. **Bottom,** Lateral view of a human skull compared to a camper; the expanded forebrain results in an upright forehead above the face. *From Enlow, 1990.*



• **Figure 1-16** Expansion of the brain and a reduction in the facial prominence of humans result in a flat facial angle compared with the smaller brain and more protrusive face of the gorilla, resulting in a sloping facial angle.

TABLE 1-1 Normal Growth and Modification of the Craniofacial Skeleton with Age

	Cartilaginous Growth	Sutural Growth	Skeletal Mass	Bone Remodeling
Infancy and Childhood	Active	Passive	Increases	Deposition greater than resorption
Adulthood	Absent	Absent	Remains the same	Deposition equals resorption
Old Age	Absent	Absent	Decreases	Resorption greater than deposition



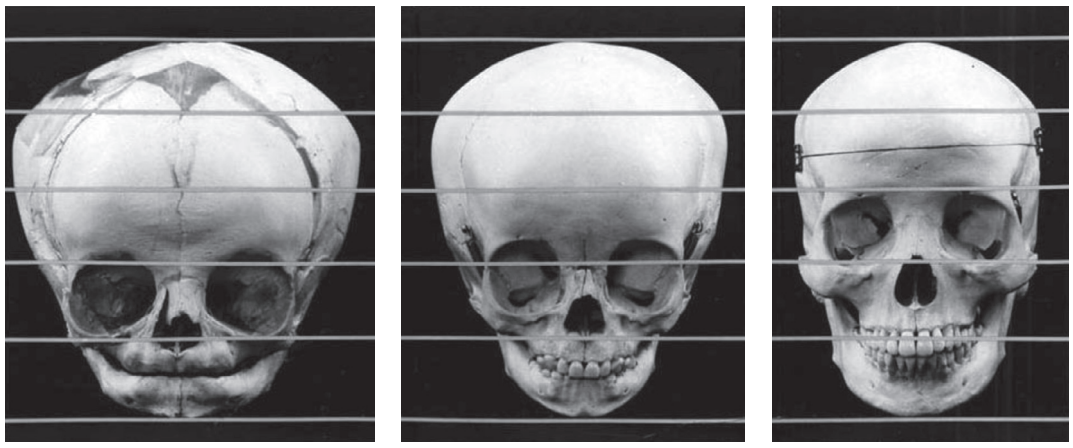
• **Figure 1-17** Morphology of the chimpanzee and human skulls. Compared with the chimp, the adult human skull resembles its fetal counterpart. Neoteny—a slowdown in the growth rate with a delay in maturation—has occurred in human evolution. *Based on Gould, 1977.*

Homo sapiens *Homo neanderthalensis*

• **Figure 1-18** Lateral view of the skulls of *Homo sapiens* and *Homo neanderthalensis*. See text for description.

A *Homo sapiens* B *Homo neanderthalensis* C *Homo floresiensis*

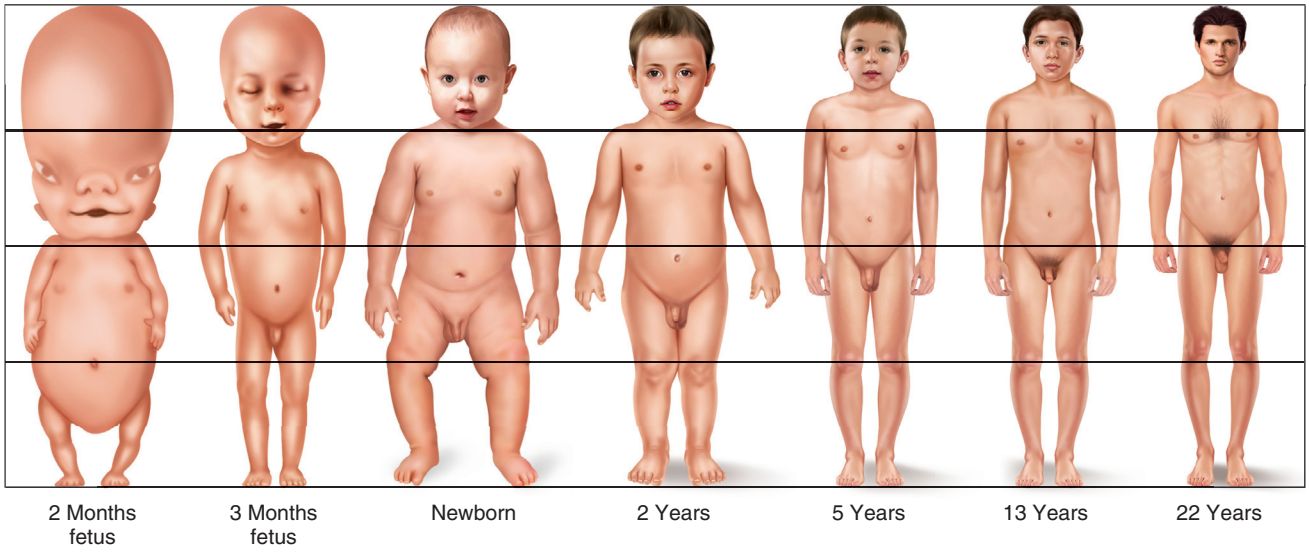
• **Figure 1-19** Comparison of **A,** *Homo sapiens,* **B,** *Homo neanderthalensis*, and **C,** *Homo floresiensis*. See text for description. *Based on the work of M. Michael Cohen Jr.*



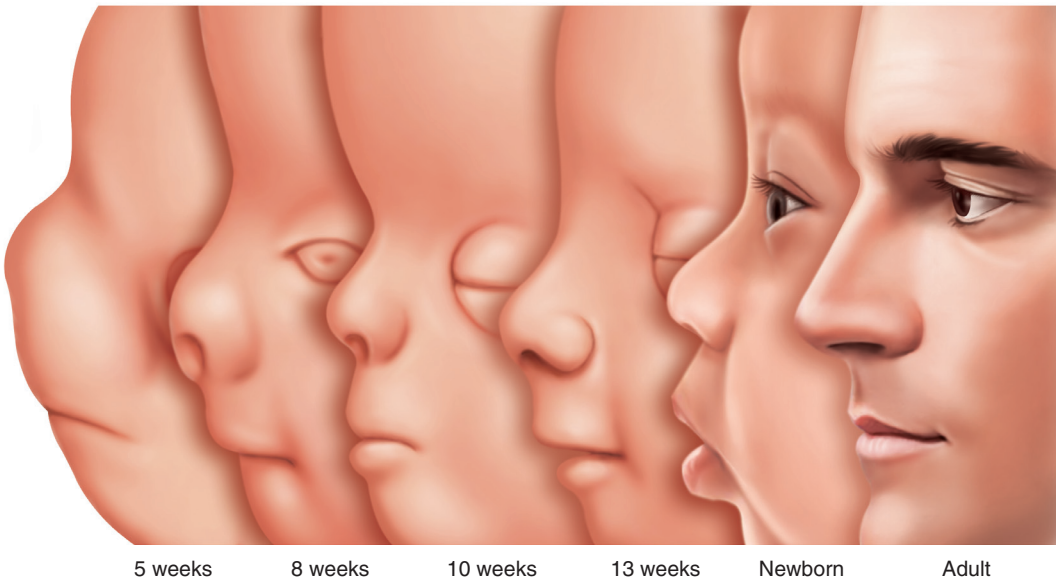
A B C

Ratio of cranial volume to facial volume
- Birth (8:1)
- 2 years (5:1)
- 6 years (3:1)
- 12 years (2.5:1)
- 18 years (2:1)

• **Figure 1-20** Alterations of the skull in the newborn, in childhood, and in adulthood. The ratios of the cranial volume to the facial volume change during growth, and these ratios appear under the figure. For a description of skull alterations with age, see text. *From Cohen, 2006.*



• **Figure 1-21** Diagram illustrating the changing proportions of head size and body size. From the 2-month-old embryo to the 22-year-old adult, the relative head size decreases significantly, but it is greatest in the fetus and the infant. *Based on Scammon, 1953.*



• **Figure 1-22** Line drawing of the human profile illustrating alterations in form and proportion from 5 weeks prenatally through the newborn period to the adult. *Based on Scammon, 1953.*

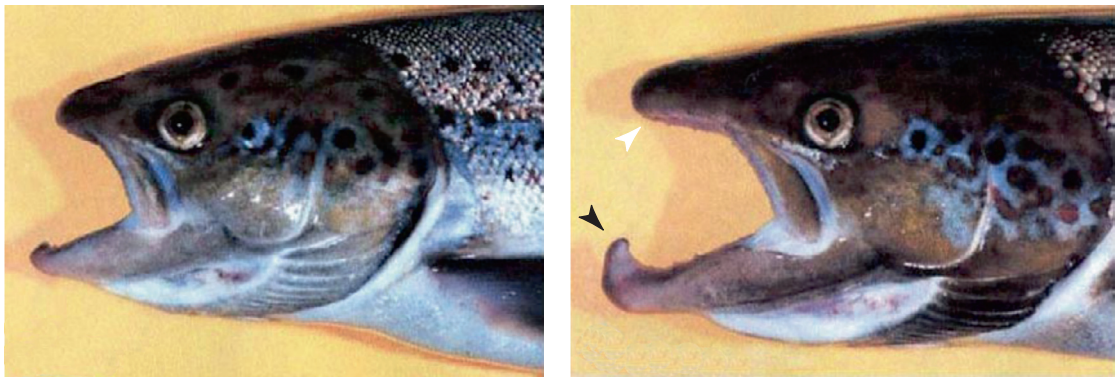
lower jaw in the male Atlantic salmon is extraordinary. There is rapid and pronounced growth *during adult life* (Fig. 1-23), when the *starving* salmon migrate upriver for spawning.[42]

Facial Asymmetry

Those psychologists who study faces and state that "beautiful faces are symmetric" don't know what they're talking about, because all normal faces are asymmetric. With respect to the normal face, subtle degrees of asymmetry become particularly evident when properly oriented frontal photographs are divided along the median plane and reprocessed, each side being paired with its mirror image (Fig. 1-24).[10]

The Skull as a Community of Bones versus the Skull as a Community of Joints

According to Pruzansky,[34] the skull is a community of bones separated by joints, but according to Moffett[32] the skull is a community of joints separated by bones. Which view is correct? They are simply different contexts in which to view the development of the skull.[10]



A B

• **Figure 1-23** Rapid and pronounced growth of the lower jaw in a starving adult male salmon migrating upriver for spawning. **A,** Normal adult male jaw. **B,** Extraordinary upriver growth of the lower jaw. *From Witten et al, 2003.*

Normal face (All normal faces are asymmetric)

Frontal photographs are reprocessed, each side being paired with its mirror image

Left side reprocessed with its mirror image

Right side reprocessed with its mirror image

• **Figure 1-24** Demonstration of the facial asymmetry that is so typical of everyone. *From Cohen, 2006.*

Pruzansky[34] stated the following:

"The skull is a community of bones and organ systems of diverse phylogenetic origin and variable patterns of development, altogether relating to several functions vital to the life and well-being of the organism. If in the course of development one member of this community is affected adversely, inevitably other parts will suffer."

• **Figure 1-25** Midface deficiency secondary to hypoplasia of the nasal capsule in achondroplasia. *From Cohen, 2000.*

Achondroplasia, caused by several *FGFR3* gain-of-function mutations (particularly Gly380 Arg[9]) illustrates what Pruzansky[34] meant (Fig. 1-25): if one member of the community is affected, inevitably other parts will suffer. In this case, the nasal capsule and cranial base, both cartilaginous in origin, have a secondary effect on membrane bones, because all craniofacial bones articulate with one another. Thus, in achondroplasia, the hypoplastic nasal capsule and short anterior cranial fossa result in midface deficiency.[7,9,10]

The skull may also be considered a community of joints separated by bones. Types of joints, known as *craniofacial articulations,* include synovial, cartilaginous, fibrous, and dental.[32] Table 1-3 summarizes craniofacial articulations together with their physiologic and mechanical functions and their remodeling responses.[10]

TABLE 1-2 Some Examples of Abnormal Alterations in the Craniofacial Skeleton with Age

	Abnormal Skeletal Condition	Pathologic Process
Infancy and Childhood	Achondroplasia	Bony midface deficiency secondary to hypoplasia of cartilaginous nasal capsule and cranial base
	Craniosynostosis	Premature sutural fusion by bone deposition
Adulthood	Acromegaly	Bone deposition dramatically exceeds bone resorption
	Hemifacial atrophy	Localized bone resorption exceeds bone deposition
Old Age	Paget disease of bone	Bone deposition exceeds bone resorption
	Senile osteoporosis	Bone resorption exceeds bone deposition

Dysmorphic Faces

Four different types of faces are contrasted in Figure 1-26: (1) the normal face; (2) Down syndrome with minor anomalies; (3) Crouzon syndrome; and (4) a bizarre prenatally determined face. Three views of Williams syndrome are illustrated in Figure 1-27. The combination of minor facial anomalies—strabismus, anteverted nares, and thick lips—are evident in all three patients. In contrast, the severe facial anomalies associated with holoprosencephaly—cyclopia, ethmocephaly, cebocephaly, and premaxillary agenesis—are shown in Figure 1-28.[6,8,10-12] A newborn with diprosopus—a double face with two nostrils, four eyes, two noses, and two mouths—is illustrated in Figure 1-29. The most bizarre case of all is shown in Figure 1-30. This patient is missing his lateral orbital walls, and he is able to pull his eyes apart laterally without ripping his optic heads from the back of his eyeballs. How he does this is mysterious, but I suspect he started doing this very early in childhood and gradually increased the distance over time.

The Outer Limits in Facial Surgery

The problems surrounding complete facial transplantation are technical, psychological, and ethical. Earlier, facial transplantation was rejected by many institutions, with the exception of the Cleveland Clinic's Institutional Review Board, and I have discussed the concept of facial transplantation and its associated problems elsewhere.[10] Although it was rejected earlier by the French National Ethics Advisory Committee, the door was left open for a severely disfigured woman bitten by a dog for which she received a lower facial transplant. Since then, at least seven facial transplants have been carried out. The result of a facial transplant after a gunshot wound is shown in Figure 1-31.

Making Blind People See Again

The cornea covers the eye globe and a narrow zone between the cornea and the conjunctiva known as the *limbus* (Fig. 1-32), which is a source of stem cells for corneal epithelium. In recent years, new techniques that can restore vision in certain types of blindness have come to the fore. In a surgical procedure called *limbal cell transplantation,* extraction of stem cells from the healthy contralateral eye of a patient or from a relative in the family is transplanted into a patient's eye with corneal degeneration, blindness, or some other ocular disease. The stem cells then differentiate into corneal epithelial cells and improve vision.[19] The limbus itself can be destroyed by chemical burns, by thermal burns, or by infection, which results in corneal stem-cell deficiency. However, it has been recently reported that a viable alternative source of cells for transplantation consists of limbal cells maintained in culture.[36] A biosynthetic cornea created from human collagen has also been developed. It mimics the protein's scaffolding and can be used to trigger regeneration of a patient's own corneal cells.[20]

Age-related macular degeneration is a major cause of blindness. Although there is no treatment for the avascular type, the neovascular type results from an imbalance in antiangiogenic and proangiogenic factors and can be treated. Intravitreal injection of VEGF inhibitors results in significant recovery of vision in 30% to 40% of patients.[40]

Leber congenital amaurosis is an inherited group of rod–cone dystrophies resulting in congenital blindness. One form caused by the *RPE65* gene accounts for 16% of the cases. Using rAAV as a vector for retinal gene therapy, a cannula is passed through the front of the eye and across the vitreous gel. Working copies of the gene are injected into the back of the eye. Maguire and colleagues[30] reported improvement in visual acuity for all patients.

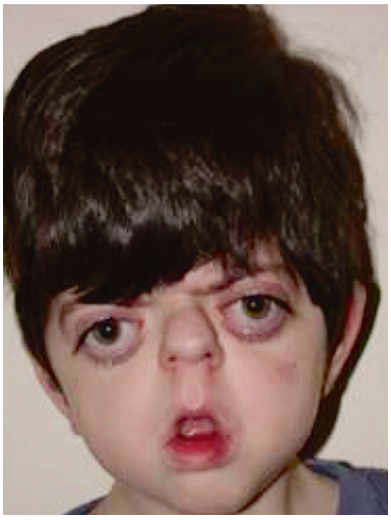
Several animal studies show great promise for future treatment. The development of retinal cell transplants has been studied in rabbits,[38] and retinal repair by transplantation of photoreceptor precursors has been studied in mice.[29]

Prosopagnosia

The ability to identify human faces is remarkable. Although the face ages with time, it is possible to instantly identify a face of someone who has not been seen for 25 or 30 years. An area in the brain that governs facial recognition is found

Normal face

Down syndrome



Crouzon syndrome

Severe prenatal anomaly

• **Figure 1-26** Four kinds of faces with different implications. A normal child's face is shown. Down syndrome involves many minor facial anomalies that are diagnostic. Crouzon syndrome is severe but can be treated successfully with surgery. There is no effective treatment for the infant with severe facial anomalies and hydrocephalus. *Based on the work of M. Michael Cohen Jr. Crouzon syndrome is by courtesy of Bonnie Padwa, Boston.*

• **Figure 1-27** Three patients with Williams syndrome of different ages. All have strabismus, anteverted nares, and thick lips. *From Cohen, 1997, 2002, 2006, and 2007, a and b.*

Cyclopia | Ethmocephaly | Cebocephaly | Premaxillary agenesis

• **Figure 1-28** Faces associated with holoprosencephaly. *From Cohen, 1997, 2002, 2006, and 2007, a and b.*

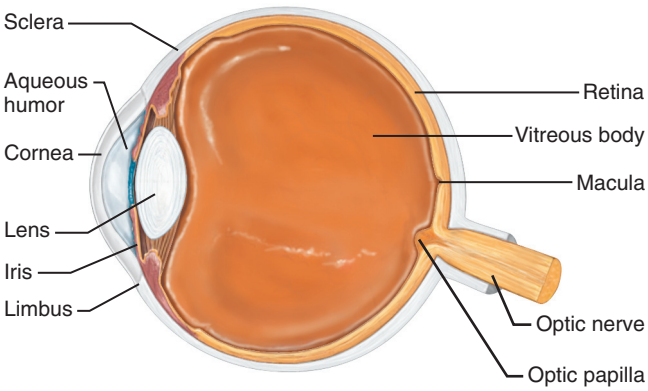
• **Figure 1-29** Diprosopus. *Courtesy of Gosla Reddy.*

• **Figure 1-30** Hypertelorism with absent lateral orbital walls. This man is able to manipulate his eyes without ripping his optic heads from the back of his eyeballs. I suspect that he learned to do this very gradually. *Courtesy of M-Reza Farahvash.*

12/3/08 | 5/4/09 | 8/23/10

• **Figure 1-31 A,** Three frontal views by dates. 12/3/08: A 45-year-old woman with a shotgun blast to the face. She underwent a nearly total facial transplantation, including a composite LeFort III midfacial skeleton, overlying skin, soft tissue, nose, lower eyelids, upper lip, total infraorbital floor, both zygomas, both parotid glands, the anterior maxilla with central maxillary incisors, the whole alveolus, the anterior hard palate, and intraoral mucosa. Note tracheostomy. 5/4/09: Second facial transplantation with construction of the nose and lips. 8/23/10: Reduction of facial fat and better facial appearance. **B,** Three profile views with the same dates as in **A**. 5/4/09: Note better projection of the nose. 8/23/10: Mandibular advancement with normal lower facial appearance. Both the upper and lower portions of the face are balanced. *Courtesy of the Cleveland Clinic.*

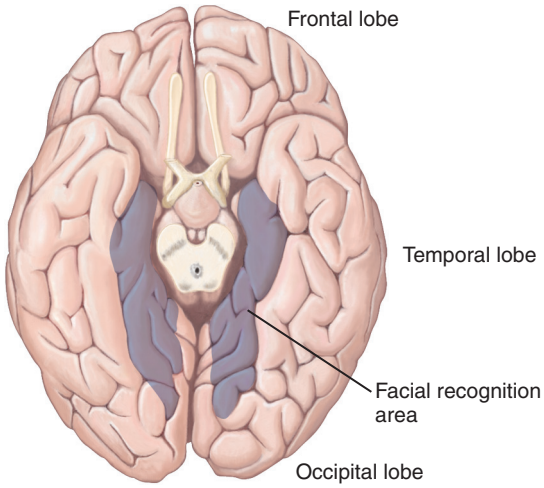
at the base of the cerebral cortex (Fig. 1-33). Any lesion that destroys this area impairs the ability to identify faces but has almost no other effects, although the dimming of vision may occur in some affected individuals. The patient can read, name objects, and match a full-face picture with a profile picture of the same person. Only the ability to recognize specific faces is lost; this is a neurological condition known as *prosopagnosia.* An affected man who cannot recognize his own wife can see her, but he can only recognize who she is when he hears her speak.[21] An epidemiologic study has found a prevalence rate of prosopagnosia of 2.47%; of 17 cases, 14 subjects had at least one first-degree relative with prosopagnosia.[26]



• **Figure 1-32** Eye globe, horizontal plane. Note the limbus. *Based on Cohen, 2006.*

The New Psychiatric Genetics

The brain, as part of the craniofacial complex, is associated with a number of psychiatric and neurodegenerative disorders. In medical genetics, we used to delineate newly recognized syndromes by describing them as single entities and then finding a Mendelian pattern, a chromosomal anomaly, or a teratogenic cause. After molecular genetics became established, it became clear that the same disorder might



• **Figure 1-33** Prosopagnosia. Note the area of the brain that governs facial recognition found at the base of the temporal and occipital lobes on both sides of the cerebral cortex. *Based on Geschwind, 1979.*

TABLE 1-3 Craniofacial Articulations

Type of Articulation	Example	Physiological Function	Mechanical Function	Remodeling Response
Synovial	Temporomandibular joint	Jaw movement	Resists compression and shear to some extent	Limited, avascular
Cartilaginous	Cranial base synchondroses	Active growth	Resists compression	Limited, avascular
Fibrous	Cranial sutures	Allows passage through narrow birth canal; growth secondary to brain enlargement	Respond to tension	Great, vascularized
	Facial sutures	Mastication	Sutures remain patent until old age; shock absorbers for forces of mastication	Great, vascularized
	Periodontal fibers	Eruption of teeth; anchoring support of teeth	Responds to tension, compression, and shear	Great, vascularized
Dental	Occlusal and interproximal articulations	Mastication and speech	Subject to compression and shear	None, acellular

have causative mutations in several different genes on different chromosomes. For example, Biedl-Bardet syndrome can be caused by mutations in 14 different genes, all on different chromosomes. Thus, we learned that what appeared to be a single disorder was etiologically heterogeneous.

Because psychiatric thinking began by attributing behavioral problems to earlier psychological events, it was the last bastion to be stormed by molecular genetics. Behavioral disorders are now being delineated at a molecular level, but, like the early delineation of physical genetic disorders, progress in psychiatric genetics only accounts for a small percentage of cases at present. Disorders with molecular findings include intellectual disability, schizophrenia, bipolar disorder, Alzheimer disease, autism, Parkinson disease, Huntington disease, and frontotemporal dementia.[13]

Artistic Perspective

Figure 1-34 compares two Renaissance paintings: a Florentine *Portrait of a Man with a Medal of Cosimo the Elder* by Botticelli and a Venetian *Portrait of Jacopo Soranzo* by Tintoretto. The difference in painting style is striking. In the Florentine painting, the outlines of the face are sharply drawn with a three-dimensional sculptural quality together with a sense of palpability. In contrast, in the Venetian painting, the lines of the face are soft and less distinct but with a sense of atmosphere surrounding the figure.[2]

A B

• **Figure 1-34 Left,** Close-up of a Florentine *Portrait of a Man with a Medal of Cosimo the Elder* by Botticelli. **Right,** Close-up of a Venetian *Portrait of Jacopo Soranzo*, by Tintoretto. *For stylistic interpretation, see text.* ***A,*** *From the Uffizi Gallery, Florence Inv 1890 no. 1,488;* ***B,*** *from Gallerie del' Accademia, Venice. Image source: Art Resource, NY.*

References

1. Arneson DJ: *Stories: Tweety and Sylvester*, Middleton, Conn, 1971, Xerox Education Publications. Courtesy of Warner Bros.
2. Berenson B: *Italian Painters of the Renaissance*, New York, 1976, Meridian.
3. Bonnefont J, Nikolaev SI, Perrier AL, Guo S, Cartier L, Sorce S, Laforge T, Aubry L, Khaitovich P, Peschanski M, Stylianos E, Antonarakis E, Karl-Heinz K: Evolutionary forces shape the human *RFPL 1,2,3* genes toward a role in neocortex development. *Am J Hum Genet* 83:208–218, 2008.
4. Buendia JR: *Basic Guide to the Prado*, Madrid, 1989, Silex.
5. Campbell B: *Human Evolution*, Chicago, 1970, Aldine.
6. Cohen MM, Jr: *The Child with Multiple Birth Defects*, ed 2, New York, 1997, Oxford University Press.
7. Cohen MM, Jr, MacClean RE: *Craniosynostosis: Diagnosis, Evaluation, and Management*, New York, 2000, Oxford University Press.
8. Cohen MM, Jr: Perspectives on Craniofacial Anomalies, Syndromes, and Other Disorders. In Lin KY, Ogle RC, Jane JA, editors: *Craniofacial Surgery: Science and Surgical Technique*, Philadelphia, 2002, W.B. Saunders Company, Chapter 1, pp. 3–38.
9. Cohen MM, Jr: FGFs/FGFRs and Associated Disorders. In Epstein CJ, Erickson RP, Wynshaw-Boris A, editors: *Inborn Errors of Development*, 2004. New York, 2004, Oxford University Press, Chapter 33, pp 380–400.
10. Cohen MM, Jr: *Perspectives on the Face*, New York, 2006, Oxford University Press.
11. Cohen MM, Jr: Craniofacial Disorders. In Rimoin DL, Conner JM, Pyeritz RE, Korf BR, editors: *Emory and Rimoin's Principles and Practice of Medical Genetics*, ed 5, Philadelphia, 2007, Elsevier, Chapter 151, pp 3303–3348.

12. Cohen MM, Jr: Craniofacial Abnormalities. In *Potter's Pathology of the Fetus, Infant, and Child*, ed 2, Philadelphia, 2007, Elsevier, Chapter 20, pp 885–918.
13. Cohen MM, Jr: *Mental retardation, psychiatric disorders, neurodegenerative disorders, and genetics*. PowerPoint presentation dedicated to my Fellow Angela M. Castro, MD. 2010.
14. Doubilet D: *Fishface*, New York, 2003, Phaidon Press.
15. Elffers J: *Play with Your Food*, New York, 1997, Stewart, Tabori & Chang.
16. Enlow DH: *The Human Face*, New York, 1968, Harper & Row.
17. Enlow DH: *Facial Growth*, ed 3, Philadelphia, 1990, Saunders.
18. Evans PD, Gilbert SL, Mekel-Bobrov N, Vallender EJ, Anderson JR, Vaez-Azizi LM, Tishkoff SA, Hudson RR, Lahn BT: *Microcephalin*, a gene regulating brain size, continues to evolve adaptively in humans. *Science* 309:1717–1720, 2005.
19. Ezhkova E, Fuchs E: An eye to treating blindness. *Nature* 466:567–568, 2010.
20. Fagerholm P, Lagali NS, Merrett K, Jackson WB, Munger R, Liu Y, Polarek JW, Söderqvist M, Griffith M: A biosynthetic alternative to human donor tissue for inducing corneal regeneration: 24-month follow-up of a phase 1 clinical study. *Sci Transl Med* 2:46ra61, 2010.
21. Geschwind N: Specialization of the human brain. In *The Brain: A Scientific American Book*, San Francisco, 1979, W.H. Freeman and Co., pp 108–117.
22. Gilbert CA: *All Is Vanity*. New York: House of Art.
23. Gould SJ: *Ontogeny and Phylogeny*, Cambridge, UK, 1977, Harvard University Press.
24. Hayakawa T, Angata T, Lewis AL, Mikkelsen TS, Varki, NM, Varki A: A human-specific gene in microglia. *Science* 309:1693, 2005.
25. Jirásek JE: *Atlas of Human Prenatal Morphogenesis*, Boston, 1983, Martinus Nijhoff.
26. Kenerknecht I, Grueter T, Welling B, Wentzek S, Horst J, Edwards S, Gueter M: First report of prevalence of non-syndromic hereditary prosopagnosia (HPA). *Am J Med Genet* 140A:1617–1622.27, 2006.
27. Klein RG: Whither the Neanderthals? *Science* 299:1525–1527, 2003.
28. Krings M, Capelli C, Tschentdcher F, Geisert H, Meyer S, von Haeseler A, Grossschmidt K, Possnert G, Paunovic M, Pääbo S: A view of Neanderthal genetic diversity. *Nat Genet* 26:144–146, 2004.
29. MacLaren RE, Pearson RA, MacNeil A, Douglas RH, Salt TE, Akimoto M, Swaroop A, Sowden JC, Ali RR: Retinal repair by transplantation of photoreceptor precursors. *Nature* 444:203–207, 2006.
30. Maguire AM, Simonelli F, Pierce EA, Pugh EN, Mingozzi F, Benicelli J, Banfi S, Marshall KA, Tesa F, Surace EM: Safety and efficiency of gene transfer for Leber congenital amaurosis. *New Engl J Med* 358:2240–2248, 2008.
31. Mekel-Bobrov N, Gilbert SL, Evans PD, Vallender EJ, Anderson JR, Hudson RR, Tishkoff SA, Lahn BT: Ongoing adaptive evolution of *ASPM*, a brain size determinant in *Homo sapiens*. *Science* 309:1720, 2005.
32. Moffett BC, Jr: *Syllabus for course in fundamentals of skeletal biology related to clinical dentistry*, Unpublished manuscript, 1973. University of Washington.
33. Parr LA, Heintz M: Rhesus monkey expressions. *Anim Behav* 77:1507–1513, 2009.
34. Pruzansky S: Clinical investigation of the experiments of nature. *ASHA Report* 8:62–94, 1973.
35. Radinsky LB: *The Evolution of Vertebrate Design*, Chicago, 1987, University of Chicago Press.
36. Rama P, Matuska S, Paganoni G, Spinelli A, De Luca M, Pellegrini G: Limbal stem-cell therapy and long-term corneal regeneration. *New Engl J Med* 363:147–155, 2010.
37. Scammon R: *Section 1 in Human Anatomy by H. Morris*, ed 11, Philadelphia, 1953, Blakiston.
38. Sharma RK: Developmental expression of $GABA_A$ receptors in retinal transplants. *Ophthalmic Res* 32:45–51, 2000.
39. Tassabehji M, Hammond P, Karmiloff-Smith A, Thompson P, Thorgeirsson SS, Durkin ME, Popescu NC, Hutton T, Read AP, Macconochie M, Donnai D: *GTF2IRD1* in craniofacial development of humans and mice. *Science* 310:1184–1187, 2005.
40. Thumann G: Nonviral gene therapy for age-related macular degeneration. *Expert Rev Ophthalmol* 6:81–93, 2011.
41. Tibbetts EA, Dale J: A socially enforced signal of quality in a paper wasp. *Nature* 432:218–222, 2004.
42. Witten PE, Hall BK: Seasonal changes in the lower jaw skeleton in male Atlantic salmon (*Salmo salar L.*): Remodeling and regression of the kype after spawning. *J Anat* 203:435–450, 2003.

2

Pioneers and Milestones in the Field of Orthodontics and Orthognathic Surgery

JEFFREY C. POSNICK, DMD, MD

- Pioneers in the Field of Orthodontics
- Early Pioneers in the Field of Orthognathic Surgery
- Hugo L. Obwegeser: Development of the Standard Orthognathic Procedures
- William H. Bell: Experimental Studies to Confirm the Biologic Basis of the Standard Orthognathic Procedures
- Hans G. Luhr: Development of Plate and Screw Fixation for Craniomaxillofacial Surgery
- Paul Louis Tessier: Development of Craniofacial Surgery
- Conclusions

Pioneers in the Field of Orthodontics

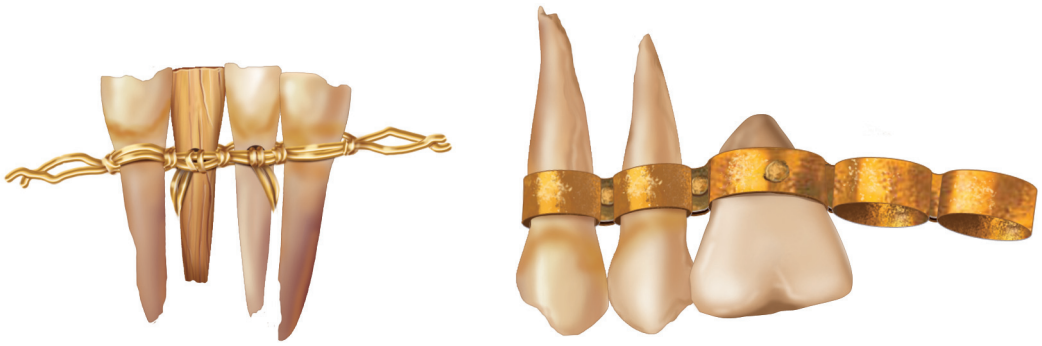
Background

Orthodontic appliances that are somewhat primitive but surprisingly well-designed for their time have been found in Ancient Greek written materials.[8] Four Etruscan specimens of young adult women dating back to 900 B.C. that specifically demonstrate appliances on the teeth have also been recovered.[23,208] The orthodontic type appliances are gold bands around teeth that are adjacent to an edentulous gap. The appliances appear to be for the purpose of managing the space left by tooth loss earlier in life (Fig. 2-1).[209] In addition, the Ancient Greek scholars Hippocrates and Aristotle both have writings that discuss various ways to straighten teeth and to treat various dental conditions.[20,51] Knowledge of dental extractions carried out for the purpose of mechanically straightening the remaining teeth was also reported by Leonardo da Vinci during the High Italian Renaissance.[166] The bandolet or bandeau, which was created in 1723 by Pierre Fauchard of France, was the first documented dental arch expansion appliance (Fig. 2-2).[202] It consisted of a heavy maxillary labial arch wire to which the teeth were ligated.[202] The term *orthodontia* is said to have been coined in 1841 by Lafoulon, and it appeared in the book about malocclusion written by J.M. Alexis Schange.[11,157,203]

In 1858, perhaps the first formal article on orthodontics published in a medical journal was written by Norman W. Kingsley (Fig. 2-3). By the 1880s, Norman Kingsley's *Treaties on Oral Deformities as a Branch of Mechanical Surgery* systematically described the current techniques of the day for the repositioning of teeth (Fig. 2-4).[109] Kingsley was among the first to fully describe how extraoral forces (e.g., headgear) could be used to correct protruding maxillary anterior teeth. His emphasis was primarily on the alignment of the anterior maxillary teeth in an attempt to improve facial appearance. To Kingsley, the extraction of teeth for the purpose of uncrowding and to reduce the protraction of the incisors was acceptable practice. Details of how the upper and lower teeth articulated with each other (i.e., occlusion) were of secondary importance. He used a wide variety of apparatus to reposition the anterior teeth, including wires, bands, custom palatal plates, elastics, wedges, linen twine, rubber tubing, leathers, gold bands, and vulcanite. Impressions of the teeth and dental models were used during planning and for the precontouring of labial placed wires. The use of an external apparatus placed over the top of the head and then either secured to the chin or held with bands secured to the maxillary incisors became part of his everyday practice (Fig. 2-5).

Kingsley's basic principles for the mechanical alignment of the teeth can be summarized as follows:

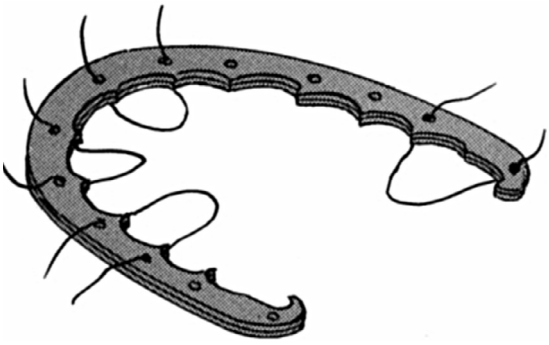
- The positioning of the six maxillary anterior teeth with regard to each other and in relationship to the lip is essential for facial aesthetics.
- The contact of a certain number of maxillary and mandibular posterior teeth, at least on one side, is important for the grinding and mastication of food.



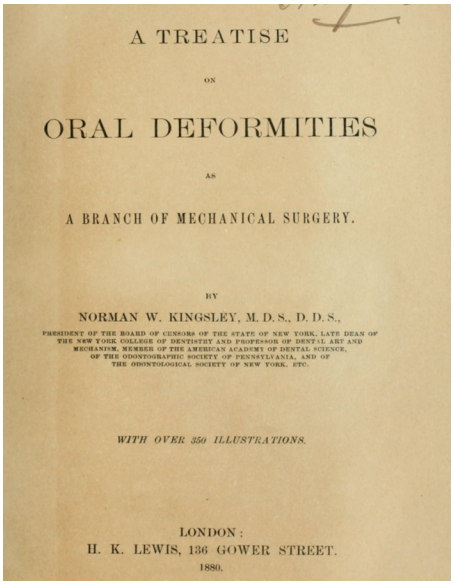
A Ancient Grecian appliance

B Ancient Etruscan appliance

• **Figure 2-1 A,** A drawing of the anterior mandibular dentition in a mummy specimen from ancient Greece (approximately 1000 B.C.) currently housed in the Archaeological Museum in Athens, Greece. A gold wire was used to tie teeth together and to band others to support replacements. The use of human, animal, carved wood, bone, or ivory for replacement teeth was practiced by the ancient Grecian society. **B, A** drawing of an ancient Etruscan orthodontic appliance found on the maxilla of a mummy in a tomb (approximately 700 B.C.), which is currently at the Civic Museum in Cornet, Italy. It demonstrates gold soldered rings and rivets to hold dental replacements as a bridge. The specimen has two natural teeth, one riveted oxtooth and four spaces for others



• **Figure 2-2** Illustration of the "bandolet" bandeau, which was created in 1723 by Pierre Fauchard of France. This was the first documented dental arch expansion appliance.



A TREATISE

ON

ORAL DEFORMITIES

AS

A BRANCH OF MECHANICAL SURGERY.

BY

NORMAN W. KINGSLEY, M. D. S., D. D. S.,

PRESIDENT OF THE BOARD OF CENSORS OF THE STATE OF NEW YORK, LATE DEAN OF THE NEW YORK COLLEGE OF DENTISTRY AND PROFESSOR OF DENTAL ART AND MECHANISM, MEMBER OF THE AMERICAN ACADEMY OF DENTAL SCIENCE, OF THE ODONTOGRAPHIC SOCIETY OF PENNSYLVANIA, AND OF THE ODONTOLOGICAL SOCIETY OF NEW YORK, ETC.

WITH OVER 350 ILLUSTRATIONS.

LONDON:
H. K. LEWIS, 136 GOWER STREET.
1880.

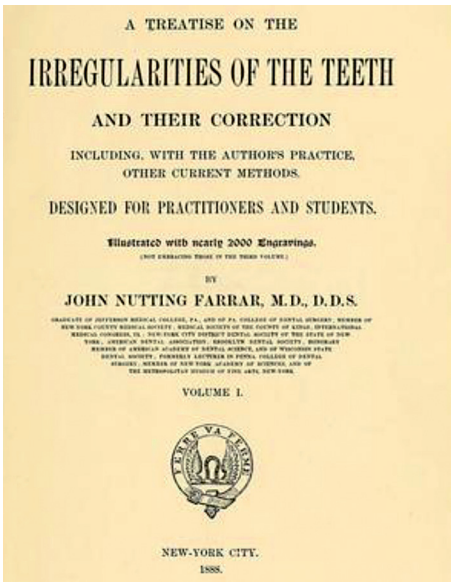
• **Figure 2-4** Norman Kingsley's text entitled *A Treatise on Oral Deformities as a Branch of Mechanical Surgery,* which was published in 1880.

• **Figure 2-3** Photograph of Norman W. Kingsley (1829-1913).

- All other teeth are potentially expendable, depending on the effort required by the clinician for straightening and the individual's desire to maintain them.
- From both a mastication and an aesthetic perspective, there is no particular need for the maintenance of all 32 teeth or to achieve a specific and detailed way of the upper and lower teeth occluding with one another.

Dr. Kingsley also commented on facial aesthetics, stating that "the eye soon tires of the stiffness and formality of unbroken uniformity, and is only permanently pleased with the beauty which comes from graceful variation."[109] He acknowledged the occurrence of jaw deformities when he said that "[a] lack of harmony between the maxilla and mandible is occasionally seen. For instance, when the

• **Figure 2-5** Headgear designed and used by Norman W. Kingsley. According to Kingsley, "I sat to work to make an apparatus that would pull the lower jaw back. As you can see from the photograph taken at the time she was wearing the apparatus, it consists of two parts. For the lower part, I made a brass plate to fit the chin, having arms with hooked ends reaching to a point just below the point of the chin. These arms were arranged in such a way that the distance between them could be altered at will by simply pressing them apart or to gather. The upper part consisted of a simple network going over the head and having two hooks on each side, one hook being above and the other below the ear. When the apparatus was completed and in use, there were four ligatures of ordinary elastic rubber pulling in such a way as to force the lower jaw almost directly backward." *From Kingsley NW:* A treatise on oral deformities as a branch of mechanical surgery, *London, 1880, H.K. Lewis, pp 137, Figure 68.*


A TREATISE ON THE

IRREGULARITIES OF THE TEETH

AND THEIR CORRECTION

INCLUDING, WITH THE AUTHOR'S PRACTICE, OTHER CURRENT METHODS.

DESIGNED FOR PRACTITIONERS AND STUDENTS.

Illustrated with nearly 2000 Engravings.

BY

JOHN NUTTING FARRAR, M.D., D.D.S.

VOLUME I.

NEW-YORK CITY.

1888.

• **Figure 2-6** John Nutting Farrar's two-volume illustrated text entitled *A Treatise on the Irregularities of the Teeth and Their Correction,* which was published in 1897.

• **Figure 2-7** Photograph of Edward H. Angle (1855-1930).

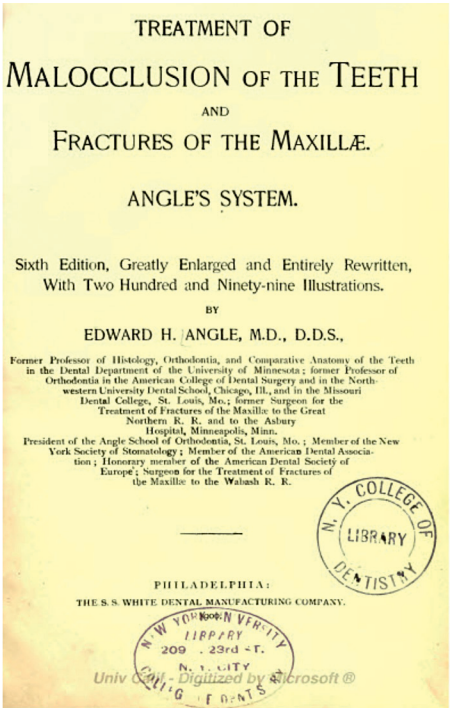
mandible is very large and massive or unusually small in comparison to the upper jaw. When present, this will interfere with the harmony of the surrounding parts of the face."[109]

In 1897, J.N. Farrar published a two-volume illustrated text entitled *A Treatise on the Irregularities of the Teeth and Their Corrections* (Fig. 2-6).[12] In that text, he also demonstrates detailed orthodontic appliances, and he was among the first to suggest the advantages of mild (rather than heavy) force applied at intervals to effectively move the teeth.[10]

Edward H. Angle

It was common practice through the later half of the 19th century to extract the teeth in response to most dental problems.[9,60] Restorative dentistry was generally not the first choice for the management of "toothaches." Most adults at that time (i.e., the 1880s) did not have a full complement of teeth nor was there much stigmata associated with partial edentulism.[210] Details of how the teeth occluded with each other seemed important only when establishing how upper and lower dentures were to meet so as to prevent loosening or mobility when wearing them and to improve chewing ability. It was a natural extension of this thinking for a prosthodontic-trained dentist like Edward H. Angle to develop a more holistic concept regarding the importance of the occlusion of the natural dentition (Fig. 2-7).[5-8] During his career, Angle taught prosthetics at the University of Minnesota (Minneapolis, Minn), Northwestern University Dental School (Chicago, Ill), and the Missouri Dental College (St. Louis, Mo). Dr. Angle's interest in dental occlusion and his ingenuity and dedication with regard to the correction of malocclusion led to the development of the specialty that we now call "orthodontics."

Angle's textbook, *Treatment of Malocclusion of the Teeth,* was first published in 1887 (Fig. 2-8).[6] It went into seven much-revised editions, and it laid the foundation for the modern specialty of orthodontics. The publication of Angle's classification of malocclusion during the 1890s was a key step in the development of orthodontics (Fig. 2-9).[42,43]


TREATMENT OF

MALOCCLUSION OF THE TEETH

AND

FRACTURES OF THE MAXILLÆ.

ANGLE'S SYSTEM.

Sixth Edition, Greatly Enlarged and Entirely Rewritten, With Two Hundred and Ninety-nine Illustrations.

BY

EDWARD H. ANGLE, M.D., D.D.S.,

Former Professor of Histology, Orthodontia, and Comparative Anatomy of the Teeth in the Dental Department of the University of Minnesota; former Professor of Orthodontia in the American College of Dental Surgery and in the North-western University Dental School, Chicago, Ill., and in the Missouri Dental College, St. Louis, Mo.; former Surgeon for the Treatment of Fractures of the Maxillæ to the Great Northern R. R. and to the Asbury Hospital, Minneapolis, Minn. President of the Angle School of Orthodontia, St. Louis, Mo.; Member of the New York Society of Stomatology; Member of the American Dental Association; Honorary member of the American Dental Society of Europe; Surgeon for the Treatment of Fractures of the Maxillæ to the Wabash R. R.

PHILADELPHIA:

THE S. S. WHITE DENTAL MANUFACTURING COMPANY.

N. Y. COLLEGE OF DENTISTRY LIBRARY
NEW YORK UNIVERSITY LIBRARY 209 . 23rd ST. N. Y. CITY
Univ Calif - Digitized by Microsoft ®

• **Figure 2-8** Dr. Angle published a comprehensive text entitled *A Treatment of Malocclusion of the Teeth and Fractures of the Maxillae,* the first edition of which was published in 1887. It went into seven revised editions, and it laid the foundation for the modern specialty of orthodontics.

Angle introduced the first precise and simple definition of the normal human occlusion of the natural dentition. He postulated that a key aspect of how the teeth should fit together was the positioning of the maxillary first molars. He then went on to describe three classes of malocclusion that were based on the occlusal relationships of the first molars:

Class I: There is a normal relationship of the molars, but the line of occlusion is incorrect as a result of malposed teeth, rotations, or other causes.

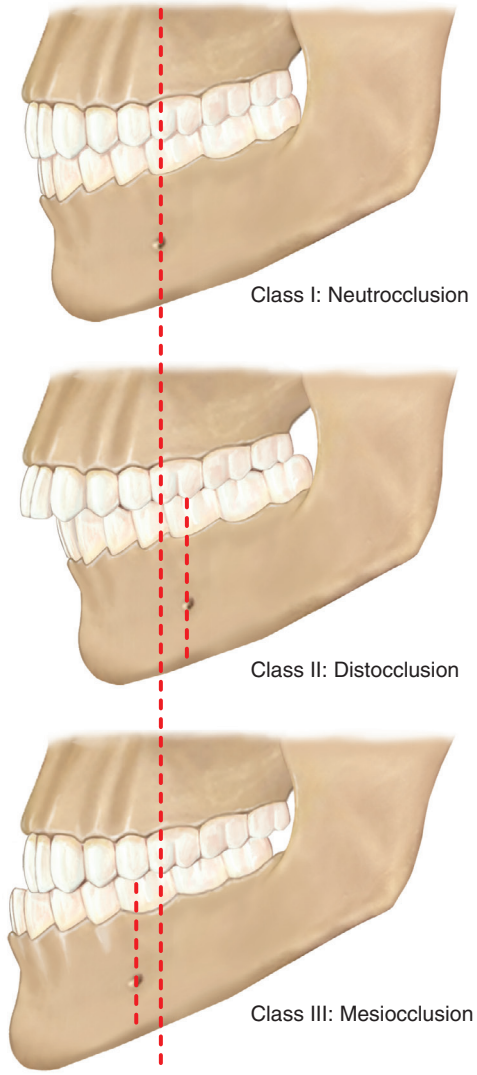
Class II: The lower molar is distally positioned relative to the upper molar, and the line of occlusion is not specified.

Class III: The lower molar is mesially positioned relative to the upper molar, and the line of occlusion is not specified.

Angle's classification actually has four classes:

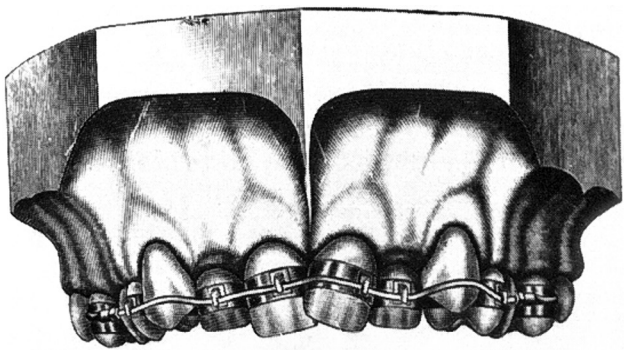
- Normal occlusion (Class I, with a normal line of occlusion)
- Class I malocclusion
- Class II malocclusion
- Class III malocclusion

Normal occlusion and Class I malocclusion share the same molar relationship, but they differ with regard to the arrangement of the other teeth relative to the line of occlusion. The line of occlusion may or may not be correct in Class II and Class III.

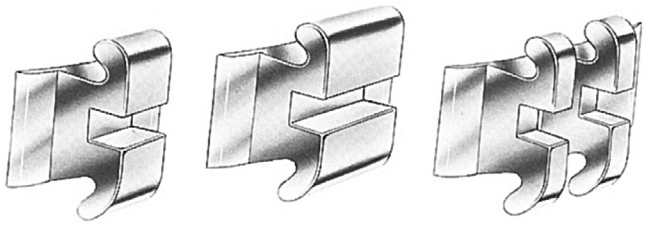


• **Figure 2-9** Angle described three classes of malocclusion based on the relationships of the first molars: Class I, Class II, and Class III.

By the early 1900s, orthodontists no longer just aligned irregular teeth in each arch.[175,218] The practice had now evolved into the treatment of malocclusion (i.e., how the upper and lower teeth align with each other). Angle's philosophy was to achieve a specific relationship between the upper and lower teeth for each patient. Since Angle's precisely defined dental relationships also required a full complement of teeth in each arch, the maintenance of an intact dentition became an important component of orthodontic treatment. Angle and his followers strongly opposed extraction for orthodontic purposes.[69] In an attempt to achieve the three-axis control of tooth movement, Angle introduced the *ribbon arch* (Fig. 2-10).[70] He then devised the *edgewise appliance* to overcome the weaknesses of the ribbon arch (Fig. 2-11). The Angle reoriented bracket slot was designed to control the bodily movement of teeth and to limit tipping.[135] Angle also heavily relied on the use of intraoral



• **Figure 2-10** The ribbon arch appliance, which was introduced by Angle in 1916. The vertical slot in the bracket accepts either round or rectangular wire, which in turn is held in place by a locked pin.



• **Figure 2-11** The edgewise bracket attachment comes in a variety of modifications. The original single bracket *(left)* has been widened for molar teeth and for greater rotational control *(center)*. The twin edgewise modification *(right)* is available in different widths, and it is used most frequently because of its greater versatility, its reduced frictional component, and its adaptability to various light-wire techniques.

rubber bands and limited the use of extraoral appliances (e.g., headgear) as much as possible.

Angle was among the first to state that jaw discrepancies exist in some individuals and that these discrepancies will prevent the achievement of a normal occlusion. He then acknowledged the need for jaw-straightening surgery. In Chapter 14: Operative Surgery of the sixth edition of Angle's textbook, he states that the use of appliances to move teeth may be properly called *conservative surgery* to distinguish it from more bold or aggressive operations involving the use of cutting instruments, which were designated as *operative surgery*. He states the following: "While such operations [mandibular osteotomies] should probably be employed only as auxiliary to the conservative method, they are doubtless destined to play an more important part in the practice in the future."[6]

Dr. Angle worried that no technique that relied solely on tooth movement could establish the appropriate relationships of the teeth or truly improve the facial lines in cases involving the severe overdevelopment of the inferior maxilla [mandible].[6] He felt that these cases may be successfully treated by removing a section of bone from each of the lateral halves of the mandible. At the time, the removal of a single complete section of the jaw had been reported for cases involving such conditions as ankylosis, tumors, and gunshot wounds, but Dr. Angle was unable to find information about the removal of complete sections from each of the lateral halves of the mandible. This idea was discussed by Dr. Angle with various surgeons and dentists and determined to be feasible. His proposal involved the following[6]:

- Careful photographs should first be taken of the patient.
- Two accurate models should be made of the lower dental arch, and one should be made of the upper.
- One of the plaster models of the lower jaw should then be sawed through and the sections removed.
- The positions and extent of the sections should be carefully experimented with until the three remaining sections of the model could be made to best harmonize with the upper arch so that the teeth may be in best possible occlusion with those of the upper jaw.
- The sections of the plaster model should then be cemented or waxed together.
- Over this reconstructed model, a vulcanite or metal splint should then be formed.
- With the use of careful comparisons and measurements of the reconstructed model as compared with the unchanged model, the exact size and form of both resections of bone to be removed can be determined so that there need be no guessing as to the relationships of the bone. In this way, complete apposition of the cut ends can be made.
- Because there is a lingual inclination of the lower incisors in all cases of mandibular prognathism, the sections of bone to be removed must not be parallel on their sides but instead more or less wedged or V-shaped to gain the best positions for the occlusion of the incisors as well as for the appearance of the chin.
- The degree of variation from the parallel of the lines of bone resection must be determined by the conditions that are present (i.e., patient variation).
- Because the operation must be skillfully performed, the most rigid support should be given to the reconstructed jaw. This technique (i.e., a cap splint over the teeth) should give more rigid support than is possible with the crude, unstable, and unmechanical plan of wiring the ends of the bone together, which is so often employed for the reduction of fractures.

According to Dr. Angle, "The question most often raised by dentists in discussing the practicability of an operation as outlined above was the uncertainty as to union of the bones and as to impairment of vitality of the teeth in the anterior segment [of the mandible]."[36] However, Dr. Angle helped with the overcoming of these concerns via two case reports that were presented in his textbook, *Treatment of Malocclusion of the Teeth and Fractures of the Mandible: Angle's System*[6] (pp 173-184):

First Patient (Operation)

June 23, 1897, the author [Dr. Angle] assisted the late Professor Henry H. Mudd in one of these operations performed at St. Luke's Hospital, St. Louis, upon Ms. M.J. of Arkansas, a delicate girl, 13 years old. She had likely suffered with generalized sepsis at 3 years of age

with the mandible becoming fixed and firmly closed [temporomandibular joint ankylosis]. The upper jaw was normal in size and contained a full complement of teeth. The lower jaw was deformed. The ramus of the jaw on each side passed downward and backwards so that the angle of the jaw came a little behind rather than in front of the vertical line dropped from the lobe of the ear. The arch of the lower jaw was broad and the incisor teeth were impinging the surface of the palate. The only point of entrance for food was the opening under the arch of the hard palate above the incisor teeth of the lower jaw.

To free the body of the jaw so that the mouth might be opened, a triangular segment with the base downward and the apex upward and forward was removed from the angle of the jaw on each side. A complete section of the bone was thus removed on each side to make false joints at the junction of the ramus and the body of the mandible. When the bone was separated, the tongue and jaw dropped down, making respiration difficult. An urgent tracheostomy was made. Wire ligatures were used to establish IMF [intermaxillary fixation]. This pulled the tongue and neck muscles superior and anterior, and established a good airway. The ligatures were occasionally removed in the ensuing weeks to allow movement of the jaw and establish a hinge joint instead of osseous union in the ramus regions of the mandible. Unfortunately, the patient was lost to follow-up several weeks later without permanent record of how things turned out.

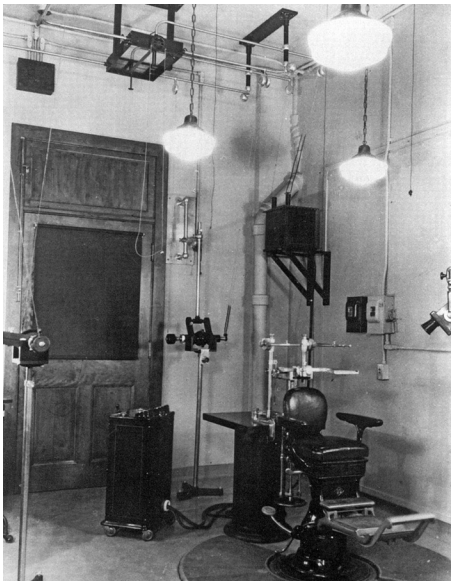
Second Patient (Operation)

A double resection [of the mandible] for the purpose of shortening a prognathic mandible was performed successfully at the Baptist Hospital in St. Louis [Dr. Vilray Blair was the surgeon, and the procedure was performed on December 19, 1897]. Such an operation might at first seem formidable, but there is no reason why, if skillfully performed according to modern aseptic methods of surgery and the plan is indicated, that clean smooth ends of the bone should not unite at least as readily as they do in the common cases of double [mandibular] fractures and the result being nearly ideal.

Holly Broadbent

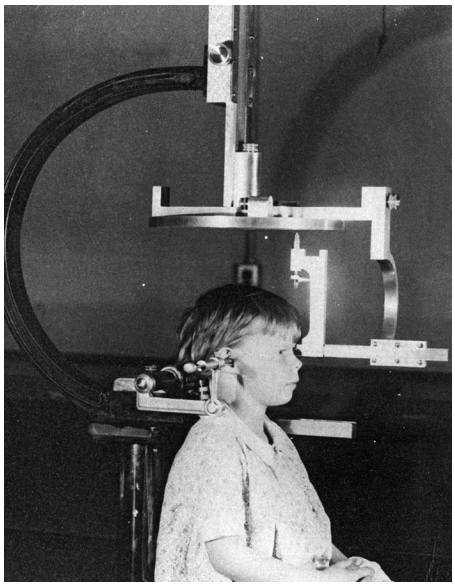
The introduction of the cephalometer by Holly Broadbent in 1931 was another important milestone that placed orthodontic research on a scientific foundation (Fig. 2-12).[157] For the first time, it made possible the accurate study of the facial bones in the growing child (Figures 2-13 and 2-14). It became a valuable supplement to the orthodontist's plaster models and intraoral x-rays. The value of serial cephalograms in clinical practice was quickly realized. Broadbent's original work on the "face of the normal child" and Brody's classic research "on the growth pattern of the human head from the third month to the eighth year of life" were among the earliest contributions.[163] These were followed by the research of Down's "Variations in Facial Relationships,"

• **Figure 2-12** Photograph of Holly Broadbent (1894-1977). *From Broadbent BH Sr., Broadbent BH Jr and Golden WH: Bolton Standards of Dentofacial Developmental Growth, St. Louis, C.V. Mosby, 1975.*



• **Figure 2-13** Broadbent was the first to develop the standardized lateral "head plate." The cephalometer is a device for holding the patient's head, the x-ray film, and the central ray of the x-ray machine in proper relationship with one another. Ear-rod extensions are necessary to adjust the head so that the profile is centered, regardless of the size or shape of the head. The orbital pointer and the ear rods adjust the patient's head along the horizontal or Frankfort plane. An adjustable chair is used, and the teeth are placed in "centric occlusion" unless otherwise indicated. The cephalometer may be rotated so that posterior and anterior radiographs as well as profile radiographs can be taken. The central ray is directed through the ear rods, thereby producing a circle on the x-ray film. *From Broadbent BH Sr., Broadbent BH Jr and Golden WH: Bolton Standards of Dentofacial Developmental Growth, St. Louis, C.V. Mosby, 1975.*

the work of Thompson in "Functional Analysis of Occlusion," Wylie's "Assessment of Anterior-Posterior Dysplasia," and the work of Margolis' "Basic Facial Pattern and Its Application in Clinical Orthodontics."[174] The use of cephalometric radiography in clinical practice became widespread



• **Figure 2-14** The cephalostat device, as described in Figure 2-13, is shown with a patient who is positioned and ready for film exposure. *From Broadbent BH Sr., Broadbent BH Jr and Golden WH: Bolton Standards of Dentofacial Developmental Growth, St. Louis, C.V. Mosby, 1975.*

after World War II and made clear to all clinicians that many of the Class II and Class III malocclusions being treated actually resulted from abnormal skeletal relationships rather than just malposed teeth.[165] These radiographic truths were hard to ignore and further inspired orthodontists and surgeons to search for operative solutions.[181]

Charles Tweed and Raymond Begg

Another "orthodontic truth" was also soon to fall. A purest "Angle" approach to maintaining and straightening all of the teeth in all patients frequently proved untenable with regard to the maintenance of the occlusion after the appliances were removed.[54,144,145] This non-extraction, arch-expansion approach gave little consideration to long-term periodontal health or to the individual's overall facial aesthetics.[161] The extraction of teeth (typically the bicuspids) to achieve occlusal stability and periodontal health was reintroduced into American orthodontics during the 1930s by Charles Tweed (Fig. 2-15) and simultaneously into the United Kingdom (Australia) by Raymond Begg (Fig. 2-16) (see Chapter 17 for in-depth discussion).[47-49,59,204]

Lawrence F. Andrews

Dr. Lawrence F. Andrews' pioneer contributions to the field of orthodontics and the treatment of dentofacial deformity must also be mentioned (Fig. 2-17).[2-4] After completing orthodontics training at Ohio State University in 1958, he entered private practice that was limited to orthodontics. Early in his career, Dr. Andrews recognized clinical problems that were yet unsolved and sought solutions. This sent him

• **Figure 2-15** Photograph of Charles Tweed (1895-1970).

• **Figure 2-16** Photograph of Raymond Begg (1889-1983). *From Wahl N: Orthodontics in 3 millennia. Chapter 6: More early 20th-century appliances and the extraction controversy.* Am J Orthod Dentofacial Orthop *128:795–800, 2005.*

• **Figure 2-17** Photograph of Lawrence F. Andrews.

on a search to better understand occlusion as well as deviations from normal. Between 1960 and 1964, Dr. Andrews began to gather data for analysis. One hundred and twenty non-orthodontic normal (dental) models were acquired with the cooperation of local dentists, orthodontists, and a major university. The models selected for study were of teeth that had never undergone orthodontic treatment; that were straight and pleasing in appearance; that had a bite that looked generally correct; and that, in Dr. Andrews' judgment, would not benefit from orthodontic treatment.

Dr. Andrews studied the crowns of this multisource collection of models to ascertain which characteristics, if any, would be found consistently in all of them. Angle's molar cusp groove concept was validated but found to be incomplete. After meticulous evaluation, including comparison with "treated cases" from the nation's most skilled orthodontists (n = 1150), six consistent occlusal characteristics were formulated by Dr. Andrews in general terms and then explained in detail.[2] The six fundamental qualities or *Six Keys of Optimal Occlusion* were validated not solely because all were present in each of the non-orthodontic normal models (n = 120) but because the lack of even one of the six was a defect that was predictive of an incomplete end result in the orthodontically treated models that were studied (n = 1150).

The significant characteristics (i.e., the *Six Keys of Optimal Occlusion*) described by Andrews that were found in all of the non-orthodontic normal models were as follows:

Key I: Interarch (molar) relationship
Key II: Crown angulation
Key III: Crown inclination
Key IV: Rotations
Key V: Tight contacts
Key VI: Curve of Spee

In his published article, Dr. Andrews acknowledged that, although each normal person is unique in his or her own way, they nevertheless have much in common.[2] The 120 non-orthodontic normal models studied differed with regard to certain aspects, but all shared the Six Keys. Dr. Andrews suggested that we use as our benchmark "nature's best" and that, in the absence of abnormalities outside of our control, we limit compromise and strive for the ideal. He developed the *straight-wire appliance* in 1970 for orthodontic use to help accomplish these objectives. This preadjusted appliance soon became the standard of the specialty.[2-4]

Dr. Andrews then went on to describe six fundamental principles that are necessary to achieve both facial and dental harmony: the *Six Elements of Orofacial Harmony*. Both of these aspects—facial and dental harmony—are frequently absent in the patient with dentofacial deformity. Dr. Andrews was one of the first orthodontist to clarify the importance of addressing both facial and dental harmony from the beginning of treatment to achieve the best facial form and head and neck function for each patient. Dr. Andrews stated that, for the maxillomandibular complex to be in harmony (i.e., to have all elements in alignment) with the overall face, the following must be present:

Element I: Proper arch shape and positioning of the maxillary and mandibular teeth (roots) over the basal bone
Element II: Proper horizontal (sagittal) projection of the maxilla
Element III: Proper width of the maxillary and mandibular arches
Element IV: Proper vertical height of the maxilla
Element V: Proper prominence (shape) of the chin (i.e., pogonion prominence)
Element VI: Establishment of the *Six Keys to Optimal Occlusion*

He studied each of these Six Elements and defined both qualitatively and quantitatively how the orthodontist and the surgeon can work together to achieve these objectives for each patient.

William R. Proffit

By the mid 1970s, Dr. Proffit recognized the benefits of a collaborative surgeon–orthodontist interaction for the correction of dentofacial deformities.[164] He and others convincingly demonstrated to surgeons that rectangular orthodontic arch wires in edgewise brackets were satisfactory for surgical patients without the need for the intraoperative placement of Erich arch bars. This paved the way for the routine preoperative orthodontic relief of dental compensations, and it was followed by the placement of orthodontic "surgical wires and hooks" for intraoperative use. After the initial surgical healing, a smooth transition to orthodontic maintenance and detailing soon became the standard of care. Dr. Proffit was also an early believer in the importance of a collaborative effort to understand the patient's presenting dysfunction and dysmorphology and then to develop a comprehensive treatment plan. The use of a cephalometric analysis with prediction tracings followed by dental model planning with splint construction became the routine. Dr. Proffit and others also insisted on analysis of the early results that were achieved. This was followed by critical studies of the long-term outcomes after various surgical approaches and orthodontic techniques were employed. This analytic approach has been responsible for much progress in the field over the past four decades.

According to Dr. William R. Proffit* (Fig. 2-18), orthodontics in the 21st century differs from that of the previous century in three fundamental ways[162,163]:

1. *There is more emphasis on both facial and dental aesthetics and less on the details of dental occlusion as an overriding objective.* This has much to do with the safe and reliable orthognathic surgical techniques that are currently

*Please note that the author of this text has paraphrased Dr. Proffit's original work.

• **Figure 2-18** Photograph of William R. Proffit (1936-Present)

available and with the recognition that long-term stable occlusion and periodontal health can best be achieved in conjunction with harmonious jaw relationships. This will require extractions when necessary to uncrowd and achieve periodontal health; decompensating orthodontic therapy to place teeth that are solidly in the alveolar bone; and jaw-straightening surgery, when indicated.

2. *Patients and families expect greater involvement in the treatment planning process*. Full disclosure requires the orthodontist to consider and review ideal and compromised treatment options. Providing a balanced and objective assessment of each option's effect on facial aesthetics, dental stability, periodontal health, speech articulation, and the upper airway is ideal. Consultation with appropriate dental, surgical, and medical specialists before instituting treatment has become the standard of care.
3. *Orthodontics is now offered not just to children, adolescents, and young adults but also to older adults through an interdisciplinary approach.* Consideration should be given to facial aesthetics, occlusion, periodontal health, the upper airway, speech articulation, and the long-term maintenance of the dentition. There is increased emphasis on coordinated treatment with other dentists, surgeons, and medical specialists. Taking advantage of currently available sophisticated orthodontics and orthognathic surgery to provide individuals of all ages with quality care should now be considered routine.

Early Pioneers in the Field of Orthognathic Surgery

Mandibular Setback for Prognathism

Simon Hullihen described a procedure for the correction of mandibular dentoalveolar protrusion in the *American Journal of Dental Science* in January 1849 (Fig. 2-19, *A*).[18,98] Technically, this was a bilateral bicuspid region wedge osteotomy to "set back" the anterior mandibular dentoalveolar segment. The osteotomy did not violate the inferior border of the mandible. The procedure was performed on a middle-aged woman whose mandibular deformity was the result of a severe burn scar contracture of the anterior neck and lip from an injury that had occurred during her childhood (see Fig. 2-19, *B* and *C*).

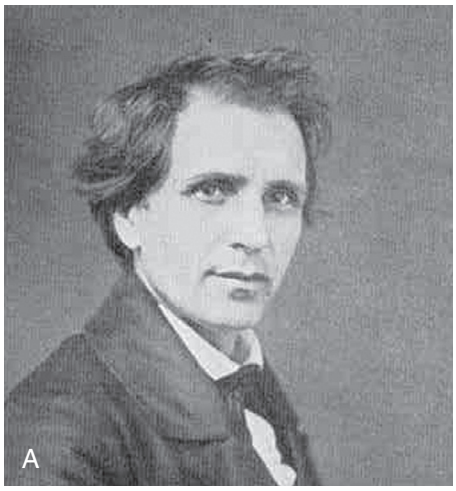
In 1887, Berger from Lyon, France, described bilateral condylar neck osteotomies to set back the prognathic mandible.[90] In 1895, Jaboulay also reported the bilateral osteotomy of the condylar neck (condylectomy) for the correction of prognathism.[100] This method continued to be popular in France for the treatment of prognathism through the 1950s.[75]

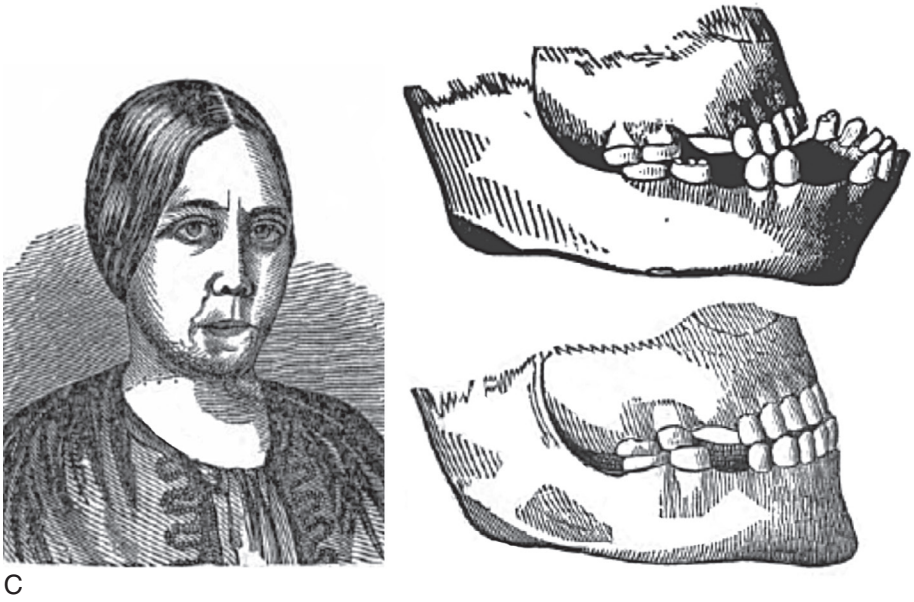
On December 19, 1897, Vilray Blair in St. Louis, Mo, carried out a distinct modification of the original Hullihen procedure for the treatment of mandibular prognathism (see the "Second Patient (Operation)" case report by Dr. Angle earlier in this chapter) (Fig. 2-20).[38] The operation was performed on a 22-year-old man with asymmetric mandibular prognathism. The young man had difficulty with chewing and speech articulation, and he also was self-conscious about his appearance; thus, he was enthusiastic about proceeding with the recommended surgical plan. Dr. Blair stated that the immediate surgical objective was to carry out bilateral body osteotomies to shorten the horizontal ramus [body of the mandible] and to change the mandibular angle [rotate the distal mandible counterclockwise], thereby allowing for the closure of the open bite. Dr. Blair's preoperative concerns about the operation included the following:

1. Could a portion of the mandible be cut out on each side in a satisfactory way?
2. Would sacrifice of the inferior dental nerve cause detrimental injury to the distal mandible?
3. After healing had occurred, would the mandible continue to grow and cause a recurrence of the deformity?

Through cadaver dissections, Dr. Blair satisfied his concerns regarding whether the mandible could be sectioned and repositioned successfully. As a result of the age of the patient (22 years), he had little concern regarding further growth after surgery. He conferred with two other surgeons (Dr. E.H. Gregory and Dr. P. Tupper), both of whom concurred that the surgical plan seemed reasonable. The patient was also under the care of Dr. J.W. Whipple (orthodontist). Dr. Blair had discussed the feasibility of a surgical solution for mandibular prognathism with Dr. Angle (the patient had also consulted with Dr. Angle before surgery), and Dr. Angle "had advised such an operation."[38]

The surgery was carried out at the Missouri Baptist Sanatorium. The surgery commenced with the patient under chloroform anesthesia at 9:20 AM with the patient in the recovery room by 10:30 AM. During the operation, the patient's head was hyperextended over the end of the operating room table. Skin incisions below the inferior border of the mandible on each side provided exposure. The incisions extended up into the mouth and involved cutting through





• **Figure 2-19 A,** Photograph of Simon Hullihen (1810-1897). **B,** Illustration of anterior mandibular dento-alveolar segmental osteotomy proposed and **C,** carried out by Dr. Hullihen. **A** *from Ambrecht EC: Hullihen, the oral surgeon.* Int J Orthod Oral Surg *23:377, 511, 598, 711, 1937.* **B** *and* **C** *from Hullihen S: Case of elongation of the under jaw and distortion of the face and neck, caused by a burn, successfully treated.* Am J Dent Sci *9:157, 1849.*

• **Figure 2-20** Photograph of Vilray Blair (1871-1955).

the mucous membrane. A full-thickness osteotomy was to be completed on the left side of the mandible between the first and second bicuspids and on the right side between the second bicuspid and the first molar. The osteotomy was initiated with a "double-bladed saw" three quarters of the way through on each side; the inferior border remained intact.[38] This allowed for the presence of a stable mandible on each side before the cut was fully completed on either side. A drill was used to place holes on either side of the osteotomy site for eventual "cooper wire fixation"[38] near the inferior border on each side before cutting through the inferior border on either side. After the completion of both full-thickness osteotomies, soft gutta-percha was placed over the mandibular dentition, and the upper and lower teeth were firmly wired together through the gutta-percha. This method of intermaxillary fixation immobilized the distal mandible in its new position for the closure of the open bite. Dr. Blair stated that "this method of fixation was suggested to him by seeing Dr. Angle's fracture bands."[38] The fracture band method of mandible fracture management was commonly in use.

Dr. Blair stated that, as the patient was emerging from the anesthesia, the vomiting of mucus and blood occurred, thereby necessitating the cutting of the intermaxillary fixation.[38] After the vomiting subsided, rather than reapplying intermaxillary fixation, a Barton bandage of plaster was used to secure the mandible into occlusion with the maxilla.[21] When the Barton bandage was removed several weeks later, Dr. Blair felt that there was "bony union on the right side and a very slight but perceptible motion on the left side when the fragments were grasped."[38] Dr. Blair decided to take the patient to the office of the treating dentist (orthodontist), Dr. Whipple. He requested that Dr. Whipple place two dental bands with a bar between them across the weak left osteotomy site. This would afford the patient sufficient support and allow him to go without the additional protection of a Barton bandage or intermaxillary fixation." Unfortunately, while he was tightening the bands across the left and right osteotomy sites, Dr. Whipple "broke the union of the provisional callus."[38] At this point, Dr. Whipple applied properly adjusted bands to the teeth on the left side of the mandible, and Dr. Blair reapplied a Barton plaster external facial bandage.[38] Dr. Whipple also placed crowns on the mandibular teeth posterior to the osteotomy sites so that the molars would occlude properly with the upper teeth, and he filled some points on the teeth to improve the occlusion of the incisors.[38] Apparently, the combination of the mandibular setback with the clockwise rotation resulted in "a small [osteotomy] gap between the second bicuspid and first molar on the right side" but "without a gap [previous edentulous space] in the left first bicuspid region."[38] Despite these difficulties, the osteotomies went on to heal with a reasonable correction of the occlusion.[38] Dr. Blair stated that there was "almost a complete loss of cutaneous sensation of the lower lip" but that the "sensation of the tooth-pulp is good."[38]

Interestingly, Dr. Whipple, the treating dentist and orthodontist, published his version of the patient's surgery, convalescence, and ultimate result in *Dental Cosmos* in 1897.[212] This was before the journal publication by Dr. Blair, in which he gave a full account of the case from his perspective.[38] Although Dr. Whipple's description of events varied somewhat from what was eventually reported by Dr. Blair, both concurred that the procedure had taken place and that it was a success.

In his textbook, Dr. Blair cautions the surgeon that "the lower jaw should be set back only far enough to be in harmony with the rest of the face" and that the surgeon should "leave it to the orthodontist to bring forward the upper incisors if necessary."[40] He stated that "an orthodontist should be involved in the planning of the patient's treatment from the beginning."[40] For some patients, Dr. Blair notes that "it would be of considerable advantage to have the upper jaw [orthodontically] expanded and the upper incisors and canines brought forward before the lower jaw is surgically set back."[40]

A horizontal osteotomy through the ascending ramus, on each side and above the occlusal plane, as a method of "setting back" the prognathic mandible was first described by Lane.[117] During the early 1900s, Blair[39] and Babcock[19] also made use of the horizontal ramus osteotomy on each side for the correction of prognathism.[36]

In 1912, Harsha reported bilateral wedge resections of the mandible body near the angle regions to set the mandible back; this was similar to the procedure that Blair described in 1897.[89] Unfortunately, at least one of his reported patients suffered the loss of (and the eventual need to remove) the entire anterior part of the jaw. In 1945, Moose[143] may have been the first to describe a fully intraoral technique to carry out horizontal ramus osteotomies for the correction of mandibular prognathism. In 1950, G.V. Barrow and Reed O. Dingman discussed the surgical management of mandibular prognathism.[22] They described a method to determine the exact amount and shape of mandibular bone to be resected on each side by using dental models that involved the construction of splints and templates. Their described meticulous approach was similar to that of Angle in the 1903 edition of his textbook.[7] The operation was reported by Dingman as having a two-stage approach[71,72]; this was to separate the intraoral and extraoral aspects to maintain control of the segments and to limit the risk of infection. During the first stage, the teeth to be extracted (i.e., the first molars) were removed with the use of local anesthesia. As determined by the "preconstructed template," osteotomies for wedge resection were initiated through the intraoral approach at the time of the extractions. Approximately 4 weeks later, the patient was anesthetized in the operating room for the second stage. An incision of the skin of the neck was made 2 cm below the inferior border, and the lower aspect of the mandible was exposed on each side. The previous intraoral vertical cuts on each side were identified. With a power-driven bur or saw, the two vertical cuts were continued through the inferior border. Attempts to preserve the inferior alveolar nerve were made. Stabilization across the osteotomy on each side site was with 24-gauge stainless-steel wires. The teeth were placed into occlusion with the use of either orthodontic appliances or Erich arch bars. In either case, to improve accuracy, the mandibular dentition was secured into a prefabricated acrylic wafer before intermaxillary fixation occurred.

In 1954, J.B. Caldwell and G.S. Letterman devised a vertical osteotomy of the ascending ramus that involved the decortication and perforation of the fragments and then the splitting of the medial and lateral cortices of the ramus to allow for setback of the mandible followed by direct wire fixation.[45,46] This innovative technique had the advantage of providing at least a degree of overlapping of the bone for more reliable and rapid healing.

All of these techniques for the management of mandibular prognathism[79,87,91-93,96,138,167,211] eventually fell by the wayside after the introduction by Hugo Obwegeser of what we now refer to as the "intraoral sagittal splitting of the mandibular ramus," which is described later in this chapter.[149] The stabilization of the osteotomy segments is now generally accomplished with titanium bicortical screws

or with a titanium plate and screws in accordance with the pioneering innovations of H. Luhr, who is also discussed in a later section of this chapter.[127]

Mandibular Advancement for Retrognathism

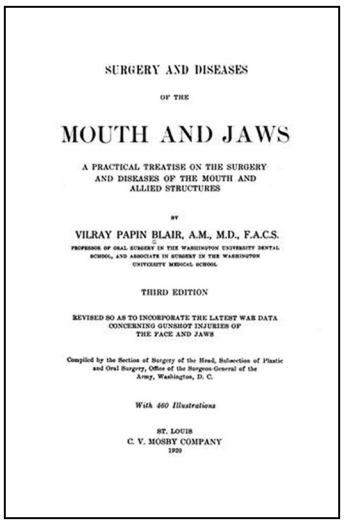
An important early maxillofacial textbook publication was *Surgery and Diseases of the Mouth and Jaws* by Vilray P. Blair (Fig. 2-21).[40] At the time of publication, Dr. Blair was a Major in charge of the subsection of Plastic and Oral Surgery, Section of Surgery of the Head Office of the Surgeon General of the Army in Washington, DC.[197] He was also a professor of oral surgery at the Washington University Dental School and an associate in surgery at the Washington University School of Medicine in St. Louis, Mo. In his textbook, Chapter 21 deals with the treatment of deformities and malrelations of the jaws.

Dr. Blair cautions that, before entering into jaw-straightening surgery, the surgeon would be well advised to do the following:

- To not undertake any cases without first having the fullest confidence in his or her patient
- To remember that procedures undertaken for only "moderate" deformity should not be taken lightly
- To remember that maximum restoration will also require orthodontics
- To remember that the earlier a competent and genial orthodontist is associated with the case, the better it will be for both the surgeon and the patient

In the 21st century, these remain useful words of wisdom for the surgeon to consider before entering into the treatment of a patient with a jaw deformity.

According to Dr. Blair, for the treatment of mandibular micrognathia, a surgical procedure may be used to "bring forward the lower jaw to harmonious outline" with the maxilla.[40] Malocclusion in a patient with a jaw deformity results from the "pressure and counter pressure of growth and apposition."[40] Normal occlusion cannot be established by jaw surgery alone. To reach certain objectives, the surgeon "shall have knowledge of occlusion and of the scope and limitations of orthodontic procedures."[40]


SURGERY AND DISEASES
OF THE
MOUTH AND JAWS
A PRACTICAL TREATISE ON THE SURGERY AND DISEASES OF THE MOUTH AND ALLIED STRUCTURES
BY
VILRAY PAPIN BLAIR, A.M., M.D., F.A.C.S.
PROFESSOR OF ORAL SURGERY IN THE WASHINGTON UNIVERSITY DENTAL SCHOOL, AND ASSOCIATE IN SURGERY IN THE WASHINGTON UNIVERSITY MEDICAL SCHOOL
THIRD EDITION
REVISED SO AS TO INCORPORATE THE LATEST WAR DATA CONCERNING GUNSHOT INJURIES OF THE FACE AND JAWS
Compiled by the Section of Surgery of the Head, Subsection of Plastic and Oral Surgery, Office of the Surgeon-General of the Army, Washington, D. C.
With 460 Illustrations
ST. LOUIS
C. V. MOSBY COMPANY
1920

• **Figure 2-21** Vilray P. Blair published a text entitled *Surgery and Diseases of the Mouth and Jaws,* the first edition of which was published by the C.V. Mosby Company in 1912.

Blair went on to say this: "We have to accept the upper jaw position but the lower jaw is capable of almost any kind of surgical adjustment. The complication of an ununited fracture of the lower jaw is rare. The complication of necrosis or loss of teeth from surgically sectioning the ramus is not reported in the world literature."[40] However, "osteotomy of the ramus of the mandible is a recognized procedure for the treatment of temporomandibular joint ankylosis."[40] Esmarck, who was a prominent surgeon when Dr. Blair was discussing this treatment, recommended the removal of a whole section of the horizontal ramus to avoid a union (i.e., reankylosis) when creating a pseudoarthrosis for the treatment of temporomandibular joint ankylosis. However, Blair felt that surgeons did not need to concern themselves with the possibility of necrosis or nonunion when "completing an osteotomy through the inferior alveolar neurovascular bundle."[40]

Blair felt that the surgeon could correct mandibular retrognathia by completing a ramus osteotomy on each side and then "bringing the distal mandible forward to meet a good occlusion with the maxillary arch."[40] Intermaxillary fixation could then be achieved with stainless steel wires and the use of soft cement in between the occluding teeth.[40] Fixation across the osteotomy site was felt to be unnecessary. The (horizontal) ramus osteotomy was made at or just above the mandibular occlusal plane with the use of a Gigli saw placed transcutaneously.[39] The osteotomy was cut through the inferior alveolar neurovascular bundle, which was not felt to be important either to the circulation of the mandible or as a cause of disability. The ramus osteotomies were later made blindly and transorally with a Gigli saw.[40]

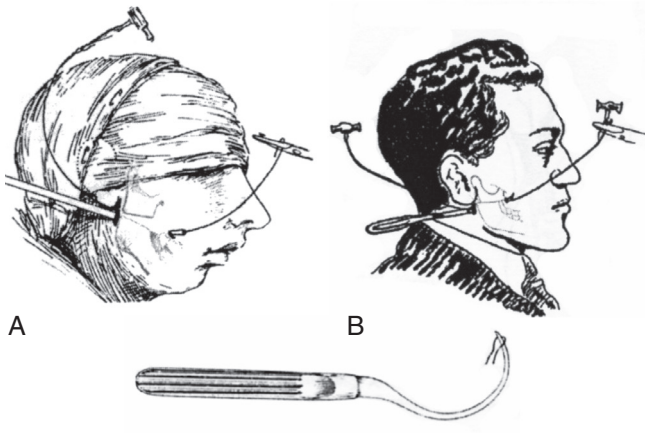
According to Dr. Blair, "the operation presented three distinct problems: (1) the cutting of the bone [which is the easiest of the three]; (2) the placing of the jaw into its new position; and (3) holding it there [for the time required for healing]."[40] He used postoperative x-rays to show that the fragments of the ramus remained in contact at the posterior border of the osteotomy. "[A]s the distal mandible is brought forward," said Dr. Blair, "a gap is created anteriorly while the fragments remain in contact at the posterior border. For healing to occur, the bone gap must be filled with granulations. The resulting bone scar tends to contract [relapse] for months afterwards."[40] The maintenance of tight intermaxillary fixation was felt to be essential to maintain the new position. Blair used iron wire for its strength, lack of stretch, and pliability; it could also be twisted, unless it was damaged by sharp instruments.[40] He also used quick-setting cement between the occlusal surfaces of the molars to prevent the placement of postoperative forces on the anterior teeth, which could cause the patient significant pain.[40]

Dr. Blair also used this same operation involving horizontal ramus osteotomies for the treatment of ankylosis associated with severe mandibular retrusion; this operation was first carried out by Dr. Mudd with Dr. Angle assisting in 1897. Dr. Blair also suggests addressing the residual chin deformity by either "injecting paraffin into the chin or by inserting a piece of cartilage or rib."[40]

Most of the early reported attempts at horizontally advancing the retrognathic mandible focused on the mandibular body. Dr. Blair specifically addressed the problem of an anterior open bite by rotating the distal mandible counterclockwise after body osteotomies and ostectomies. He states that the "sectioning of the mandible on both sides in front of the first molars [as long as the molars are in occlusion] is the preferred procedure. The anterior mandible can then be moved into good occlusion with the upper teeth. In other cases, it is necessary to remove a V-shaped section of the bone on each side with extraction of the tooth [bicuspid]. The apex of the V-shaped ostectomy segment is at the lower border of the mandible and usually a tooth is extracted from the site of the section on each side. The bone is cut using a Gigli saw or a cross-cut Fisher bur."[40]

Dr. Blair goes on to state that "the deformity is in both jaws but the operation is limited to the lower jaw. It is best to restore the lower jaw to its proper form and then manage the residual open bite by bringing down the upper incisors with orthodontic appliances or by extending the upper teeth with porcelain crowns. Fixation is achieved with a prefabricated splint constructed on plaster dental models. This will prevent pulling down of the distal segment by the digastric and geniohyoid muscles."[40]

A variety of techniques were carried out by pioneering surgeons to address concerns regarding the maintenance of contact between the fragments to achieve bony union; soft-tissue coverage to limit infection; opposing muscle pull to limit relapse; and adequate fixation. In 1928, a "step osteotomy" of the body of the mandible was proposed by Von Eiselberg.[201] Also in 1928, Gadd reported a stepped procedure for the body of the mandible for the purpose of correcting asymmetric mandibular deficiency.[80] That same year, Limberg completed L-shaped sliding osteotomies of the mandibular body to improve bone-to-bone contact after advancement.[122,123] In 1931, Kostecka published his work with the Blair-type transcutaneous horizontal ramus osteotomies with the use of a Gigli saw (Fig. 2-22).[115] In 1936, Kazanjian performed mandibular "step" osteotomies anterior to the mental foramen with extension back into the body of the mandible to allow for advancement and fragment approximation (Fig. 2-23, *A* through *D*).[68,103-108] The constant problems encountered with all of these approaches involved maintaining sufficient bone-to-bone contact and achieving adequate fixation for a stable long-term result. The problems were finally solved through the pioneering work of Hugo Obwegeser (i.e., the intra-oral sagittal splitting of the ramus of the mandible) and Hans Luhr (i.e., plate and screw fixation across the osteotomy site), which is described later in this chapter. In 1960, Caldwell described vertical osteotomies of the ramus with interpositional iliac bone graft for the correction of micrognathia.[45] A C-osteotomy of the ramus was later described by Caldwell and colleagues, in 1968.[46] Although these osteotomies with interpositional grafting are occasionally used for the management of mandibular retrognathism, the Obwegeser sagittal split ramus osteotomy is generally preferred. Throughout the 18th and 19th centuries, many surgeons saw the need to mobilize the maxilla for better access either to remove an epipharyngeal tumor or for the correction of maxillary deformity.

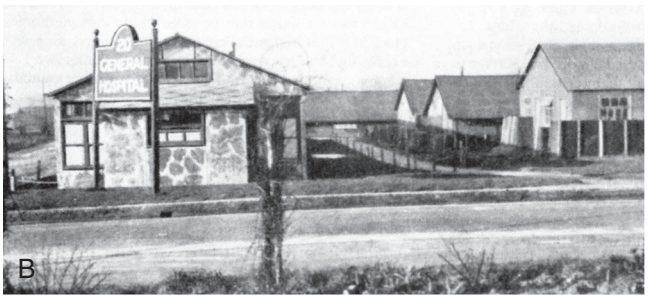


• **Figure 2-22** As originally depicted, methods for the correction of mandibular deformities by the blind technique. **A,** The method of Blair. *From Blair VP:* Surgery and diseases of the mouth and jaws, *ed 3, St. Louis, 1914, C.V. Mosby Company.* **B,** The method of Kostecka. *From Kostecka F: Die chirurgische therapie der proggeni.* Zahnaertzliche Rundschau *40:669–687, 1931.*

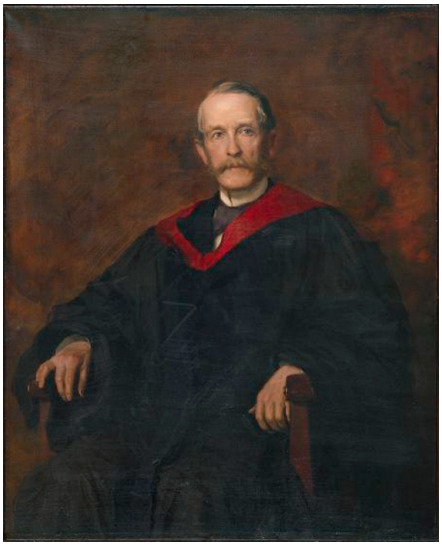
Maxillary Osteotomies for Management of Dentofacial Deformity

In 1868, David Williams Cheever of Boston published a report of a Le Fort I osteotomy for the purpose of exposing and removing a large nasopharyngeal polyp (Fig. 2-24).[55,88] After completing the tumor resection, the maxilla was placed back in its original location. Cheever stated, "so far as I know, the operation including both superior maxillary bones—is novel."[55] "Nothing but the posterior attachments of the upper maxilla now prevented their depression and hinging on the pterygoid processes. The upper jaw was brought down so as to expose the tumor."[55] By the early 1900s, reports of series of patients undergoing maxillary osteotomies for the purpose of tumor resection were detailed in the literature.[37]

With reference to the treatment of dentofacial deformities, in 1921 in Berlin, Cohn-Stock was the first to report an elective maxillary osteotomy to establish a preferred occlusion.[56,214] He used a two-stage approach (a precaution against flap necrosis) when completing an anterior maxillary osteotomy. Martin Wassmund, the founder of the "German school" of maxillofacial surgery, is credited with developing the one-stage anterior maxillary osteotomy.[205-207] The Wassmund procedure was completed through limited palatal and labial incisions with subperiosteal tunneling. The tunneling



• **Figure 2-23** **A,** *Dr. Varaztad H. Kazanjian leaving Buckingham Palace after his investiture as a Companion of St. Michael and St. George for his services during World War I, March 15, 1919,* **B,** *View of the hospital showing Dr. Kazanjian's three wards, circa 1916,* **C,** Photograph of the hospital ward within the makeshift (Harvard unit) barrack where trauma patients were under the care of Varaztad H. Kazanjian (1915-1919). **D,** Photograph of Varaztad H. Kazanjian later in his career. *A and B, From The Harvard Medical Library in the Francis A. Countway Library of Medicine. C, From Deranian HM: The miracle man of the western front. Bull Hist Dent 32:85-95, 1984.*

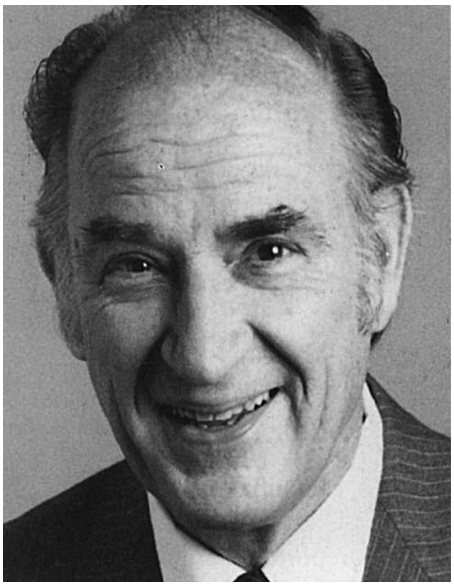


• **Figure 2-24** Photograph of David Williams Cheever of Boston (1831-1915). *Courtesy Harvard University Portrait Collection.*

incisions limited mobilization, but this was thought to be necessary to maintain circulation. Karl Schuchardt is credited with developing the posterior maxillary segmental osteotomy.[171-173] This was completed with the use of broadly elevated full-thickness palatal flaps and limited labial flaps. In 1959, Köle described the combined simultaneous maxillary and mandibular anterior segmental (alveolar) osteotomies for the correction of bimaxillary protrusion and other forms of open bite, deep bite, and posterior crossbite.[111-114] He also pioneered the "corticotomy of maxillary and mandibular bones to facilitate the orthodontic movement of either a single tooth or group of teeth."[114]

Wassmund is credited with performing a first "total maxillary osteotomy" for the correction of a dentofacial deformity in 1927.[205-207] This was in a patient with an open-bite malocclusion. The maxillary osteotomy was completed through tunneling incisions without full down-fracture. Intermaxillary elastics were applied, with the gradual movement of the maxilla into occlusion occurring over the course of 2 weeks. In 1934, Axhausen was the first to mobilize and

• **Figure 2-25** Photograph of Hugo L. Obwegeser (1920-present).

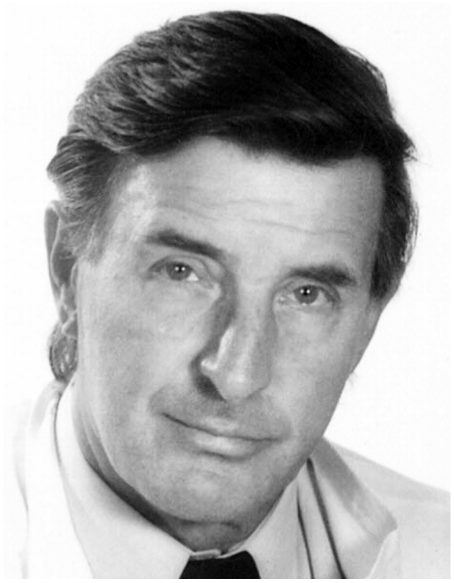


• **Figure 2-26** Photograph of William H. Bell (1927-present).

immediately reposition a malunited maxillary fracture by Le Fort I osteotomy.[15-17] Pioneering surgeons described and continued to attempt maxilla osteotomies (anterior segmental, posterior segmental, and total maxillary) to improve the occlusion in individuals with dentofacial deformities.[44,50,57-59,61,116,119-121,126,141,158,160,169,180,183,212,213,219,220] Unfortunately, circulation requirements of the maxillary segments were poorly understood, mobilization was often incomplete, and available fixation options were limited.[66,86,87,110] These problems severely hampered the results that were possible for the patient with a jaw deformity.[142,217]

It was not until Hugo H. Obwegeser described the total horizontal Le Fort I osteotomy with complete down-fracture through a circumvestibular incision followed by full mobilization that the maxillary deformity could be universally addressed in all three planes, as described later in this chapter (Fig. 2-25).[153] Animal research carried out in William H. Bell's laboratory soon confirmed the vascular integrity of each specific dentoalveolar–musculomucosal mandibular and maxillary osteotomy-created flap that had been clinically described and used by Obwegeser and other European maxillofacial pioneers (Fig. 2-26).[29] Experimental confirmation of the adequacy of the circulation to each of these flaps, when properly elevated, encouraged the rapid dissemination of knowledge and the incorporation of orthognathic procedures into maxillofacial clinical practice throughout the world; this is discussed in more detail later in this chapter.

Dr. Hans Luhr's innovative ideas with regard to the development and clinical use of plate and screw fixation in maxillofacial surgery was the final piece of the puzzle, as described later in this chapter (Fig. 2-27).[127-134] Interestingly, the third edition of Dr. Blair's text, which was published in 1912, illustrates the use of what he calls "silver splints" for fixation across a mandibular osteotomy site (Fig. 2-28).[40] He stated that the use of these fixation devices was essential for healing. The "silver splints" look much like the titanium mini-plates with screws that we use today. He states that the "silver splints" were used on a patient for the correction of mandibular prognathism.

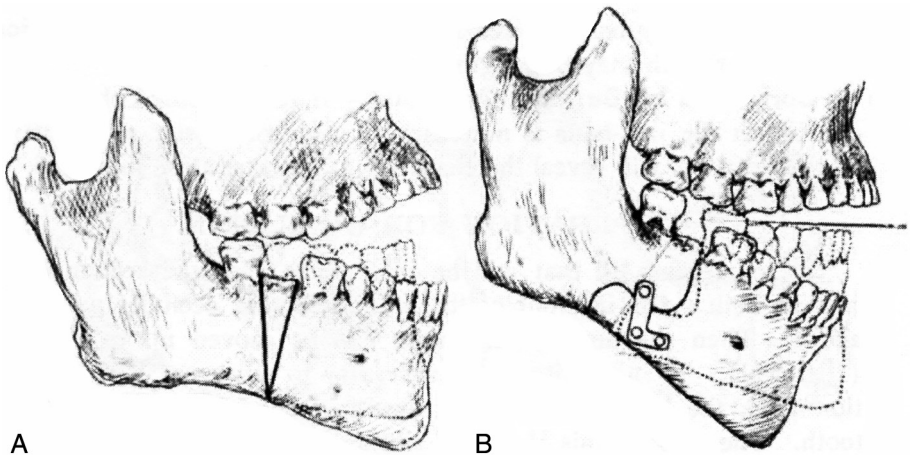


• **Figure 2-27** Photograph of Hans Luhr (1932-present).

Hugo L. Obwegeser: Development of the Standard Orthognathic Procedures*

After completing his medical studies in mid 1945, Hugo Obwegeser started his career as an unpaid visiting doctor in a small hospital near his hometown. By doing so, he had

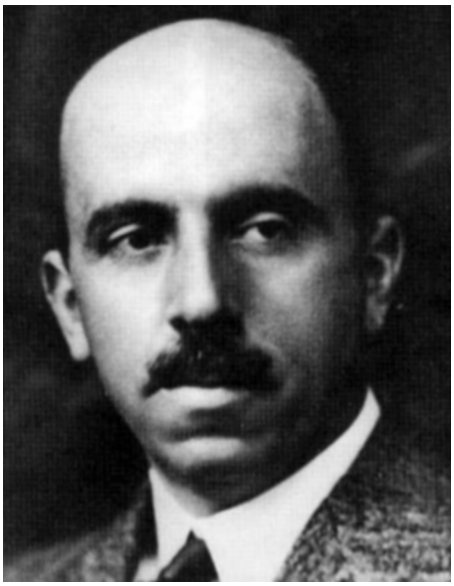
*Please note that this section was reviewed for accuracy by Dr. Obwegeser.



• **Figure 2-28 A,** Open bite of an extreme type in a young man showing how correction may be made by with V-shaped excision. This would have the disadvantage of shortening the jaw considerably, as shown in the reconstructed jaw, which is indicated by the dotted lines. **B,** A demonstration of how an operation involving an S-shaped bone cut that lengthens the jawbone provides for the better closure of the mouth. The silver splints at the site of the bone-cutter are absolutely necessary. *From Blair V:* Surgery and diseases of the mouth and jaws, *ed 3, St. Louis, 1917, C.V. Mosby Company, pp 309, Figures 287 and 288.*

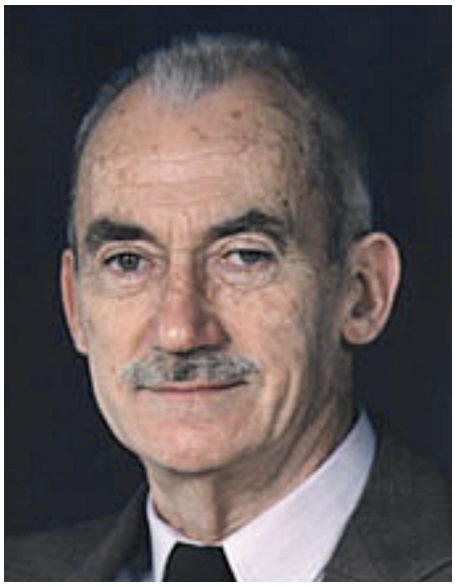
the opportunity to assist with surgery, including the treatment of extremity fractures. He did not know at that point in what direction his medical training would lead him. His uncle, an orthopedic surgeon, gave him this advice: "Whatever you want to become, a general medical practitioner in a small town or a specialist in any field of medicine, you will always need a fundamental knowledge of pathology and pathophysiology."[156] Obwegeser listened to his uncle, and, in October 1945, he accepted an unpaid position training under Professor Chiari at the Rockitansky Institute of Pathological Anatomy at the University of Vienna. At first, Obwegeser watched and listened, and he was occasionally allowed to assist at the dissecting tables. After 1 year of general surgery training and 2 years of pathology training, he was offered a paid position to train in maxillofacial surgery under Richard Trauner at Graz University. At Graz, Obwegeser would also train in dentistry and sit for the Austrian dental qualification examination (dentistry in Austria was a subspecialty of general medicine). Obwegeser trained and worked with R. Trauner for 6 years. He saw professor Trauner both as a teacher and like a father.[156] According to Obwegeser, Trauner "opened our eyes regarding the scope of the specialty [maxillofacial surgery] and stimulated us to see problems and find solutions for them."[156] The maxillofacial clinical practice at the Graz University Hospital included primary clefts, head and neck cancer, facial trauma, and the management of severe residual facial war injuries.

During that time and with a scholarship from the British Council, Trauner sent Obwegeser to Harold Gillies, the founder of modern plastic and reconstructive surgery (Fig. 2-29).[81-85] Obwegeser observed and worked with Gillies from October 1951 to February 1952, primarily on the reconstruction of war cases. From Dr. Gillies, Obwegeser



• **Figure 2-29** Photograph of Harold Gillies (1882-1960).

learned "how to plan the reconstruction of a facial defect and how to handle soft tissues."[156] Trauner also sent Obwegeser to spend time with Edward Schmid at Marien-Hospital in Stuttgart, where Obwegeser further advanced his knowledge of the handling of soft tissues, the movement of flaps to a new location, the management of cleft defects, and the closure of fistulas.[156] During his career, Obwegeser was also influenced by Dr. Paul Louis Tessier (Fig. 2-30).[184-196] Obwegeser states that, from Paul Tessier, he learned "the principal technique for advancing the whole middle third of the face and how to rotate the eye sockets into the planned position."[156] Tessier used these new techniques to



• **Figure 2-30** Photograph of Paul Louis Tessier (1917-2008).

correct posttraumatic or congenital anomalies of the upper half of the face. Tessier's "cranial approach to the orbits via the anterior base of the skull has created the possibility of producing a normal appearance and with that a normal life for many patients"[156]; this is described later in this chapter.

In June 1966, Dr. Obwegeser broadly introduced his orthognathic surgery techniques to North American surgeons when he presented a series of invited lectures and demonstrations at a comprehensive conference sponsored by the American Society of Oral Surgery at Walter Reed Military Hospital in Washington, DC.[156] At that time, Obwegeser also described other new ideas with broad implications for the scope of oral and maxillofacial surgery, including preprosthetic procedures, transoral approaches to the reduction of mandibular fractures, and the simultaneous resection and reconstruction of tumors of the mandible with the use of a transoral approach.[150-155]

According to Obwegeser, "With fundamental basic training in medicine and dentistry and through my teachers' influence, I was able to produce new ideas and procedures, assemble them in contrast to the then existing rules. The influence of my teachers enabled me to become a teacher myself. It enabled me from 1958 until 1987 to establish the so-called Zurich-School of Maxillofacial Surgery."[156]

The Transoral Sagittal Splitting of the Mandibular Ramus

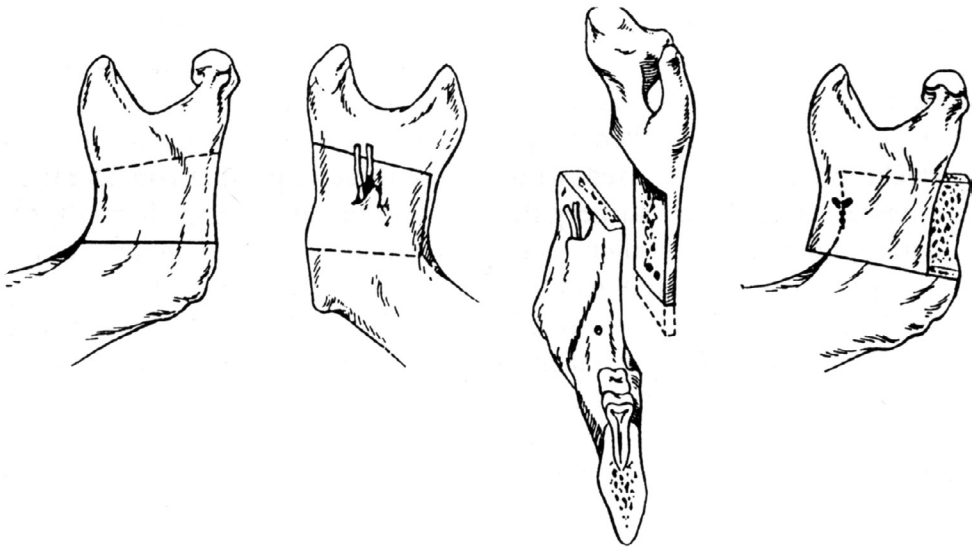
Trauner R, Obwegeser H: Zur operationstechnik bei der progenia und anderen unterkieferanomalien. **Dtsch Zahn Mund Kieferhlkd *23:11–25, 1955***

In 1947, Obwegeser started his training in maxillofacial surgery under the direction of Richard Trauner from the maxillofacial unit of the dental school at the University of Graz in Austria. At that time, orthognathic surgery was considered to be a series of unsatisfying procedures carried out primarily for the correction of prognathism. The available mandibular procedures were limited variations of horizontal ramus osteotomies. The basic techniques were those described by Blair[38-41] and Kostecka[115]:

> "The Blair-Kostecka technique included: The ascending ramus was cut on each side of the face [see Fig. 2-22]. The patient was placed in IMF [intermaxillary fixation] (6 to 8 weeks). It was estimated that approximately 50% of patients experienced significant complications including: partial or total relapse; residual malocclusion (open bite); pseudoarthrosis; injury to the facial nerve; injury to the inferior alveolar nerve; or parotid gland fistula. It was assumed that a primary reason for nonunion/malunion was the limited area of bone to bone contact across the osteotomy site once the distal mandible was placed in occlusion. This was compounded by contraction of the temporalis muscle of the non-fixed proximal segment causing further dislocation and separation. This complication was especially common when the procedure was used to correct mandibular deficiency as the distal mandible had to be advanced to achieve IMF [intermaxillary fixation]."[156]

In 1952, Dr. Obwegeser retrospectively reviewed a consecutive series (n = 36) of Dr. Trauner's mandibular osteotomies that had been treated with the Blair-Kostecka approach.[76,198-200] Obwegeser found a 50% incidence of significant complications. At his teacher's request to look for a better way to solve the mandibular deformity problem, Dr. Obwegeser made a personal decision about two aspects. First, he felt that the extraoral (cutaneous) approach left an unpredictable quality of scar and added a risk of injury to the inferior alveolar (sensory) nerve and the facial (motor) nerve, so he chose to find an intraoral approach. Second, he believed that the available techniques produced inadequate bone-to-bone contact to ensure stable bony healing. A new approach would have to provide broad contacting bone surfaces, even after anterior mandibular repositioning from micrognathia.

Dr. Obwegeser studied the cadaver mandible and concluded that "the ramus could be split along its sagittal plane—with a broad bony surface area for healing."[156] He had noticed a sagittal pattern on the radiographs of mandibular fractures that he had seen, and he wondered if such a fracture could be purposely created by a surgeon. Obwegeser was certain that "the inner and outer cortex could be cut at different levels and [then] the two corticotomies [could be] reconnected along the sagittal plane."[156] He was also convinced that the intraoral approach would not in itself result in infection. He had treated mandibular fractures via an intraoral approach by repositioning the fragments and providing intermaxillary fixation within 24 hours of the injury, and he had found that this resulted in minimal complications.[156]



• **Figure 2-31** Illustration demonstrating the first sagittal splitting of the rami. *From Trauner R, Obwegeser H: Zur operationstechnik bei der progenia und anderen unterkieferanomalien.* Dtsch Zahn Mund Kieferhlkd *23:11–25, 1955.*

Obwegeser then set out to sagittally split the ramus transorally on a cadaver. He did his cadaver work at the Institute of Anatomy in Graz, Austria (Fig. 2-31). Professor Trauner, who was Obwegeser's chief, then agreed to let Obwegeser try the new technique (i.e., intraoral approach sagittal split of the mandibular ramus) on a patient. Dr. Trauner would carry out his preferred approach (reverse L-shaped osteotomy of the ramus through a combined extraoral and intraoral approach) on one side, and Obwegeser would be allowed to try his technique on the other side. The procedure was carried out on an edentulous 27-year-old woman who presented with mandibular prognathism. A preoperatively constructed acrylic splint was made for the final occlusion, and the jaws would be wired together with the use of intermaxillary fixation (IMF). The procedure was carried out with the patient in a half-sitting position under local anesthesia on February 17, 1953. Unfortunately, when Obwegeser tried to execute his technique, the ramus "unexpectedly shattered instead of splitting."[156] The patient's mandible was set back, the occlusion was secured with the acrylic splint, and IMF was applied. Healing was satisfactory.

Two months later, a 24-year-old woman with mandibular prognathism and a nearly full dentition presented. Dr. Trauner again agreed to allow Dr. Obwegeser to try his technique. On April 22, 1953, with the patient in a half-sitting position and under local anesthesia, Dr. Obwegeser completed his osteotomy using long Lindemann burs, a keyhole saw and a fissure bur to perforate the cortex, and then a rose bur to connect the cortical perforations. An osteotome was finally used to split the ramus. The coronoid process fractured off, but, "with a gentile twisting maneuver, the cortical plates fell apart and the ramus was sagittally split."[198] On the other side, Dr. Trauner performed his inverted L-shaped osteotomy through a combined oral and extraoral approach. "This was followed by six weeks of IMF. The recovery was uneventful." The published result shows a favorable occlusion and good facial contour[198] (Fig. 2-32).

Obwegeser recognized that additional problems to be overcome included the need for special instrumentation, intraoral lighting, and improved anesthesia. He joined the Department of Maxillofacial Surgery at the University Hospital in 1954. In 1956, Dr. Hotz, an orthodontist, asked Dr. Obwegeser to perform the "sagittal splitting procedure" on one of his patients.[156] This patient was a 14½-year-old girl with a prognathic mandible who was partially maxillary edentulous.[156] On April 9, 1956, with the patient under nasotracheal intubation, Dr. Obwegeser successfully completed the sagittal splitting of the ramus of the mandible on each side. On one side, the lateral ramus broke off, but it was secured back in place at the end of the procedure with direct wires. The operation took 4 hours, and healing was uneventful. According to the surgeons, "She had a wonderful aesthetic and functional result—no external scars"[150] By the early 1960s, the sagittal splitting technique of the ramus of the mandible became routine for Dr. Obwegeser, and word soon spread worldwide.

Obwegeser mentions two important modifications to the sagittal splitting of the ramus of the mandible that later came about.[151-153] The first modification was in "the placement of the lateral corticotomy to increase the surface area of the bony contact for improved union and to accommodate a wider range of advancement of the distal mandible."[156] The change was from a horizontal orientation of the lateral corticotomy of the ramus to a vertical orientation of the lateral corticotomy of the mandibular body. In 1957, while an observer of Dr. Obwegeser in Zurich, Dr. Dal Pont "conceived the idea of changing the lateral corticotomy from horizontal in the ramus to vertical in the body."[156] Dal Pont assisted Obwegeser with this goal, and it worked. In

• **Figure 2-32** The patient shown underwent the first successful transoral sagittal split of the ramus on the left side. This operation was completed by Obwegeser. A transcutaneous inverted L osteotomy on the right side was completed by Trauner on April 22, 1953. **A,** Left profile views before surgery as well as 5 months and 22 years after the surgery. **B,** The patient's occlusion before and after surgery is also shown. **C,** The posteroanterior cephalometric radiograph shows the wire fixation on the inverted L side (the right side) and without wire fixation on the sagittal split side (the left side). *From Obwegeser HL: Orthognathic surgery and a tale of how three procedures came to be: a letter to the next generations of surgeons.* Clin Plastic Surg *34:335, 2007, Figure 4.*

1958, Dal Pont went back to his home base in Italy and eventually published these results himself.[62,63] However, Dal Pont failed to mention that the patient described was Dr. Obwegeser's patient. A second modification was also suggested by Dal Pont when he observed that, in some patients, the split did not go back all the way to the posterior border when the ramus was sectioned; satisfactory results nevertheless occurred.[64] This second modification was also recommended by Hunsuck and Epker when they "advocated that the osteotomy of the lingual side should be incomplete, extending just past the entrance of the neurovascular bundle."[156] This would result in an incomplete fracture along the medial aspect of the ramus.[65,67,77,78,99,136]

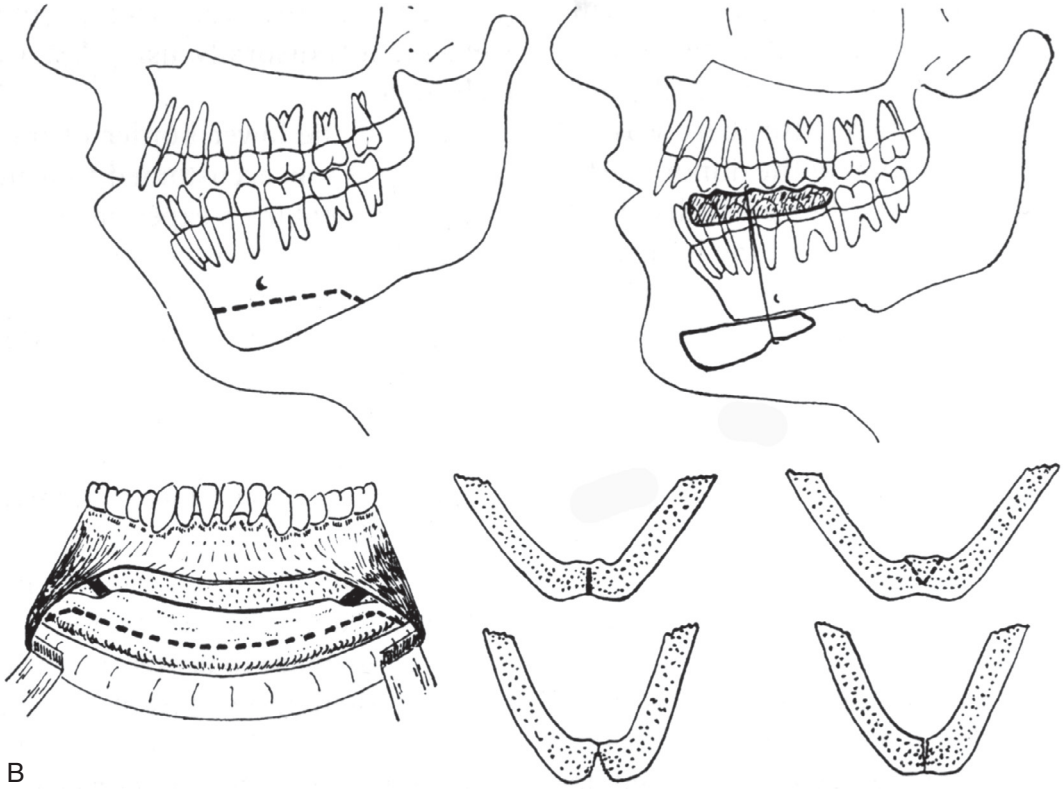
The Transoral Sliding Osseous Genioplasty

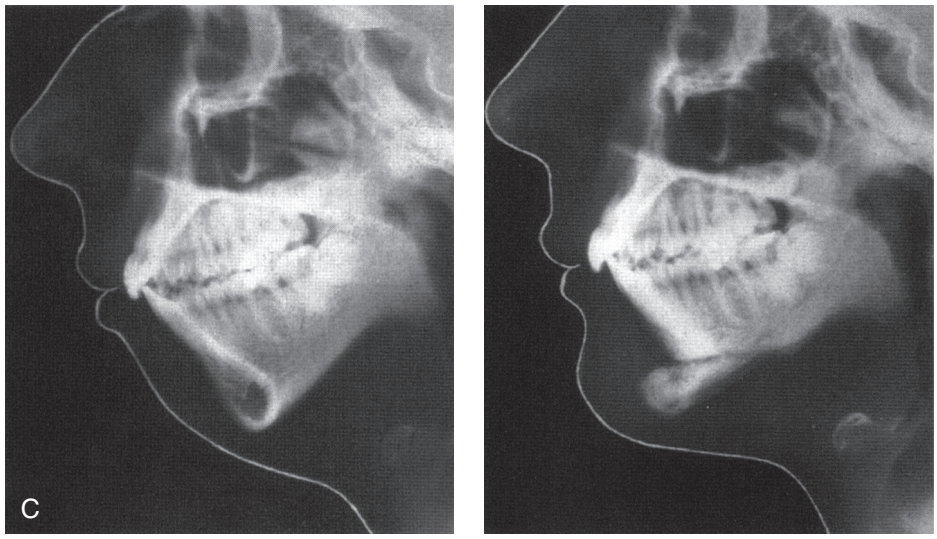
Trauner R, Obwegeser H: The surgical correction of mandibular prognathism and retrognathia with consideration of genioplasty. I. Surgical procedures to correct mandibular prognathism and reshaping of the chin. **Oral Surg Oral Med Oral Pathol *10:677–689, 1957***

Through the late 1950s, the correction of microgenia was achieved through onlay techniques to augment the deficient chin.[13,14,102,140] The procedures were carried out through a submental (skin) incision. Autogenous bone and cartilage grafts (e.g., rib, hip) and a variety of alloplastic materials (e.g., acrylic, Silastic, metal) were used to augment the deficient chin. The onlay bone grafts resorbed over time. The cartilage grafts healed in a capsule and were often mobile and displaced, thereby causing asymmetry. The alloplastic materials, although easy to fabricate and place, had a high complication rate that included infection, displacement, the erosion of underlying bone, the resorption of the dental roots, and an unfavorable appearance.

Early in his career, Dr. Obwegeser had the intention of finding a reconstructive technique that would correct microgenia through an intraoral approach, that would allow for sufficient bone contact for reliable union, and that would maintain the advancement without resorption or relapse. After evaluating a woman with a retrusive chin and normal occlusion and reviewing her lateral cephalometric radiograph, Obwegeser realized that a favorable chin contour could best be accomplished by sectioning the chin transversely (i.e., below the roots of the teeth and the mental foramen) and then advancing it. He used the same transoral incision that he was familiar with from mandibular fracture management. He used a Lindemann bur to make a horizontal osteotomy below the roots of the teeth and the mental foramen, and he then completed the osteotomy with a chisel. He repositioned the distal chin horizontally forward by 10 mm while maintaining the geniohyoid muscle pedicle. He fixed the chin in its new position with circummandibular threads around the chin and bicuspid regions. The threads were primarily secured over a prefabricated acrylic wafer that was placed on the occlusal surfaces of the mandibular teeth to prevent "cheese wiring" through the interproximal regions. Obwegeser published this successful chin osteotomy outcome in 1957 (Fig. 2-33).[150] Interestingly, an earlier publication by Hofer reported the sliding the inferior border of the chin forward.[94,95] Although this osteotomy was similar to the one reported by Obwegeser, it was completed through an extraoral incision, and it was performed on a cadaver rather than a living patient. Over the decades, few useful clinical modifications of the original chin osteotomy (i.e., the Obwegeser technique) have been found in the literature. They include a double-step advancement and the splitting of the osteotomy in the midline to control width and angulation.[150]

• **Figure 2-33** The first transoral approach of an osseous genioplasty to correct a chin deformity. **A,** Facial profile views before and after surgery. *Continued*





• **Figure 2-33, cont'd** **B,** The transoral osseous genioplasty as illustrated in a 1957 article by Obwegeser. **C,** Lateral cephalograms before and after surgery. *From Obwegeser HL: Surgical procedures to correct mandibular prognathism and reshaping of the chin. In Trauner R, Obwegeser H, editors: The surgical correction of the mandibular prognathism and retrognathia with consideration of genioplasty.* Oral Surg Oral Med Oral Pathol *10:677–689, 1957.*

The Le Fort I (Type) Osteotomy

Obwegeser H: Der offene biss in chirurgischer sicht. **Schweiz Monatschr Zahnhlkd *74;668–687, 1964***

Early on during his career, Dr. Obwegeser recognized the necessity of developing a technique that would allow for the reliable repositioning of the maxilla. He realized that, for many jaw deformities, the occlusion could be restored with a mandibular setback, but a flat appearance to the midface and the stigma of the underlying condition would remain. Obwegeser explained that, by the 1950s, he realized solving this problem would require 1) safe surgical sectioning of the maxilla; 2) the ability to three-dimensionally reposition the maxilla in the operating room; 3) the ability to maintain

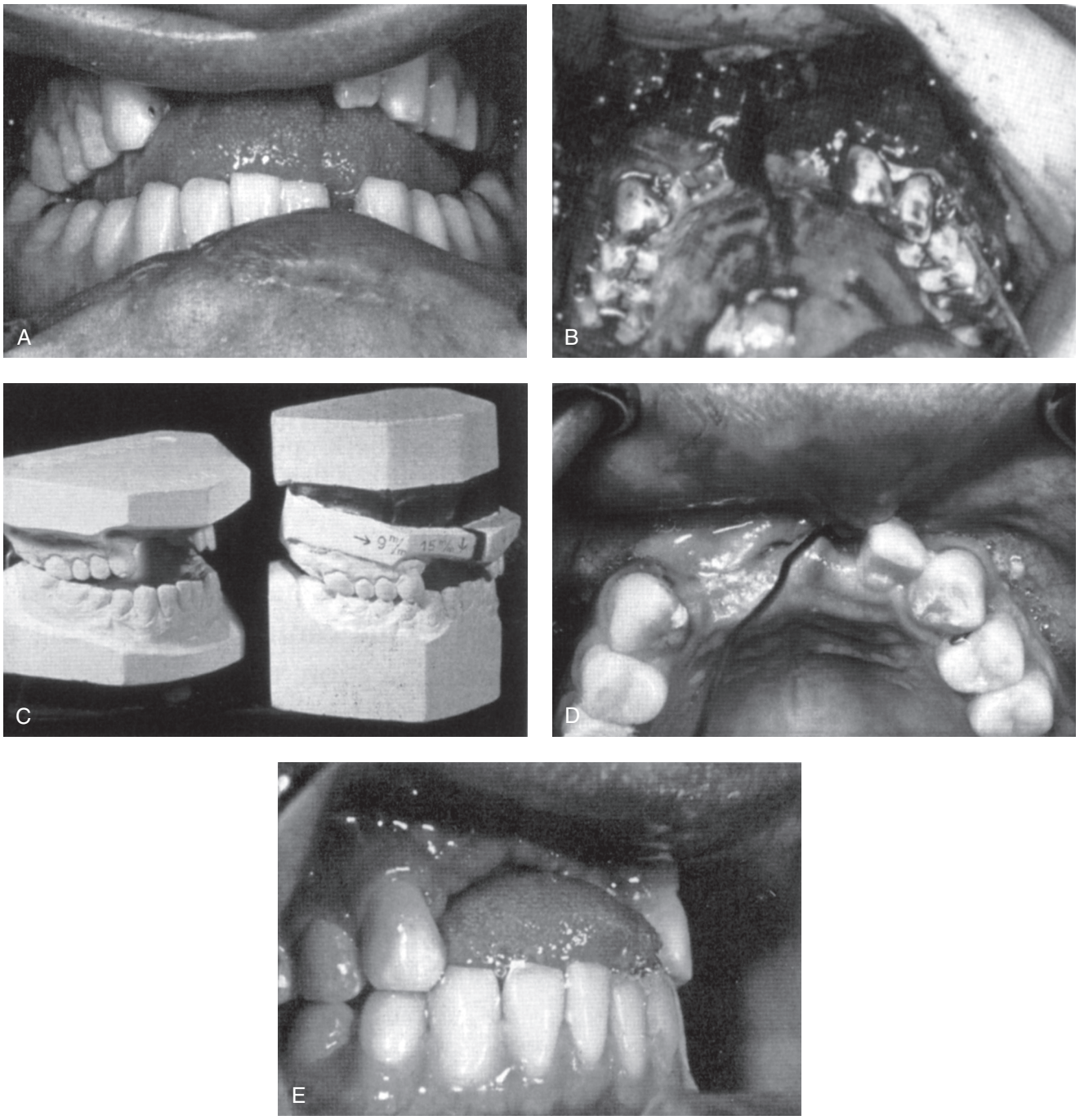
the upper jaw in the new position during healing, with 4) an acceptable complication rate and 5) a predictable outcome.

Obwegeser knew that Langenbeck[118] and Cheever[55] were able to section the maxilla horizontally above the roots of the teeth through skin incisions of the cheek and lip for the purpose of gaining access to the posterior pharynx and the skull base. Wassmund reported that, in 1927, he had "detached the maxilla as a Guerin-type fracture" (personal communication) but without separating it from the pterygoid processes.[205-207] He then used elastics (rather than a complete mobilization of the maxilla) to close the anterior open-bite malocclusion. Axhausen successfully completed osteotomies and repositioned the upper jaw in posttraumatic and cleft patients.[15-17] He used elastics to relocate the maxilla after an osteotomy of the upper jaw, which included separation from the pterygoid processes but without complete intraoperative mobilization. In 1942, Schuchardt reported about a posttraumatic midface deformity caused by a war injury in which he completed a horizontal osteotomy of the maxilla above the roots of the teeth as a first procedure.[171-173] Several weeks later, during a second operation, he fractured the pterygoid processes. After the second procedure, he used weight traction to horizontally advance the maxilla. He stated that "this procedure would have a large indication in cleft cases, but it would probably never come into use."[171-173] In 1951 and 1952, Obwegeser observed Sir Harold Gillies completing maxillary osteotomies to correct cleft maxillary deformities in numerous patients.[156] Dr. Gillies "used horizontal vestibular incisions to approach the maxilla," "but he only rotated the collapsed cleft segments with a Green-stick fracture at the pterygoid-maxillary junction."[156] This is similar to what Schmid reported in 1956 for the correction of posterior crossbites.[170] According to Obwegeser, neither Gillies nor Schmid completed the horizontal maxillary osteotomies with the purpose of advancing the maxilla.[156] Gillies and Rowe[84] and Gillies[85] did report that, in 1947, they had completed an osteotomy of the maxilla and were able to bring the upper jaw into the desired position in a patient with a cleft palate, with Gillies reopening the alveolar/palatal cleft and dividing the maxilla in two. This was followed by immobilization that lasted for 12 weeks. Gillies would secure the maxilla in its new location with the use of cast cap splints over the teeth that then attached to a vertical bar in front of the face and to a head cap. Gillies placed cancellous bone grafts on the steps (gaps) in the canine fossa regions and directly into the maxillary sinuses. Because of Gillies' reticence to advance the maxilla, the horizontal discrepancy in the jaws had to be overcome by setting the mandible back. Unfortunately, the patients retained their "dish face" with, at best, a "flat appearance."[156]

Throughout the 1950s, Obwegeser looked for solutions to correct the midface deformity seen in cleft and posttraumatic patients.[137,159] He believed that vertical vestibular incisions—which were felt by most surgeons of the day to be necessary to maintain the maxillary blood supply—limited successful outcomes.[156] He was convinced that the limited vertical vestibular mucosal incisions 1) prevented the full mobilization of the maxilla; 2) prevented the relaxed repositioning of the upper jaw in the operating room; and 3) prevented the placement of fixation and bone grafts, 4) thus further limiting the stability of the advancement and resulting in eventual relapse.

Obwegeser was able to innovate by making a quantum leap in the reconstructive possibilities of maxillary osteotomies for the correction of jaw deformities. In 1964, he was asked to treat an 18-year-old man who had been in a car accident 6 weeks earlier. The young man had sustained a fracture of the maxilla in two segments (palatal split), with telescoping occurring into the nose and the maxillary sinus. This resulted in a retropositioned maxilla with a severe anterior open bite. Three maxillary incisors were lost, there were multiple oronasal fistulas, and the vestibular mucosa had been lacerated circumferentially. There was initial healing of the soft tissues present, with scarring and malunion of the bones. To repair the deformity, Obwegeser realized that limited vertical vestibular incisions, which were part of his usual approach, were not feasible. He instead elected to reopen the circumferential vestibular incisions, also through the oronasal fistula opening (Fig. 2-34). He then completed a Le Fort I osteotomy through the two halves of the maxilla that had healed in malunion. This required horizontal osteotomies above the teeth from the canine fossa past the tuberosities and through the sinus on each side. He used osteotomies and a fissure-type bur on a rotary drill to complete the osteotomies. He raised the nasal mucosa, detached the septum, cut through the lateral nasal walls, and separated the pterygoid plates vertically. With each step, he confirmed that the vascularity to the dentoosseous–musculomucosal flap was not jeopardized. With finger pressure, the maxilla was disimpacted via the down-fracture technique. Obwegeser fully mobilized the maxilla to move it forward. He recreated the palatal fracture (segmentation) to independently reposition the two halves of the maxilla. He realized that the descending palatine arteries were no longer intact; while working through a circumvestibular incision, he was able to split the maxilla into two segments and to maintain the circulation to each half. He simultaneously closed the oronasal fistulas, fixed the maxillary segments in place, and established the preferred occlusion. By doing so, it was not necessary to complete mandibular osteotomies to correct the occlusion.

Over the next several years, Obwegeser succeeded in advancing the maxilla in cleft patients "without the need to establish the occlusion with a mandibular setback as in years past."[156] On April 14, 1968, he treated a patient with unilateral cleft lip and palate and a severe maxillary deficiency. Obwegeser advanced the lesser segment by 15 mm and the greater segment by 13 mm. The Le Fort I osteotomy was completed, and this was followed by down-fracture and mobilization. He recognized that "mobilization—release of scar tissue—and having both segments absolutely loose—was the entire key to success." He realized that he could



• **Figure 2-34** The first case of the advancement of the maxilla in two segments through a circumvestibular approach was performed by Obwegeser in 1964 to correct a posttraumatic deformity. **A,** A severe anterior open bite caused by posttraumatic telescoping retromaxillism in two segments. **B,** Oral-nasal communication and vestibular scarring on both sides. **C,** Model surgery indicating 9 mm of advancement and 15 mm of movement vertically down. **D,** Palatal view after reconstruction. **E,** The final occlusion after the complete mobilization of the two halves of the maxilla. *From Obwegeser HL: Orthognathic surgery and a tale of how three procedures came to be: a letter to the next generations of surgeons.* Clin Plastic Surg *34:341, 2007, Figure 10.*

advance the maxilla as far forward as he needed to because he had a blood supply that was, surprisingly, still satisfactory.[156] He then used blocks of cancellous bone from the iliac crest to fill the horizontal and vertical gaps.[156]

On September 5, 1969, Obwegeser completed the first simultaneous sagittal splitting of the ramus of the mandible and a Le Fort I osteotomy on a patient. He realized that he could establish the occlusion with either procedure and that he needed to vertically lengthen the mandible while at the same time setting it back. Thus, "both jaws had to be moved to improve the aesthetic result."[156] He used his clinical aesthetic judgment in combination with a review of a

cephalometric radiograph to confirm what needed to be done. With the maxilla and the mandible completely freed up, he placed them into occlusion as a single unit. He then repositioned the maxillomandibular complex "according to [his] imagination."[156] When he was satisfied with the aesthetic result, he fixed the maxilla and then the mandible in place with wires and bone grafts. He maintained the patient in IMF for 6 weeks, after which he reported, "The occlusal and facial aesthetic results were excellent!"[154]

Only minor modifications have been introduced to the basic Obwegeser design of the maxillary Le Fort I osteotomy. According to Dr. Obwegeser, "In time, internal fixation with plates and screws became a reality. In 1968, Hans Luhr published his [initial] work with plate and screw fixation. It revolutionized internal fixation, limiting the need for extended IMF and increased [the osteotomy] stability."[156]

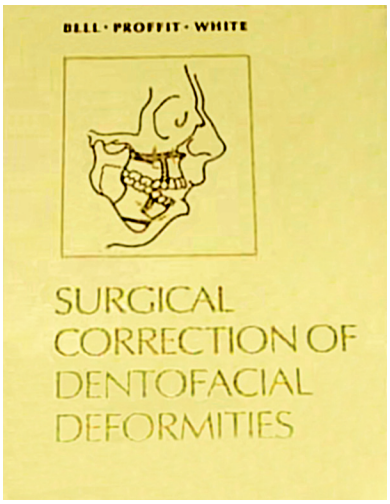
William H. Bell: Experimental Studies to Confirm the Biologic Basis of the Standard Orthognathic Procedures*

William H. Bell attended a Jesuit high school, college, and dental school in St. Louis, Mo (Fig. 2-26), and he would be forever affected and influenced by the Jesuit challenge to "be men for others." After completing dental school in 1954, he interned in oral surgery in New York City at the Metropolitan City Hospital. He recalls that "in any given week, [he] would literally see hundreds of patients who [were] candidates for either orthognathic surgery or orthodontics. Unfortunately, none of them received any treatment."[156] In 1955, he served as resident in oral surgery at the Jefferson Davis Hospital in Houston, Tex. Dr. Edward C. Hinds was the program director, and, at the time, it was considered one of the country's finest programs in reconstructive mandibular surgery. Hinds had refined and clinically applied the extraoral vertical ramus osteotomy technique for the correction of mandibular prognathism. The program was also strong in the management of facial trauma, particularly mandibular and midface fractures. In 1956, Dr. Bell was the first teaching fellow in oral surgery at M.D. Anderson Hospital in Houston, Tex. His clinical work was limited to extracting teeth and the treatment of benign tumors and cysts of the jaws, but he had the opportunity to observe innovative head and neck cancer surgery. According to Dr. Bell, "Dr. Hinds and the other American oral surgeons in Houston were aware of descriptions of both anterior and posterior maxillary osteotomies, but none were performed for orthognathic problems at that time"(personal communication). Bell envisioned the potential of maxillary osteotomies for the correction of dentofacial and posttraumatic deformities. He searched the English literature available to him for relevant studies, but he found none that supported the biologic foundation of these procedures. Dr. Sumpter Arnim encouraged Bell's interest in studying the circulation requirements of jaw osteotomies and offered experimental rabbits for use. After visiting the laboratories of orthopedic surgeons Drs. Rhinelander and Baraquay in Cleveland, Ohio, Dr. Bell performed a preliminary pilot study in rabbits to work out the details of microangiographic and histologic laboratory techniques (personal communication). This initial work gave support to these techniques for studying the biologic basis for jaw osteotomies. While at the University of Texas Dental Branch of the Texas Medical Center in Houston, Tex, and with support from U.S. Public Health Service grants, Dr. Bell carried out the first of his many revascularization bone-healing studies after completing maxillary and mandibular osteotomies with the use of adult rhesus monkeys, mongrel dogs, and baboons. In his first revascularization publication, Dr. Bell states the following: "Despite numerous clinical successes and occasional failures, the rationale for using various surgical techniques [for maxillary and mandibular osteotomies] remains virtually empiric. Basic questions concerned with the healing of the surgical wound produced by maxillary osteotomies and the vessels necessary to maintain blood supply to the bone segments and viability of the teeth have not been investigated."[24] Nine further animal laboratory studies would eventually be completed from 1969 to 1995, and these would validate the circulatory integrity of all of the basic osteotomies used in orthognathic surgery.[25-35]

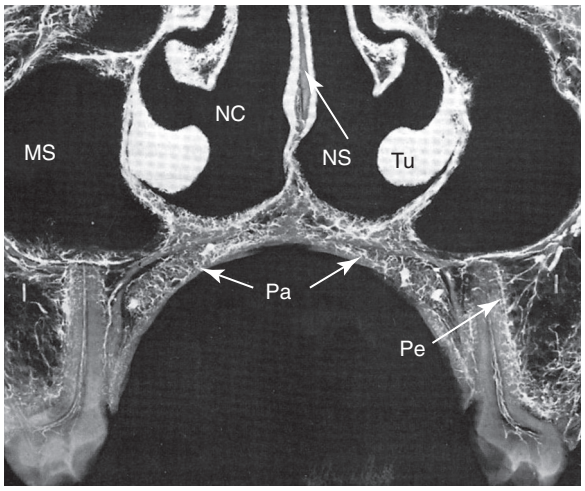
In 1971, Dr. Bell was recruited by Dr. Robert Walker to the University of Texas Southwestern Medical Center in Dallas, Tex, to develop a research laboratory that focused on the vascularity and wound healing associated with maxillary and mandibular osteotomies (Fig. 2-35). This provided the opportunity to continue animal studies and to apply the knowledge gained in the laboratory to the solving of clinical challenges. For the next 20 years, he worked with and

• **Figure 2-35** Photograph of Robert V. Walker (1924-2011). *From Fonseca RJ, Barber D, Powers MP, Frost DE:* Oral and maxillofacial trauma, *ed 4, St. Louis, 2013, Saunders.*

*Please note that this section was reviewed for accuracy by Dr. Bell.



• **Figure 2-36** A textbook entitled *Surgical Correction of Dentofacial Deformities,* which was edited by W. Bell, W. Proffit, and R. White and which was published by W.B. Saunders in 1980. This was the first comprehensive textbook to cover the subject of orthognathic surgery.



• **Figure 2-37** Microangiogram of a 1-mm transverse tissue slice from the molar region of a control animal. Areas are indicated as follows: palatal *(Pa)*, maxillary sinus *(MS)*, and nasal cavity *(NC)* blood vessels, penetrating bone and anastomosing with intramedullary blood vessels *(I)* and periodontal vascular plexus *(Pe)*, turbinate *(Tu)*, and nasal septum *(NS)*. *Courtesy William Bell.*

trained 27 oral and maxillofacial surgery residents in his research laboratory at the University of Texas Southwestern Medical School. He also conducted weekly dentofacial deformity conferences for surgical residents, faculty, and visitors from every corner of the world as well as for Dallas community orthodontists and surgeons. Beginning in 1973 and continuing through 1985, Dr. Bell, Dr. Raymond White, and Dr. William Proffit wrote and edited the text *Surgical Correction of Dentofacial Deformities* (Fig. 2-36). Volumes I and II of this text, which were published in 1980, became the comprehensive clinical reference for the interdisciplinary art and science of the correction of dentofacial deformities by surgeons and orthodontists. This work combined an atlas of surgical and orthodontic procedures with sound diagnostic and biologic guidelines for their application. The outstanding supporting original artwork by Bill Winn was remarkable for its clarity regarding the presenting dysmorphic maxillofacial anatomy and for its demonstration of how surgical procedures can be used to solve clinical problems.

Biologic Basis for the Maxillary Anterior Segmental Osteotomy

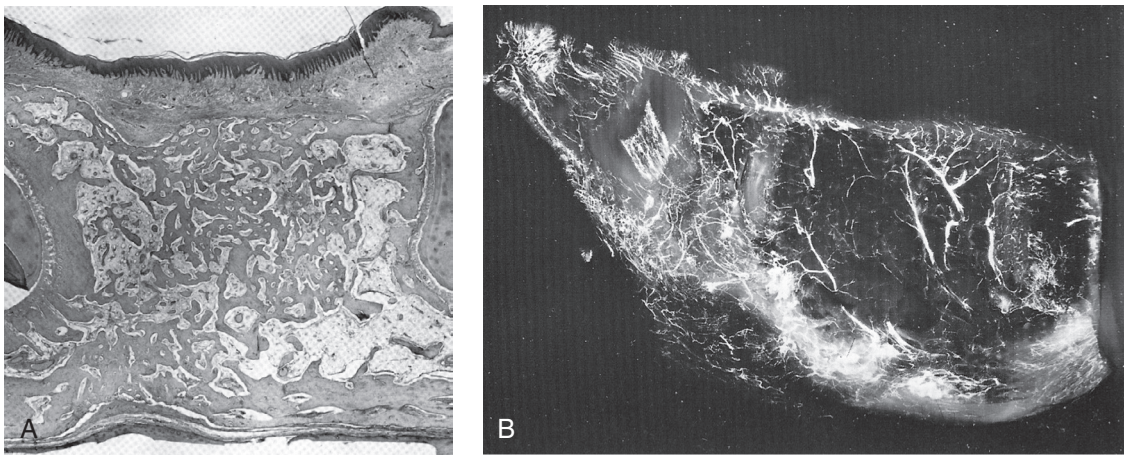
Bell WH: Revascularization and bone healing after anterior maxillary osteotomy: a study using adult rhesus monkeys.* J Oral Surg *27:249–255, 1969

An investigation was designed by Bell and colleagues to delineate the biology of anterior maxillary osteotomy wound healing. This was done by studying revascularization and bone regeneration in monkeys with the use of microangiographic and histologic techniques after completing an anterior maxillary osteotomy. Standard anterior maxillary osteotomies (i.e., labial versus palatal pedicle) were completed with the Wassmund technique, and the animals were sacrificed at 1, 3, and 6 weeks after surgery. Before sacrifice, the common carotid arteries were exposed, cannulated, heparinized, and perfused with a suspension of Micropaque injection medium. Each maxilla was then dissected from the specimen, and radiographs were taken. The 1-week specimens confirmed a "blood clot in the center of the osteotomized fragments bounded by proliferating young granulation tissue."[24] The 3-week specimens "showed early callus formation between the bone fragments. Considerable subperiosteal new bone formation was present in some of the sections."[24] The 6-week specimens "showed osseous union of the osteotomized bone fragments with no evidence of necrosis."[24] According to Dr. Bell, "The results indicated that no single blood vessel, such as the incisive canal or greater palatine arteries, is essential to maintenance of circulation to the anterior maxillary fragment. Interosseous and soft tissue collateral circulation and the freely anastomosing gingival, palatal, floor of the nose and periodontal plexuses permit many variations of the anterior maxillary osteotomy technique [labial versus palatal pedicles] without detriment to the integrity of the blood supply to the anterior maxillary segment"[24] (Fig. 2-37).

Biologic Basis for the Anterior Mandibular Segmental Osteotomy

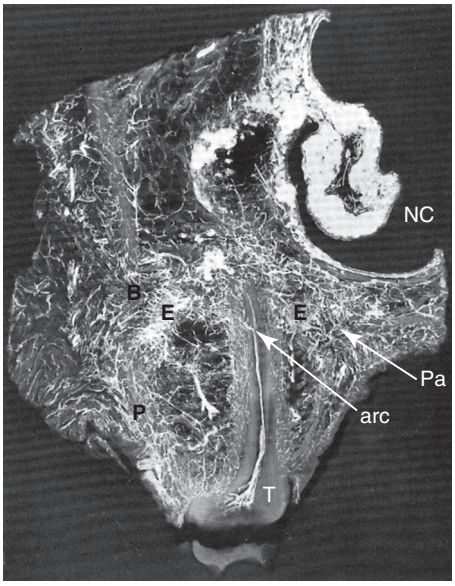
Bell WH, Levy BM: Revascularization and bone healing after anterior mandibular osteotomy.* J Oral Surg *28:196–203, 1970

In 1970, Bell and Levy published an experimental animal study to analyze the biologic basis for wound healing and revascularization after anterior mandibular segmental



• **Figure 2-38 A,** Photomicrograph of a section of the mandible of an animal 6 weeks after osteotomy. The osteotomy site is replaced by cancellous bone. Hematoxylin-eosin stain. Original magnification ×10. **B,** Microangiogram demonstrating the generalized distribution of injection medium throughout the anterior mandibular bone fragment 6 months after anterior mandibular osteotomy. Reconstitution of normal vascular architecture. Sagittal section. *Courtesy William Bell.*

osteotomy in adult rhesus monkeys (n = 11). Half of the experimental animals underwent a one-stage anterior mandibular segmental osteotomy that involved six teeth. The procedure was performed through a circumgingival degloving labial flap. The lingual soft-tissue pedicle remained attached to the osteotomized segment. Second bicuspids were extracted, and the mandibular anterior segment was set back. The second group underwent the same one-stage anterior mandibular segmental osteotomy, but this was performed through a labial vestibular incision with the maintenance of a gingival cuff around the labial aspect of the teeth. The lingual soft-tissue pedicle also remained attached to the osteotomized segment. The repositioned (set-back) anterior mandibular segment was stabilized with a prefabricated acrylic splint that was ligated to the teeth with interdental wires. The animals were sacrificed at specific postoperative intervals at weeks 1, 3, 6, and 24. Microangiograms and histologic slides were reviewed. The analysis of the data demonstrated that intraosseous and intrapulpal circulation to the anterior mandibular segments was maintained with the use of both of the surgical flap designs. The dento-osseous–musculomucosal flaps healed by osseous union within 6 weeks. In the control animals—in which the complete degloving of the labial and lingual soft tissues from the bone occurred—intraosseous necrosis, vascular ischemia, and nonunion resulted (Fig. 2-38).



• **Figure 2-39** Microangiogram of 1-mm transverse tissue slice taken from the first molar region of an experimental animal 3 weeks after posterior maxillary osteotomy. This image demonstrates the proliferation of endosteal *(E)* and periosteal *(P)* circulatory beds; the reattachment of buccal and palatal mucoperiosteal flaps to underlying bone; and the vascularization of pulp canal from periodontal vascular plexus and accessory root canal *(arc)*, buccal *(B)*, and palatal *(Pa)* osteotomy sites; the nasal cavity *(NC)*; and the first molar tooth *(T)*. *Courtesy William Bell.*

Biologic Basis for the Posterior Maxillary Segmental Osteotomy

Bell WH, Levy BM: Revascularization and bone healing after posterior maxillary osteotomy. **J Oral Surg *29(5):313–320, 1971***

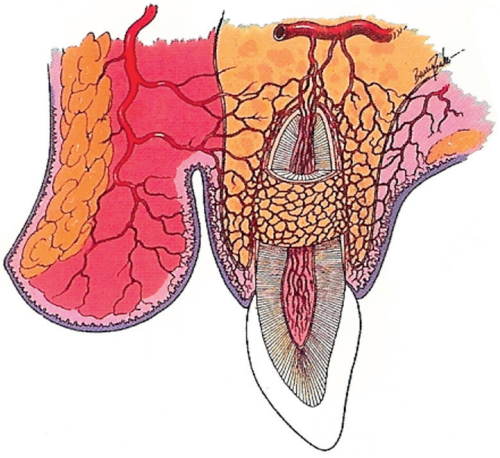
An animal model investigation was designed by Bell and Levy to clarify the biology of wound healing after posterior maxillary osteotomies with the Schuchardt technique (Fig. 2-39). In half of the posterior maxillary osteotomy flaps, the greater palatine vessel was ligated. The microangiographic and histologic studies were completed. At 1 week after surgery, the dento-alveolar segments remained freely movable. By 3 weeks, mobility had greatly diminished. After 6 weeks, the clinical union of the osteotomy segments

was seen. The individual teeth and the mobilized bone segments remained stable. There was minimal osteonecrosis and only transient vascular ischemia, and osseous union between most of the osteotomized segments occurred. No difference was seen with or without the ligation of the palatine vessels. The results of the experimental study suggested that "clinically analogous single-stage posterior maxillary osteotomies are biologically sound."[26]

Revascularization and Bone Healing after Maxillary Corticotomies

Bell WH, Levy BM: Revascularization and bone healing after maxillary corticotomies.* J Oral Surg *30:640–648, 1972

In 1972, Bell and colleagues published an experimental animal study to analyze the biologic basis for revascularization and bone healing after maxillary interdental corticotomies with the use of the Köle technique (Fig. 2-40). Five adult rhesus monkeys underwent Köle type procedures and analysis. One-stage maxillary corticotomies were performed bilaterally in the premolar and incisor regions in four monkeys; one monkey served as a control. Corticotomies were completed in each dento-osseous segment. These segments were then mobilized by a chisel between the corticotomy sites, and the wounds were closed. The mobile segments were stabilized with a preoperatively made acrylic splint by ligating each tooth to the splint with interdental wires. The experimental monkeys were sacrificed at 1, 7, 21, and 63 days after surgery. Microangiographic and histologic slides were reviewed. The procedures that were carried out were found to have a destructive effect on the maxillary incisors. The intraosseous and interpulpal circulation also appeared to be imperiled.



• **Figure 2-40** Illustration of blood supply to the anterior maxilla. The vascular architecture—which consists of freely anastomosing gingival plexus, palatal plexus, periodontal plexus, labial artery, intra-alveolar vessels, apical vessels, and pulp vessels—permits anterior maxillary osteotomies to be performed without compromising circulation to the anterior maxilla and the teeth. *Courtesy William Bell.*

Biologic Basis for the Le Fort I (Down-Fracture) Osteotomy

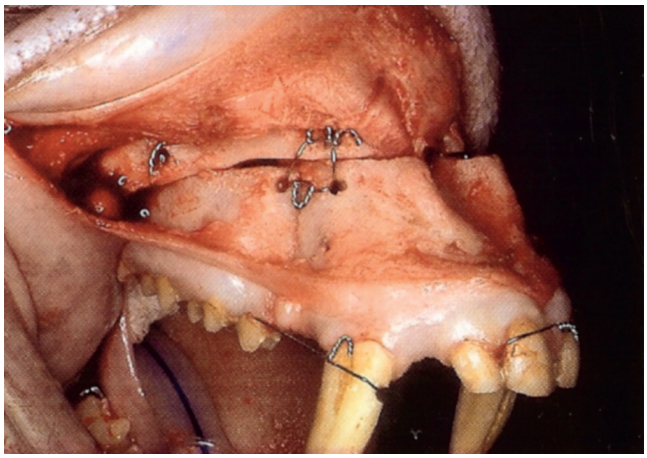
Bell WH, Fonseca RJ, Kenneky JW, Levy BM: Bone healing and revascularization after total maxillary osteotomy.* J Oral Surg *33:253–260, 1975

In 1975, Bell and colleagues published an experimental animal study that confirmed the biologic basis for bone healing and revascularization after total maxillary (Le Fort I) osteotomy carried out through a circumvestibular incision followed by down-fracture and disimpaction via the Obwegeser technique. Twelve adult rhesus monkeys underwent single-stage total maxillary osteotomy according to Obwegeser's protocol (Fig. 2-41). Two non-operated animals served as controls. In seven animals, the greater palatine arteries were preserved; in three animals, they were transected. The animals were sacrificed at the following intervals: immediately; after 2 days; and 1, 2, 4, 6, and 12 weeks after surgery. In all of the animals, the wounds healed without signs of infection or dehiscence. Revascularization and bone healing were studied with the use of microangiographic and histologic techniques (Fig. 2-42). Within 1 week after surgery, there was increased filling of both the endosteal and periosteal vascular beds. By 4 weeks, a callus that was composed of fibrous connective tissue and young bone was seen histologically. By 6 weeks, the callus contained mature bone. The 12-week specimen showed complete osseous bridging between fragments. Interestingly, the ligation of both greater palatine arteries had only minimal short-term effects on the perfusion of Micropaque through the intraosseous, intrapulpal, or soft tissues.

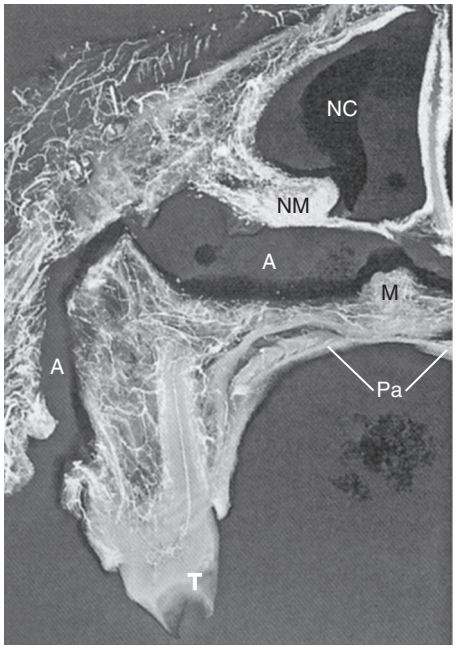
Biologic Basis for the Vertical Oblique Osteotomy of the Mandible

Bell WH, Kennedy JW, 3rd: Biological basis for vertical ramus osteotomies—a study of bone healing and revascularization in adult rhesus monkeys.* J Oral Surg *34:215–225, 1976

In 1976, Bell and Kennedy completed a microangiographic and histologic study of adult rhesus monkeys (n = 15) to evaluate the biologic basis for the vertical ramus osteotomies that were commonly in clinical use. Bone healing and revascularization of the proximal condylar segment were studied after pedicled and nonpedicled vertical ramus osteotomies were completed. The animals were sacrificed at specific postoperative intervals (1, 2, 3, 6, and 12 weeks), and microangiograms and histologic slides were reviewed. Specimens for which the proximal segment was not pedicled to soft tissue showed intraosseous necrosis, vascular ischemia, and delayed healing. Early osseous union, minimal osteonecrosis, and minimal vascular ischemia were found in pedicled specimens. The study confirmed that the circulation and viability of the proximal condylar segment was maintained after standard vertical ramus osteotomy as long as it was pedicled to the lateral pterygoid muscle and the articular capsule.



• **Figure 2-41** Clinically analogous pedicled down-fractured four-segment Le Fort I osteotomy technique performed in rhesus monkey. *Courtesy William Bell.*



• **Figure 2-42** Microangiogram of the molar region of experimental animal 2 days after surgery, revealing generalized distribution of the injection medium in soft tissues, bone, and pulp canals of the molar *(T)* as well as an avascular space *(A)* between superior surface of the maxilla *(M)* and detached nasal mucosa *(NM)*. *Pa,* Palate; *NC,* nasal cavity. *Courtesy William Bell.*

Biologic Basis for the Sagittal Split Osteotomy of the Mandible

Bell WH, Schendel SA: Biologic basis for modification of the sagittal ramus split operation.* J Oral Surg *35: 362–369, 1977

In 1977, Bell and Schendel published an experimental animal study to analyze the biologic basis for vascularization, revascularization, and bone healing in conjunction with sagittal ramus split procedures (Fig. 2-43). Ten adult rhesus monkeys were used. In each animal, on one side, the procedure was completed with complete subperiosteal stripping on the medial and lateral sides of the ramus osteotomy segment. On the contralateral side, the maximizing of the soft-tissue attachments on the medial and lateral side of the osteotomies was carried out. The animals were sacrificed at specific postoperative intervals (immediately or at 1, 2, 3, 6, 12, or 24 weeks). Microangiograms and histologic slides were reviewed at each time interval. The results of the experimental study confirmed that the Obwegeser technique of sagittal ramus osteotomy was biologically sound. Intraosseous ischemia and osteonecrosis were dramatically reduced when the mucoperiosteum and the pterygomasseteric sling were maintained to the repositioned proximal segment.

Biologic Basis for a "One Tooth" Dento-Osseous Segmental Osteotomy

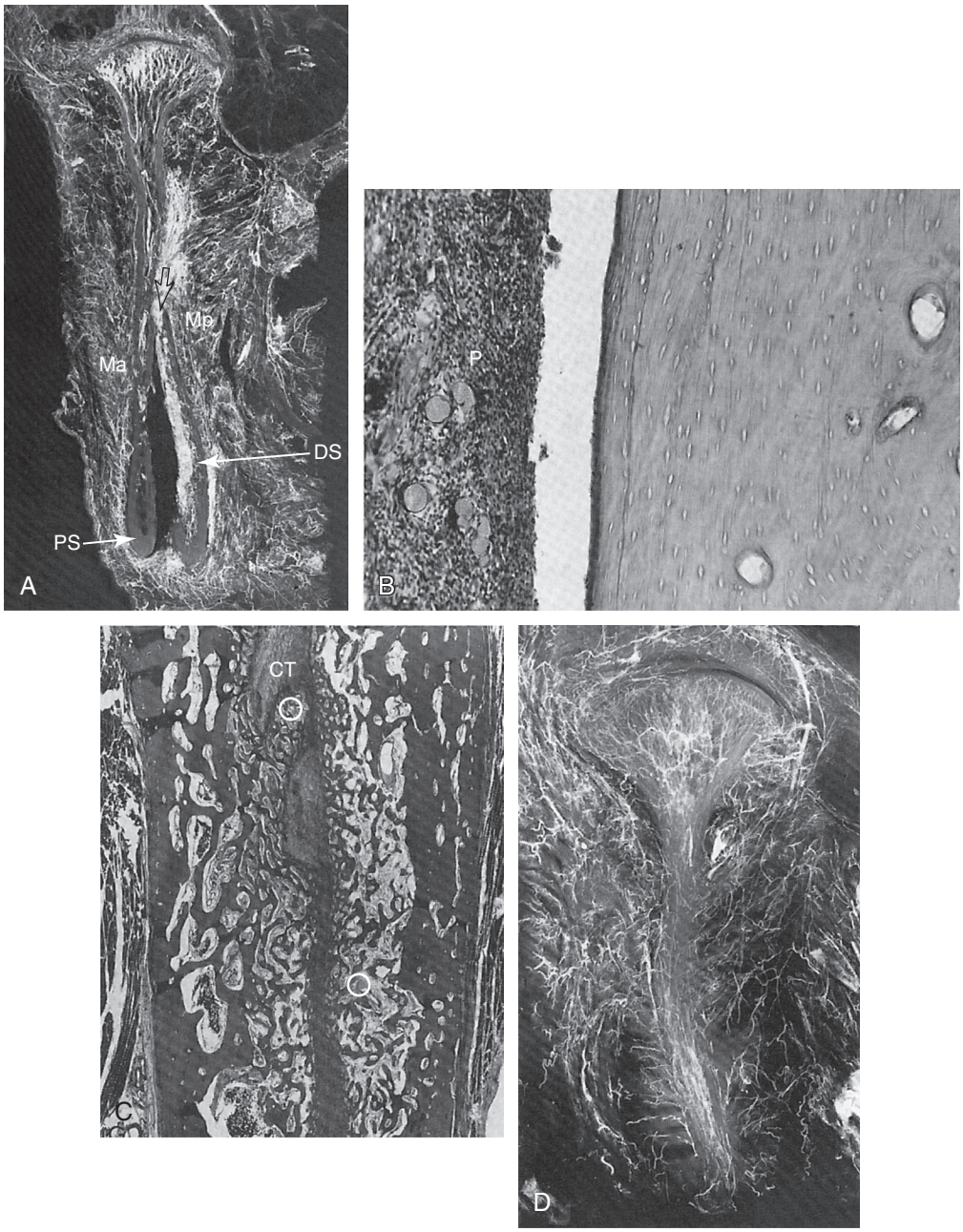
Bell WH, Schendel SA, Finn RA: Revascularization after surgical repositioning of one-tooth dento-osseous segments.* J Oral Surg *36:757–765, 1978

In 1978, Bell, Schendel, and Finn published an experimental animal study to analyze the biologic basis for wound healing and revascularization after "one-tooth" dento-osseous segmental osteotomies in six adult mongrel dogs**.** Half of the experimental animals underwent a two-stage (single-tooth dento-osseous segment) approach, whereas the other group underwent a one-stage (single tooth dento-osseous segment) approach. The animals were sacrificed at specific postoperative intervals (3 days or 1, 3, 4, or 8 weeks). Microangiograms and histologic slides were reviewed. The data showed early transient vascular ischemia, minimal osteonecrosis, and osseous union between most of the osteotomized segments. The study parameters showed that necrosis occurred at a few of the interdental osteotomy sites. The authors stated that the "excessive removal of interseptal and interradicular bone may cause protracted bone healing and have a destructive effect on the periodontium."[33] The authors cautioned that the single-tooth dento-osseous segments were critically dependent on the preservation of viable circulation through the soft-tissue attachments to the underlying bone. This was by way of a freely anastomosing vascular plexus of the gingiva, the palate, the periodontium, and the dental pulp.

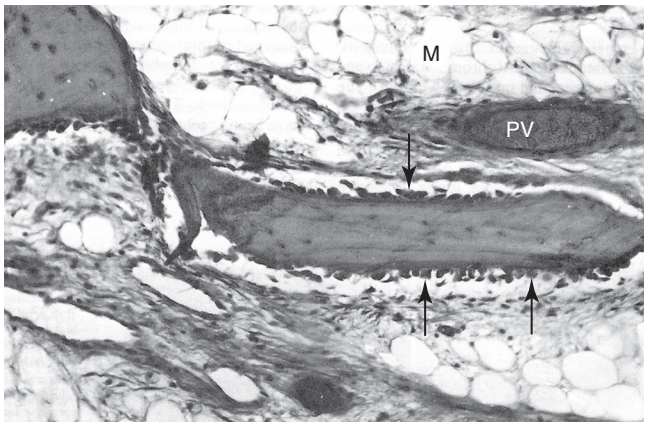
Biologic Basis for the Oblique Osteotomy of the Chin

Storum KA, Bell WH, Nagura H: Microangiographic and histologic evaluation of revascularization and healing after genioplasty by osteotomy of the inferior border of the mandible.* J Oral Maxillofac Surg *48:210–216, 1988

In 1988, Storum, Bell, and Nagura completed microangiographic and histologic studies in adult rhesus



• **Figure 2-43. A,** Microangiogram of a transverse section of vertical ramus 1 week after conventional sagittal split ramus osteotomy technique showing decreased perfusion of Micropaque into the distal region of the proximal segment *(PS)* and increased filling of the endosteal circulatory bed in the medial aspect of the distal segment *(DS)*. *Ma,* Masseter muscle; *Mp,* medial pterygoid muscle. Arrows indicate the osteotomy site. **B,** Photomicrograph of the demarcated area of proximal segment showing detached periosteum *(P)* and subjacent avascular bone with empty lacunae. **C,** Histologic appearance of osteotomy site in the vertical ramus 6 weeks after modified sagittal split ramus osteotomy technique. There was some bridging of the proximal and distal bone segments with mature fibrous connective tissue *(CT)* and focal areas of viable vascularized osteophytic bone *(O)*. **D,** Microangiogram of a 1-mm section through the ramus and condyle of a conventional saggittal split ramus osteotomy specimen 6 months after surgery showing vascular architecture similar to that seen in control animals. *Courtesy William Bell.*



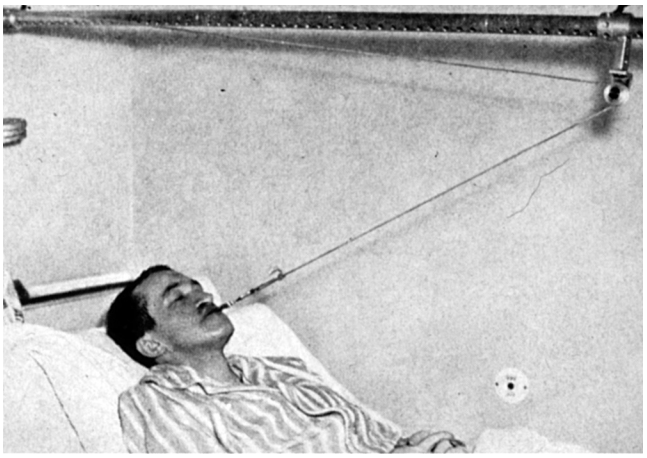
• **Figure 2-44** Biologic foundation for the use of an advancement osseous genioplasty with the maintenance of a soft-tissue pedicle. An adult *Macaca nemestrina* monkey was used as the experimental model. The genial segment was advanced 5 mm and fixed in the planned position with interosseous wires. This is a microangiogram of a 2.0-mm mid-sagittal tissue slice from an experimental animal that was sacrificed 1 week after advancement genioplasty. The photomicrograph shows vascularized and vital bone in the center of the genial segment. Proliferating osteoblasts *(arrows)* line viable trabecular bone, and bony lacunae are filled with osteocytes. Hematoxylin-eosin stain, ×125. *M,* Medullary space; *PV,* small perfused blood vessel. *Courtesy William Bell.*

monkeys. These studies indicated that an intra-oral pedicled genioplasty involving an osteotomy of the inferior mandibular border maintained circulation and osseous viability after the manipulation and repositioning of the chin segment (Fig. 2-44). Circulation to the dental pulp was not discernibly affected.[182]

Biologic Basis for the Le Fort I (Down-Fracture) Osteotomy with Segmentation

Bell WH, You ZH, Finn RA, Fields RT: Wound healing after multisegmental Le Fort I osteotomy and transection of the descending palatine vessels. **J Oral Maxillofac Surg *53:1425–1433, 1995***

In 1995, Bell and colleagues completed a clinically analogous four-segment LeFort I (down-fracture) osteotomy through a circumvestibular incision via the Obwegeser technique in nine adult rhesus monkeys. The animals were sacrificed after surgery on day 0, 3, 7, 14, or 28. Revascularization and bone healing were studied by microangiographic and histologic techniques. The findings indicated that the palatal mucosa, the labial–buccal gingiva and mucosa provide adequate nutrient pedicles for Le Fort I down-fractured segmental osteotomies accomplished through a circumvestibular incision. It was documented that segmentalization, the stretching of the vascular pedicles (within physiologic limits), and the transection of the descending palatine vessels have only transitory discernable effects on revascularization and bone healing.



• **Figure 2-45** Roll extension with the application of 1.5 kg of weight on the side of the hospital bed. Disimpaction of the maxilla was gradual and occurred over a period of 2 weeks. *Courtesy Hans Luhr.*

Hans G. Luhr: Development of Plate and Screw Fixation for Craniomaxillofacial Surgery*

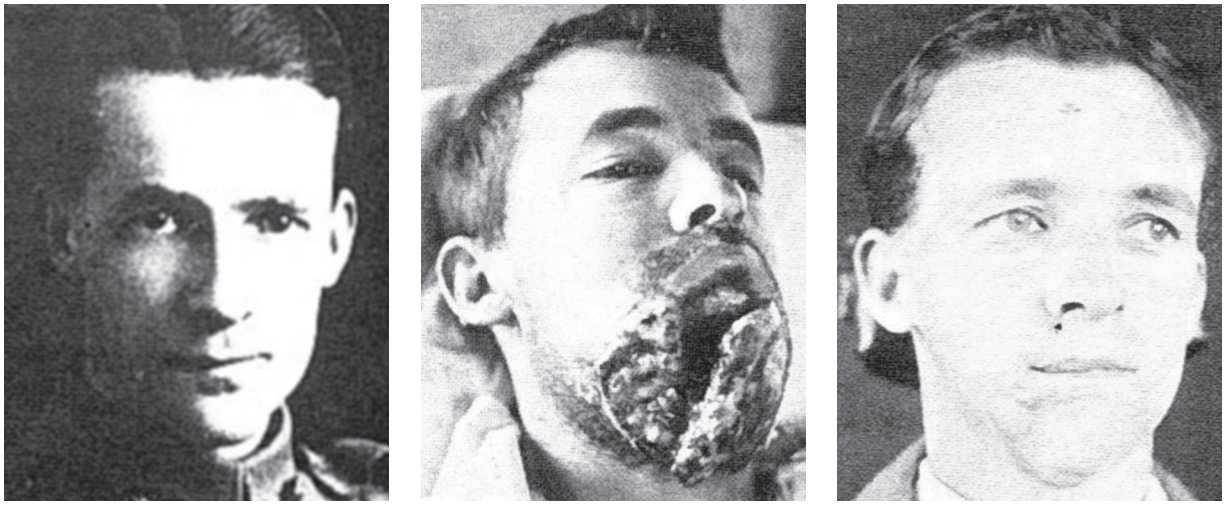
Hans Luhr was born in Germany in 1932. Like so many others during this time, his childhood years were disrupted by World War II. He graduated from the University of Bonn with a degree in medicine in 1958 at the age of 26, and he went on to complete a degree in dentistry in 1960. He worked as a ship's physician, and he then completed his postgraduate training and research in general pathology at the University of Bonn. In 1961, after receiving a public health scholarship award from the German Developing Aid Program, he served as a dentist in charge of a mobile unit that provided aid in the hinterland of Liberia in Western Africa.

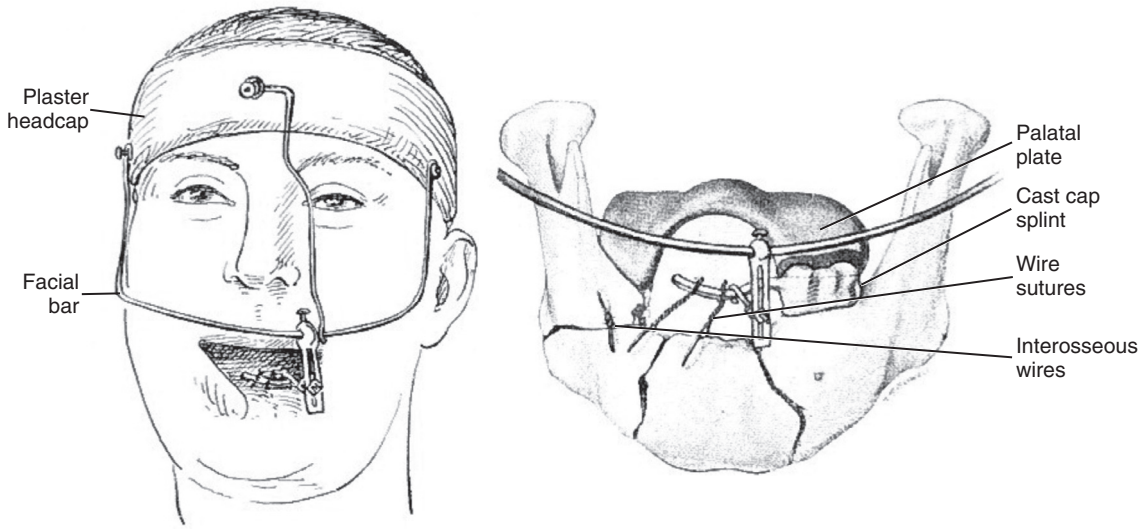
In 1963, Luhr began his training in maxillofacial surgery under the direction of Professor Karl Schuchardt. At that time, the Hamburg unit was considered the most prestigious in Germany. It was a regional maxillofacial trauma center, and, because Schuchardt had pioneered segmental osteotomies of the maxilla, there was much activity concerning jaw deformities with malocclusion. Schuchardt had also developed the acrylated arch bar, which was considered the treatment of choice for most mandibular fractures as well as for stabilization after elective mandibular osteotomies. Until Obwegeser described the Le Fort I down-fracture procedure with full mobilization, the Schuchardt method of horizontal Le Fort I osteotomy without pterygoid–maxillary disjunction was the procedure of choice. The incompletely mobilized maxilla was then managed by a "roll extension" and the application of 1.5 kg of weight via a sack of sand that was attached over the end of the hospital bed (Fig. 2-45). Disimpaction was gradual and

*Please note that this section was reviewed for accuracy by Dr. Luhr.

occurred over a 2-week period. When the preferred occlusion was finally realized, the jaws were secured with IMF.

Although it was clear that interosseous wire fixation was not rigid enough to counteract the masticatory muscle forces, it and other methods of fixation (e.g., plaster head cap with reinforced bar, palatal plate, cast cap splint, wire suture) were all that was known for the treatment of mandibular fractures and elective osteotomy stabilization (Fig. 2-46). Extended periods of IMF were also applied in an attempt to achieve healing. With these limited fixation and immobilization methods, complications such as osteomyelitis and nonunion frequently occurred, especially in the mandible. Skeletal wire suspension via the "belt-and-suspenders approach" had been introduced by Milton Adams in 1942 for the added stabilization of midface fractures, and this approach was later applied to elective jaw (orthognathic) osteotomies (Fig. 2-47).[1] These suspension wires were occasionally supplemented by threaded Steinmann pins as clinicians searched for improved ways of establishing stable fixation. Long periods of IMF continued to be the workhorse of immobilization in an attempt to achieve bony union of the jaws. Prolonged periods of limited mouth opening had negative effects on speech, breathing, chewing, swallowing, hygiene, and temporomandibular joint mobility. As a resident in training, Dr. Luhr viewed these maxillofacial challenges with fresh eyes. Appreciating the limitations and hazards of these methods of immobilization and with background knowledge of what was known at the time about long bone fractures, he hypothesized that the addition of compression across a mandibular fracture would

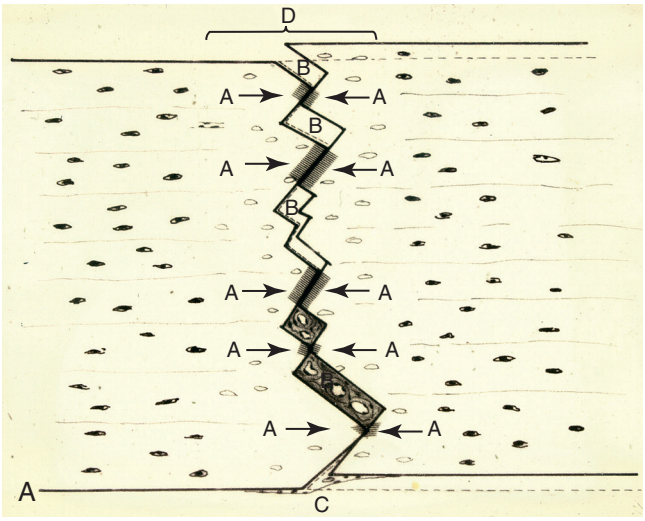




• **Figure 2-46** Kazanjian published an example of his management of a complex facial injury that was sustained by a soldier during World War I. The wound involved the soft tissues and skeletal structures of the face. Kazanjian used a combination of a plaster head cap, a facial bar, a palatal plate, cast cap splints, wire sutures, and interosseous wires to establish bony healing and soft-tissue maintenance. *From Kazanjian VH, Converse EJ:* The surgical treatment of facial injuries, *Baltimore, 1949, The Williams and Wilkins Company.*

provide the necessary interfragmentary stability and eliminate the need for intermaxillary fixation. With research interests in the biomechanics of bone healing and jaw fracture management in particular, Luhr established an animal laboratory to study these issues. His experimental dog model involved a traumatically induced mandibular fracture. The fracture model confirmed that interfragmentary pressure created by a compression bone plate was at least partially maintained during the healing period (i.e., approximately 50% at 4 weeks). Further experiments by Luhr determined that the use of compression increased the rate of strength during fracture union as compared with fixation techniques without the use of compression. Interestingly, Luhr found that, with an actual fracture model, the histologic pattern of primary bone healing differed significantly from the descriptions by Schenk and Willenegger (i.e., Association for Osteosynthesis/Association for the Study of Internal Fixation investigators), who used a "saw-cut" tibia osteotomy dog model.[129] During healing, these researchers observed the primary penetration of the osteotomy line via an ossification process that originated predominantly or exclusively in the cells and vessels within the Haversian canals. They termed this process *intracanalicular osteogenesis,* and they also called it *contact primary bone healing.* In their "saw-cut" tibia osteotomy dog model, the bone surfaces were in direct apposition to one another as fusion occurred. This was in contrast with "microgap healing," which was described by Luhr in the mandible fracture model (Fig. 2-48, *A*). Luhr's experimental model more closely simulated a real-life mandibular fracture. He found that the microscopic reduction of the fracture did not occur, despite the fact that the fracture appeared to the clinician to be grossly reduced. Small gaps still existed between the opposing bone spicular surfaces. Blood vessels and cells that originated in the endosteal cavity and in the periosteal side first invaded the microgaps between the opposing spicular surfaces. Lamellar bone was then deposited on the partially

• **Figure 2-47** An illustration of Adams suspension wires used to stabilize a midface fracture. In the past, this was a common method that was used in an attempt to immobilize a midface fracture.



• **Figure 2-48 A,** A schematic diagram of microgap healing at the microscopic level after the compression osteosynthesis of an experimental mandibular fracture in a dog. Although the fracture looks reduced at the time of surgery, microscopically, there were discrete areas of contact and separation between the fracture ends. The regeneration of bone took place in the non-contacting areas. Blood vessels had grown in both from the endosteal cavity and the periosteal side. Lamellar bone is deposited at the partially devitalized marginal fracture ends *(C)* with empty lacunae. **B,** Primary lamellar bone is deposited on the partially devitalized fracture ends around the blood vessels (cross-sectioned), which had grown in from the endosteal cavity and the periosteal side. This is a microscopic slide from 4 weeks after the compression osteosynthesis of an experimental mandible fracture in a dog. *From Luhr HG: Callus symposion.* Nova Acta Leopold *44:85–93, 1976.*

devitalized bone within the small interfragmentary gaps, and it soon filled up the microgaps and united the fracture segments (Fig. 2-48, *B*). As soon as 4 weeks later, the fracture was already clinically solid. The Haversian system contributed only to the late remodeling of the whole fracture zone, which could take several months. However, "gap healing" was seen with regularity, and it is viewed as a form of primary bone healing.

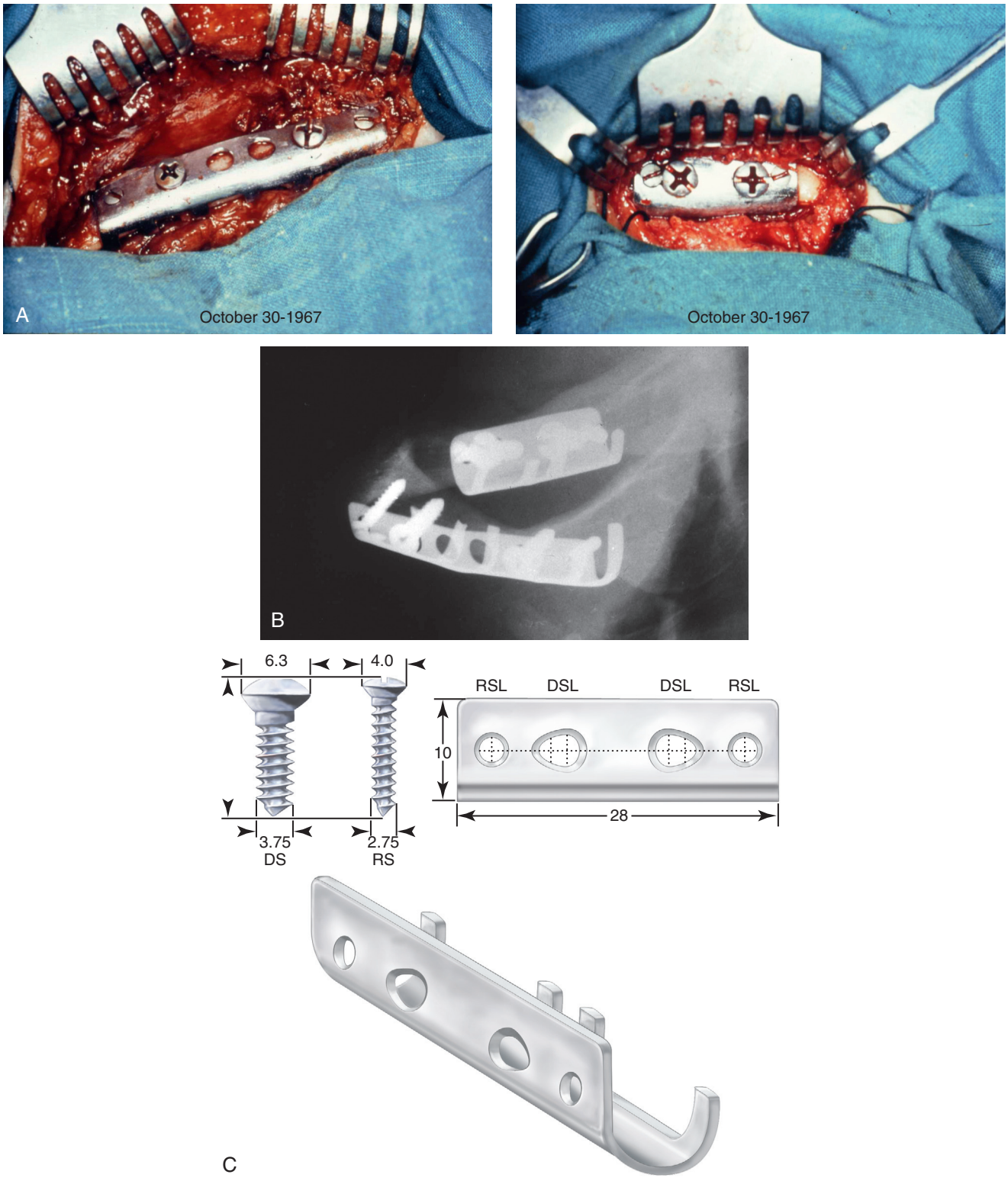
After confirming its success in experimental animals, Luhr was the first to apply maxillofacial compression osteosynthesis in a human. He did this on an edentulous woman who had sustained bilateral body fractures in an atrophic mandible (Fig. 2-49).[127] Within 6 months of this initial success, Luhr used similar compression osteosynthesis techniques on mandibular fractures in 16 additional patients, all with good results. He immediately saw the value of "self-tapping screws" in maxillofacial surgery. However, this went against the grain of general orthopedic surgeons, who at the time were beginning to use compression osteosynthesis for long bone injuries. They preferred to "pretap" the bone before the placement of fixation screws. Dr. Luhr chose Vitallium as a material, because it was a corrosive-resistant cobalt/chromium/molybdenum alloy with good strength. The small plates that he designed for use in the mandible were first modeled in wax, and they were then cast at a private dental lab. He would grind eccentric holes into each Vitallium fixation plate with the use of a diamond bur to complete the required design (Fig. 2-50). When drilling holes in the bone for the placement of self-tapping screws, Dr. Luhr initially used a hand-drill technique, because low-speed drills were not available in the operating room at that time. In 1968, Luhr reported on his initial consecutive series of mandibular fractures that involved the use of compression plating.[127] The Luhr Maxillofacial Fixation System was then manufactured by Howmedica in 1969.[133] Remarkably, all of the commercially available plate configurations in this system were designed by Luhr himself. He first completed pencil drawings to scale, and these served as design patterns for the manufacturer. This was true for all of the plating systems that Luhr introduced over the following two decades.

In January 1970, Dr. Luhr was presented with a patient who had been operated on elsewhere to correct a developmental jaw deformity that was characterized by mandibular prognathism. A Dingman osteotomy technique with interosseous wire fixation had been used, and this was followed by 12 weeks of IMF. The result was osteomyelitis with extraoral fistulas and nonunion on both sides (Fig. 2-51). Dr. Luhr initially removed the involved tooth at each osteotomy site and achieved drainage to manage the infection. He was able to stabilize each osteotomy site with the use of a compression plate with screws. A lower jaw arch bar was also left in place for 8 weeks to provide additional stability. With the stable direct fixation described, the intermaxillary fixation was immediately released, and healing was uneventful. This was likely the first case of plate and screw fixation used in orthognathic surgery. Although this case was not officially published by Dr. Luhr at the time, word quickly spread regarding this new technique of bone fixation for elective maxillofacial osteotomies.

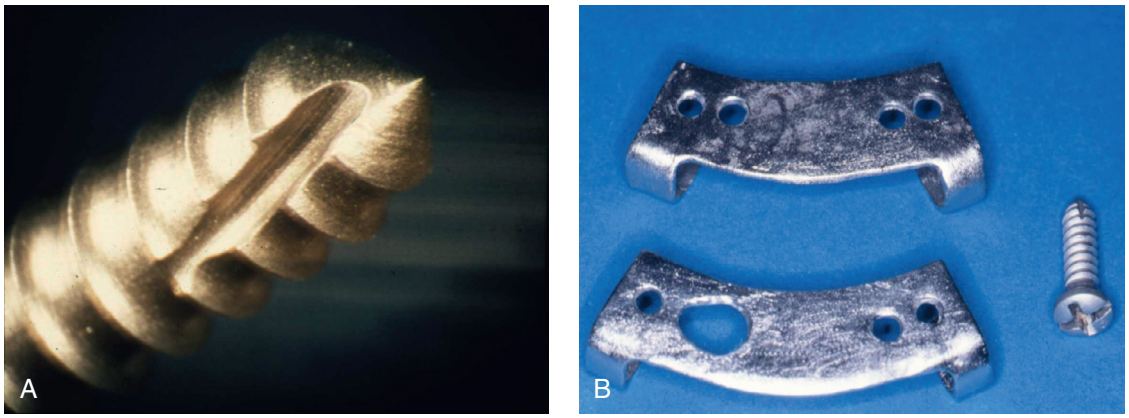
The advantages of plate and screw fixation for orthognathic surgery soon caught on. Francois Michelet and colleagues from Bordeaux, France, published their results involving mini-plate fixation of the Obwegeser sagittal split osteotomy in 1971.[139] Two years later, mini-plate stabilization of elective maxillary osteotomies was also reported, with clear illustrations demonstrating how it should be done. Maxime Champy also described the use of mini-plates and monocortical screws for the treatment of mandibular fractures as well as the use of mini-plates and screws for elective Le Fort I osteotomies.[52,53] Bernd Spiessl preferred the use of lag screw techniques to stabilize sagittal split osteotomies.[176-178] He and others soon realized that, unless this technique was meticulously executed, the use of lag screws at the sagittal split osteotomy site could easily result in condyle dislocation with malocclusion as a result of the torquing effects. For the majority of surgeons, the philosophy of osteotomy fixation for sagittal splits soon shifted to the concept of "bicortical" (non–lag screw) technique in an attempt to avoid these complications. The so-called "positional screw" technique described by Lindorf[124,125] and Niederdellmann[146-148] gradually took hold. The potential risk of condyle dislocation with the application of mandibular osteotomy fixation during orthognathic surgery was recognized by Luhr.[132] To help prevent this occurrence, he designed a temporomandibular joint positioning device, which remains a viable alternative to the freehand positioning approach that is most frequently used today (see Chapter 15).

Publications describing the value of mini-plate and screw fixation after Le Fort I osteotomy were initially described by Michelet[139] and then by Champy,[52,53] Hörster,[97] Steinhäuser,[179] and Drommer and Luhr (1981).[73,74] Dr. Luhr reported on 17 cases of Le Fort I osteotomies using mini-plate and screw fixation specifically to correct the maxillary deformity in patients with cleft palates. During the late 1970s, the advantage of Dr. Luhr's L-shaped mini-plates (specifically designed for the fixation of Le Fort I osteotomy sites) became known. The L-shaped plates overcame the difficulties of establishing fixation along the dense bicortical vertical pillars (i.e., the piriform rim region and the maxillary buttress) of the maxilla while also gaining maximum horizontal fixation directly above the roots of the teeth on the dentoalveolar side.

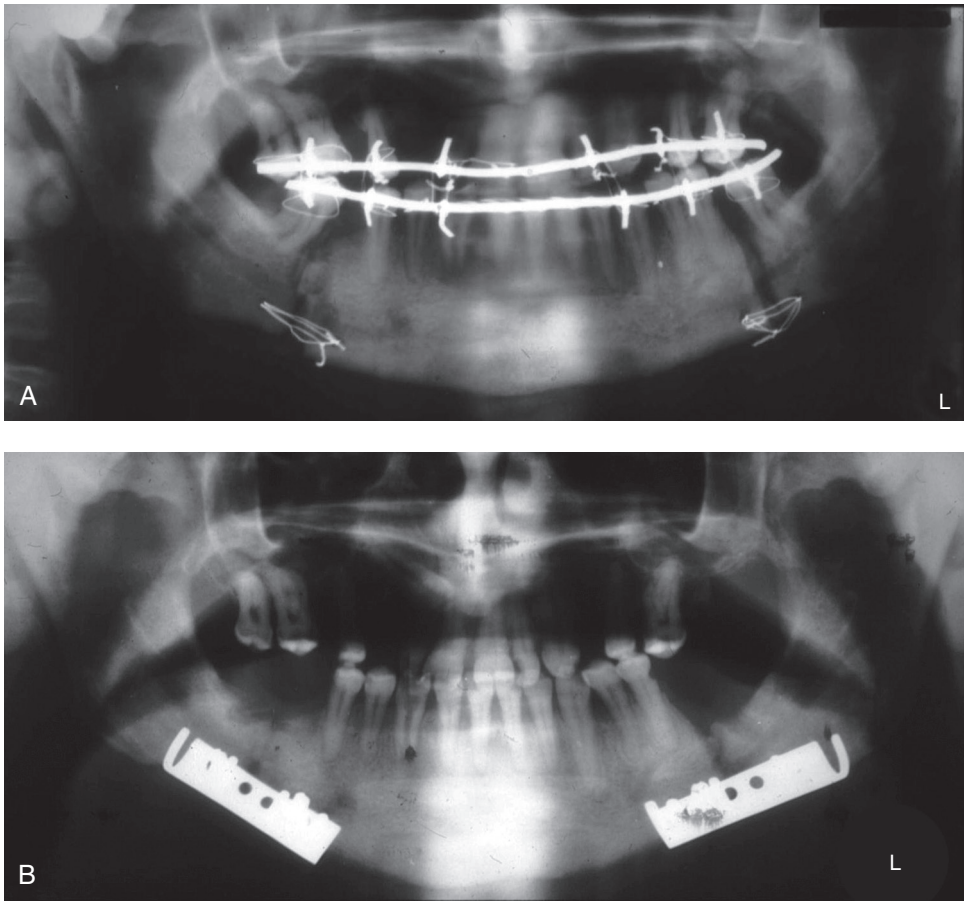
Dr. Luhr reported one of the first bimaxillary orthognathic cases in which plate and screw fixation was used at all locations without the need for intermaxillary fixation after surgery. This was for a dental student who presented with a dentofacial deformity during the early 1980s. The operation included a maxillary Le Fort I osteotomy with advancement and bilateral sagittal split osteotomies of the mandible with setback. It is believed that the first bimaxillary orthognathic procedures that involved the use of rigid fixation in the United States were completed in 1983 by Dr. Harvey



• **Figure 2-49** The first-ever compression osteosynthesis in maxillofacial surgery, which was performed by Luhr in 1967. **A,** An intraoperative view of the compression bone plates after the stabilization of bilateral body fractures of an edentulous mandible. **B,** A postoperative radiograph showing the fixation plates and screws that secured the reduced fracture. **C,** An illustration of the plate and screws (in millimeters) as designed by Luhr. *Courtesy Hans Luhr.*



• **Figure 2-50** Self-tapping screw with a cutting flute is shown (A), along with an example of the raw plates that were cast for Dr. Luhr in Vitallium by a private dental lab (B). These plates were then refinished, and eccentric gliding holes were placed by Dr. Luhr with the use of a drill with a diamond bur. *Courtesy Hans Luhr.*

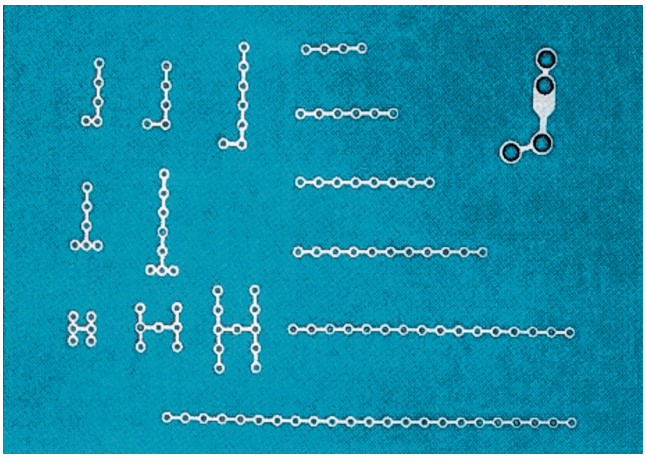


• **Figure 2-51** Osteomyelitis and nonunion resulted from a Dingman-technique osteotomy for the correction of mandibular prognathism, which included 12 weeks of intermaxillary fixation. **A,** A panorex radiograph is shown. **B,** The involved teeth were removed and drainage was achieved to manage the infection. Each of the osteotomy sites were stabilized with the use of a compression screw plate that was designed and placed by Dr. Luhr. Intermaxillary fixation was immediately released, and the final result after healing was uneventful. *Courtesy Hans Luhr.*

Rosen and Dr. Luhr (personal communication between Dr. Rosen and Dr. Luhr). In July 1983, after observing Dr. Luhr operating in Göttingen, Germany, Dr. Rosen was given a fixation set and encouraged to make use of it back home. In Philadelphia, Dr. Rosen completed bimaxillary surgery in a teenage girl with a cleft jaw deformity. He used Luhr plates and screws for the fixation of the Le Fort I and bilateral ramus osteotomies in November 1983. I served as Dr. Rosen's first assistant, and I was impressed by the new technology. The following month (December 1983), while serving as a visiting professor with Dr. Linton Whitaker at the University of Pennsylvania, Dr. Luhr demonstrated his techniques on a teenage girl with a long face mandibular deficiency anterior open bite who required a Le Fort I osteotomy with vertical intrusion and horizontal advancement as well as bilateral sagittal split osteotomies of the mandible with advancement. Under Dr. Whitaker, as a craniofacial surgery fellow, I was fortunate to have the opportunity to assist Dr. Luhr during this surgery. To rapidly disseminate the knowledge of Dr. Luhr's innovations, Dr. Whitaker organized a 1-day symposium at the University of Pennsylvania entitled "Fixation Options in Cranio-Maxillofacial Surgery." This event was attended by the leading craniofacial surgeons from throughout Canada, the United States, and Mexico. Recognizing the value of Luhr's application of plate and screw fixation to patient care, within months, this procedure became routine at their centers. Luhr's techniques were enthusiastically embraced by their residents in training and by the surgeons within their respective communities.

By the mid to late 1980s, the advantages of plate and screw fixation for use in a spectrum of elective craniofacial osteotomies, for fractures, and for tumor management was becoming known throughout the world. It was clear to Dr. Luhr that plates and screws of smaller dimension were required to accomplish clinical objectives, especially in the upper face. He designed the Luhr Micro-Fixation System, which is the smallest bone fixation system in the world; it includes screw diameters of as small as 0.8 mm and microplates of various design to match (Fig. 2-52).[133] The system's applicability to upper face clinical problems (e.g., cranial vault, orbit, zygomas, nose) was immediately clear and quickly adapted by craniofacial surgeons and neurosurgeons throughout the world.

Until the 1980s, chin osteotomy via Obwegeser technique fixation was accomplished with interosseous wires or circumsymphyseal wires. Some clinicians also used threaded Steinmann pins. During the late 1980s, Dr. Luhr published articles about a series of genioplasties in which he used plate and screw fixation on a routine basis.[134] There were few complications, and the clinical success was remarkable (Fig. 2-53). Dr. Luhr found the plate and screw fixation techniques for chin osteotomy stabilization to be especially useful when vertical lengthening and interpositional grafting were required. In 1988, Dr. Rosen also published a successful series of osseous genioplasties involving vertical lengthening with the use of Luhr (rigid) fixation.[168]



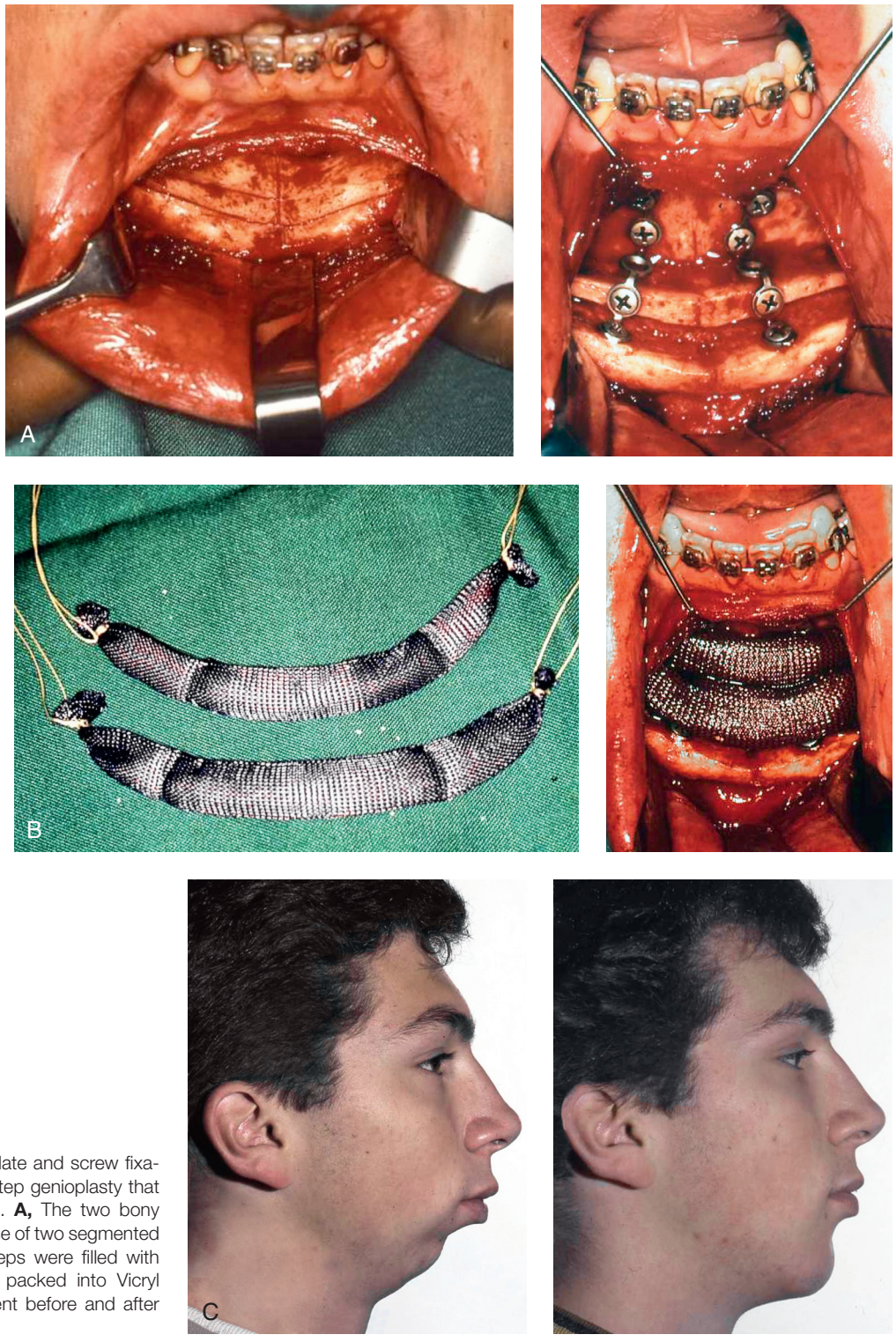
• **Figure 2-52** The first microplates designed by Dr. Luhr. A typical mini-plate from the time is shown in the upper right corner to demonstrate the difference in size. *From Luhr HG: Indication for use of a microsystem for internal fixation in craniofacial surgery.* J Craniofac Surg *1:36, 1990, Figure 2.*

As professor and head of the Department of Maxillofacial Surgery at the University of Göttingen, Dr. Luhr continued primate laboratory research and provided care to patients who had sustained facial trauma, who presented with dentofacial deformities, and who arrived with a spectrum of head and neck tumors until he retired from clinical practice in 2000. During this time, he published more than 200 scientific articles and book chapters. He continues to serve as Professor Emeritus in Maxillofacial Surgery at the University of Göttingen.

Throughout the 1960s and into the 1990s, Dr. Luhr stimulated a generation of craniomaxillofacial surgeons to embrace plate and screw fixation for trauma, elective osteotomies, and tumor management.[127-134] Luhr's initial ideas and commercially available fixation system served as a springboard for many of today's craniomaxillofacial innovations. Stable plate and screw fixation is one of the most significant advances in craniomaxillofacial surgery to have occurred during the 20th century. On the basis of his laboratory research, technical innovations, and clinical contributions, it is right to consider Dr. Luhr "the father of rigid fixation."

Paul Louis Tessier: Development of Craniofacial Surgery[101,215,216]

Paul Tessier (1917-2008) was born into a family of wine merchants in Héric on France's Atlantic Coast (see Fig. 2-30). He was an only child, and he had no family background in medicine. It has been stated that his initial ambition was to join the Navy, but he was excluded from this by early ill health. Because of his love for the outdoors, he also considered a career in forestry. In 1936, at age 19, he entered medical school. However, just halfway through his



• **Figure 2-53** The advantage of plate and screw fixation is demonstrated via a double-step genioplasty that resulted in a 20-mm advancement. **A,** The two bony segments were stabilized with the use of two segmented mini-plates and screws. **B,** The steps were filled with hydroxyapatite granules that were packed into Vicryl sacks. **C,** Profile views of the patient before and after surgery. *Courtesy Hans Luhr.*

studies, he became a prisoner of war in 1940. While he was interred, he became critically ill with a typhoid infection, but he eventually recovered. When he was released, he finally receiving his Doctor of Medicine degree from the Faculté de Médecine de Paris in 1943. When he came to Paris in 1944 (because the U.S. 8th Air Force had destroyed the hospital that he worked at in Nantes), Tessier worked with Maurice Virenque, a maxillofacial surgeon, at Hôpital Puteauz. After the war, the service moved to Foch, where there were, in essence, two maxillofacial services: one with Virenque and Tessier and one with Gustave Ginestet, an officious martinet who held the rank of General in the French Army. After Virenque died, Ginestet wrote Tessier several letters forbidding him to perform any maxillofacial

surgery; his only function was to be running the burn service. Tessier ignored Ginestet, but many of his methods of interlocking bone fixation were the result of the fact that Ginestet controlled the dental lab and forbade the technicians to provide any help to Tessier. Jacques Dautrey, who was Tessier's friend but who was also on Ginestet's service, used to sneak in to help Tessier.

Between 1946 and 1950, Dr. Tessier made yearly visits to England to observe Harold Gillies, Archibald McIndoe, and others whose advances in plastic surgery in response to severe military injuries were ongoing. Through his observations and his passion to find solutions to complex reconstructive problems, Dr. Tessier began to find innovative ways to perform osteotomies for the correction of congenital midface retrusion but without the problems that had plagued the performance by his mentor Harold Gillies. By the mid 1950s, Tessier had become the head of his department in Paris. He was consulted in 1957 by a young man with a facial deformity of "monstrous aspect" who was found to have Crouzon syndrome.[186] The patient's facial deformities included severe orbital dystopia/eye proptosis, midface deficiency/malocclusion, and obstructed breathing. Dr. Tessier was aware that Gillies had completed an elective Le Fort III osteotomy on a young adult with Crouzon syndrome who had a moderate degree of proptosis and midface deficiency (Fig. 2-54). Dr. Gillies was reported to be discouraged with the eventual outcome and had recommended to others "never to do it."[82]

Tessier was undeterred, and he set out to solve the problem initially by performing an anatomic study of dry skulls. For access to the anatomy laboratory, he traveled by train in the evenings to his medical school in Nantes. After his late-night cadaver dissections, he returned during the early hours for the next Parisian working day. His painstaking preparatory work paid off when he successfully advanced his patient's face by 25 mm and achieved stability with the novel use of bone grafts (which Gillies had not used), improved osteotomy design, incision placement, and immobilization techniques, including use of an external fixation apparatus (Fig. 2-55).

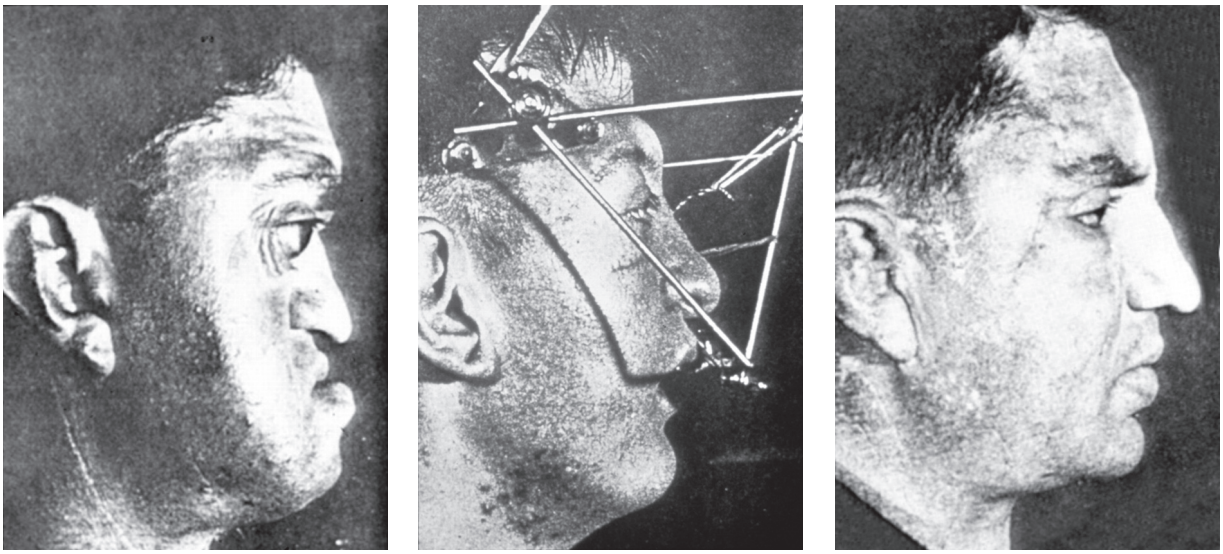
Dr. Tessier's next advance was to devise a technique for separating the orbits from the skull, thereby allowing for the relocation of the orbits and eyeballs for the protection of vision and improved aesthetics without damaging the brain or the eyes in the process. Working with Gerard Guiot, a neurosurgeon, Tessier took 3 more years to perfect his technique, which included the making of the bony cuts through the skull base by what soon became known as the "Tessier intracranial approach" to the orbits and the midface.

The surgical procedures developed by Tessier were shown to the world at a presentation that he made in Rome at the International Plastic Surgery Meeting in 1967.[187] The techniques were so revolutionary that Tessier sought to establish their validity before continuing further. In the most humble and unique way, Tessier invited prominent surgeons from around the world to observe him operating live so that he could demonstrate these techniques before a distinguished

• **Figure 2-54** Gillies published the results of a Le Fort III osteotomy that was completed in 1942 for an adult with Crouzon syndrome. *From Gillies H, Harrison SH: Operative correction by osteotomy of recessed malar maxillary compound in case of oxycephaly.* Br J Plast Surg *3:102, 1950.*

international group of his peers. He asked for their vote on whether his demonstrated surgical techniques had a place in the practice of surgery. The resounding positive responses of his peers were clear evidence of their support and approval. During the late 1960s and throughout the 1970s, Dr. Tessier developed all of the basic procedures and essential techniques that are currently used to perform what has come to be known as *craniofacial surgery,* including the transcranial and subcranial correction of orbital dystopia such as orbital hypertelorism; the correction of midface deficiency or deformity among patients with conditions such as Crouzon syndrome and Apert syndrome; the correction of facial deformities associated with such conditions as Treacher Collins–Franceschetti syndrome; and the description and correction of the spectrum of oro-ocular clefts. The impact of Dr. Tessier's work has affected every surgical specialty that seeks solutions to head and neck problems, including plastic surgery, otolaryngology/head and neck surgery, ophthalmology, neurosurgery, trauma surgery, and oral and maxillofacial surgery.

By the late 1960s, Paris become the birthplace of the new specialty of craniofacial surgery, where visitors would flock to observe Tessier's techniques and approach to problem



• **Figure 2-55** Tessier reported early results with an intracranial Le Fort III osteotomy.

solving. Dr. Tessier began traveling to the United States and England to demonstrate his procedures in cities such as Philadelphia, Chicago, Kansas City, Houston, Boston, Norfolk, and London.[185] Dr. Tessier's work became so far reaching that, by the 1990s, virtually everyone performing craniofacial procedures was either trained by Dr. Tessier himself or by surgeons whom he had trained.

Dr. Tessier's contributions cannot be overestimated.[184-196] His basic innovations to safely separate the orbits and the midface from the base of the skull with the use of an intracranial approach satisfy the needs for exposure during complex surgery and craniofacial reconstruction. His techniques have revolutionized the treatment of birth defects of the head and neck; tumors of the midface, orbit, and skull base; severe facial deformities and asymmetries; craniofacial trauma; and complex infections. In response to questions about what inspired his unique approach to problem solving, Tessier would quote Guiot's response: "Pourquoi pas?" This phrase—which is translated into English as "Why not?"—became the motto of the International Society of Craniofacial Surgery, founded in 1983, of which Tessier was invited to be Honorary President.

Dr. Tessier understood the importance of interspecialty collaboration, because "no one man could master all techniques and be an island onto himself" (personal communication). The recognition that individuals with complex surgical problems and deformities of the head and neck often require a team approach did not deter him. Tessier's painstaking preparatory work to achieve the best results for each and every patient and his insistence on the highest level of collaborative care was the mark of the man. He did not settle for mediocrity, and he expected the same commitment from others. This was true in all aspects of his professional and personal life. Hard work, determination, intelligence, focus, innovation, meticulous organization and planning, and—above all—imagination were the characteristics that Tessier demonstrated in his everyday life and that he appreciated in others.

Conclusions

Modern practicing orthodontists and maxillofacial surgeons have learned much from the pioneers in this field. We stand on the shoulders of the giants who came before us. Thanks to them, when evaluating an individual with a dentofacial deformity, we are now able to safely and reliably offer effective treatment to achieve long-term occlusal stability, periodontal health, an improved upper airway, and enhanced facial aesthetics in most cases. Even so, further innovations are expected from those who will come next.

References

1. Adams WM: Internal wiring fixation of facial fractures. *Surgery* 12:4, 1942.
2. Andrews LF: The six keys to normal occlusion. *Am J Orthod* 63:296–309, 1972.
3. Andrews WA: Andrews. *Journal of Orthodontics and Orofacial Harmony* 1(1):1–56, 2000.
4. Andrews WA: Andrews. *Journal of Orthodontics and Orofacial Harmony* 1(2):1–60, 2001.
5. Angle EH: Double resection of the lower maxilla. *Dent Cosmos* 40(July-Dec): 1898.
6. Angle EH: *Treatment of malocclusion of the teeth and fractures of the mandible: Angle's system*. Philadelphia, 1900, White Dental Manufacturing.
7. Angle EH: Double resection for the treatment of mandibular protrusion. *Dent Cosmos* 45:268, 1903.
8. Angle JL: Greek teeth: ancient and modern. *Hum Biol* 16:283–297, 1944.

9. Asbell MB: Bicentenary of a dental classic: John Hunter's natural history of the human teeth. *J Am Dent Assoc* 84:1311–1334, 1972.
10. Asbell MB: A brief history of orthodontics. *Am J Orthod Dentofacial Orthop* 98: 176–183, 1990.
11. Asbell MB: Simeon Hayden Guilford (1841-1919). *Am J Orthod Dentofacial Orthop* 113:367, 1998.
12. Asbell MB: John Nutting Farrar 1839-1913. *Am J Orthod Dentofacial Orthop* 114:602, 1998.
13. Aufricht G: Combined nasal plastic and chin plastic: correction of microgenia by osteocartilaginous transplant from large hump of nose. *Am J Surg* 25:292, 1934.
14. Aufricht G: Combined plastic surgery of the nose and chin. *Am J Oral Surg* 95:231, 1958.
15. Axhausen G: Zur Behandlung veralteter disloziert geheilter oberkieferbruche. *Dtsch Zahn Mund Kieferheilkd* 1:334, 1934.
16. Axhausen G: Ueber die korrigierende osteotomie am oberkiefer. *Dtsch Z Chir* 248:515–522, 1936.
17. Axhausen G: Die operative orthopädie bei den fehlbildungen der kiefer. *Dtsch Zahn Mund Kieferhlkd* 6:582–600, 1939.
18. Aziz SR, Simon P: Hullihen and the origin of orthognathic surgery. *Oral Maxillofac Surg* 62:1303–1307, 2004.
19. Babcock WW: Surgical treatment of certain deformities of the jaw associated with malocclusion of the teeth. *JAMA* 53:833, 1909.
20. Baker CR: Orthodontic profiles. *Am J Orthod* 43:210–218, 1957.
21. Barton JR: Systemic bandage for fracture of the lower jaw. *Am Med Rec* 2:153, 1819.
22. Barrow GV, Dingman RO: Orthodontic considerations in the surgical management of developmental deformities of the mandible. *Am J Orthod* 36:121–124, 1950.
23. Begg PR: Stone age man's dentition. *Am J Orthod* 40:298–312, 373–383, 462–475, 517–531, 1954.
24. Bell WH: Revascularization and bone healing after anterior maxillary osteotomy: a study using adult rhesus monkeys. *J Oral Surg* 27:249–255, 1969.
25. Bell WH, Levy BM: Revascularization and bone healing after anterior mandibular osteotomy. *J Oral Surg* 28:196–203, 1970.
26. Bell WH, Levy BM: Revascularization and bone healing after posterior maxillary osteotomy. *J Oral Surg* 29:313–320, 1971.
27. Bell WH: Correction of skeletal type of anterior open bite. *J Oral Surg* 29:706, 1971.
28. Bell WH, Levy BM: Revascularization and bone healing after maxillary corticotomies. *J Oral Surg* 30:640–648, 1972.
29. Bell WH, Fonseca RJ, Kenneky JW, Levy BM: Bone healing and revascularization after total maxillary osteotomy. *J Oral Surg* 33:253–260, 1975.
30. Bell WH: Le Fort I osteotomy for correction of maxillary deformities. *Oral Surg* 33:412–426, 1975.
31. Bell WH, Kennedy JW: Biologic basis for vertical ramus osteotomies—a study of bone healing and revascularization in adult rhesus monkeys. *J Oral Surg* 34:215–225, 1976.
32. Bell WH, Schendel SA: Biologic basis for modification of the sagittal ramus split operation. *J Oral Surg* 35:362–369, 1977.
33. Bell WH, Schendel SA, Finn RA: Revascularization after surgical repositioning of one-tooth dento-osseous segments. *J Oral Surg* 36:757–765, 1978.
34. Bell WH, Mannai C, Luhr HG: Art and science of the Le Fort I downfracture. *Int J Adult Orthod Orthognath Surg* 3:23, 1988.
35. Bell WH, You ZH, Finn RA, Fields RT: Wound healing after multisegmental Le Fort I osteotomy and transection of the descending palatine vessels. *J Oral Maxillofac Surg* 53:1425–1433, 1995.
36. Biederman W: The strange story of the Angle operation. *Ann Dent* 15:115, 1956.
37. Bier A, Braun A, Kummel H: *Chirurgische operationslehre. Band I*. Leipzig, Germany, 1914, Barth.
38. Blair VP: Report of a case of double resection for the correction of protrusion of the mandible. *Dent Cosmos* 48:817–820, 1906.
39. Blair VP: Operations on the jaw-bone and face. *Surg Gynecol Obstet* 4:67–78, 1907.
40. Blair VP: *Surgery of the mouth and jaws*. St. Louis, 1914, Mosby.
41. Blair VP: Instances of operative correction of malrelation of the jaws. *Int J Orthod* 1:395, 1915.
42. Brodie AG: Orthodontic history and what it teaches. *Angle Orthod* 4:85–97, 1934.
43. Brodie AG: Orthodontic concepts prior to the death of Edward H Angle. *Angle Orthod* 26:144–154, 1956.
44. Cadenat H, Aussenac R, Barthélémy I, Combelles R: Le maxillaire supérieur, facteur de prognathie inférieur. *Rev Stomatol Chir Maxillofac* 72:527–530, 1971.
45. Caldwell JB, Letterman GS: Vertical osteotomy in the mandibular rami for correction of prognathism. *J Oral Surg* 12:185, 1954.
46. Caldwell JB, Hayward J, Lister R: Correction of mandibular retrognathia by vertical L-osteotomy: a new technique. *J Oral Surg* 26:259, 1968.
47. Case CS: Principles of occlusion and dentofacial relations. *Dent Items Int* 27:489, 1905.
48. Case CS: *A practical treatise on the techniques and principles of dental orthopedia and prosthetic correction of cleft palate*, ed 2, Chicago, 1922.
49. Casto FM: A historical sketch of orthodontia. *Dent Cosmos* 76:111–135, 1934.
50. Cernea, Grignon, Crépy, Benoist: Les osteotomies totales de maxillaire superieurs. *Rev Stomatol* 56:700–716, 1955.
51. Chadwick J, Mann W: *The medical works of Hippocrates*. London, 1950, Blackwell Scientific, pp 260–265.
52. Champy M, Lodde JP, Wilk A, Grasset D: Plattenosteosynthesen bei mittelgesichtsfrakturen und osteotomien. *Dtsch Z Mund-Kiefer-u Gesichts-Chir* 2:26–36, 1978.
53. Champy M: Surgical treatment of midface deformities. *Head Neck Surg* 2:451–465, 1980.
54. Chapman H: Orthodontics: Fifty years in retrospect. *Am J Orthod* 41:421–442, 1955.
55. Cheever DW: Displacement of the upper jaw. *Med Surg Rep Boston City Hosp* 1:156, 1870.
56. Conn-Stock G: Die chirurgische immediate—gulierung der kiefer, speziell die chirurgische behandlung der prognathie. *Vjscher Zahnhkd* 37:320, 1921.
57. Converse JM, Shapiro H: Treatment of developmental deformations of the jaws. *Plast Reconstr Surg* 10:473–510, 1952.
58. Converse JM, Wood-Smith D: Horizontal osteotomy of the mandible. *Plast Reconstr Surg* 34:464, 1964.
59. Converse JM, Horwitz SL: The surgical orthodontic approach to the treatment of dentofacial deformities. *Am J Orthod* 55:217–243, 1969.
60. Cryer M: Typical and atypical occlusion of teeth. *Dent Cosmos* 46:713, 1904.
61. Cupar I: Die chirurgische behandlung der form und stellungs veranderungen des oberkiefers. *Ost Z Stomatol* 51:565, 1954.
62. Dal Pont G: L'osteotomia retromolare per la correzione della progenia. *Minerva Chir* 1:369–371, 1958.
63. Dal Pont G: L'osteotomia retromolare per la correzione della progenia. *Minerva Chir* 14:1138–1141, 1959.
64. Dal Pont G: Retromolar osteotomy for the correction of prognathism. *J Oral Surg Anaesth Hosp D Serv* 19:42–47, 1961.
65. Delaire J: De l'interet des osteotomies sagittales dans la correction des infragnathies mandibulaires. *Ann Chir Plast* 15:104, 1970.
66. Delaire J, Verdon P, Lumineau J, et al: Quelques resultats des tractions extra-orales B appui fronto-mentonnier dans le traitement orthopedique des malformations maxillomandibulaires de classe III et des sequelles osseuses des fentes labio-maxillaires. *Rev Stomatol (Paris)* 73:633, 1972.
67. Delaire J: Sagittal splitting of the body of the mandible (Mehnert's technique) for correction of open bite and deep over bite. *J Maxillofac Surg* 5:93, 1977.
68. Deranian HM: The miracle man of the Western Front; the story of Dr. Varaztad Kazanjian. *Bull Hist Dent* 32:85–95, 1984.
69. Dewel BF: The Case-Dewey-Cryer extraction debate. *Am J Orthod* 50:862–865, 1964.
70. Dewel BF: The ribbon arch: its influence in the development of orthodontic appliances. *Angle Orthod* 51:263–268, 1981.
71. Dingman RO: Surgical correction of mandibular prognathism: an improved

method. *Am J Orthod Dentofacial Orthop* 30:683, 1944.
72. Dingman RO, Harding RL: The treatment of malunited fractures of the facial bones. *Plast Reconstr Surg* 7:505–519, 1951.
73. Drommer R, Luhr HG: The stabilization of osteotomized maxillary segments with Luhr miniplates in secondary cleft surgery. *J Maxillofac Surg* 9:166–169, 1981.
74. Drommer R: The history of the "Le Fort I osteotomy,". *J Maxillofac Surg* 14:119–122, 1986.
75. Dufourmentel L: Le traitement chirurgical du prognathisme. *Presse Med* 29:235, 1921.
76. Egyedi P, Obwegeser H: Zur operativen zungenwerk-leinerung. *Dtsch Zahn Mund Kieferheilkd* 41:16, 1964.
77. Epker BN: A modified anterior maxillary osteotomy. *J Maxillofac Surg* 5:35, 1977.
78. Epker B: Modifications in the sagittal osteotomy of the mandible. *J Oral Surg* 35:157–159, 1977.
79. Ernst F: Uber die chirurgische beseitigung der prognathie des unterkiefers (progenie). *Zentralbl Chir* 65:179, 1938.
80. Gadd P: Cited in Limber. *J Am Dental Assoc* 15:851, 1928.
81. Gillies HD: *Plastic surgery of the face*. London, 1920, Oxford University Press.
82. Gillies HD, Harrison SH: Operative correction by osteotomy of recessed malar maxillary compound in case of oxycephaly. *Br J Plast Surg* 3:123, 1950.
83. Gillies HD, Kristensen HK: Ox cartilage in plastic surgery. *Br J Plast Surg* 4:63, 1951.
84. Gillies HD, Rowe NL: L'ostéotomie du maxillaire supérieur enoisagée essentiellement dans le cas de bec-de-lièvre total. *Rev Stomatol* 55:545–552, 1954.
85. Gillies HD, Millard DR, Jr: *The principles and art of plastic surgery*. Boston, 1957, Little, Brown.
86. Gilmer TL: Fractures of the inferior maxilla. *Ohio St J Dent Sci* 2:14, 57, 112, 1882.
87. Gunning TB: The treatment of lower jaw by interdental splints. *NY Med J* 3:433, 1866.
88. Halvorson EG, Mulliken JB: Cheever's double operation: the first Le Fort I osteotomy. *Plast Reconst Surg* 121: 1375–1381, 2008.
89. Harsha, WM: Bilateral resection of the jaw for prognathism. *Surg Gynecol Obstet* 15:51, 1912.
90. Hausamen JE: The scientific development of maxillofacial surgery in the 20th century and an outlook into the future. *J Craniomaxillofac Surg* 29:2, 2001.
91. Herbert JM, Kent JN, Hinds EC: Correction of prognathism by an intraoral vertical subcondylar osteotomy. *J Oral Surg* 28:651, 1970.
92. Hinds EC: Correction of prognathism by subcondylar osteotomy. *J Oral Surg* 16:209, 1958.
93. Hinds EC, Girotti WJ: Vertical subcondylar osteotomy: a reappraisal. *Oral Surg* 24:164, 1967.
94. Hofer O: Die operative behandlung der alveolare retraktion des unterkiefers und ihre anwendungsmoglichkeit fur prognathie und mikrogenie. *Dtsch Zahn Mund Kieferheilkd* 9:121–132, 1942.
95. Hofer O: Die osteoplastiche verlagerung des unterkiefers nach von eiselberg bei mikrogenie. *Dtsch Zahn Mund Kieferheilkd* 27:81, 1957.
96. Hogemann KE: Surgical orthopaedic correction of mandibular protrusion. *Acta Chir Scand Suppl* 159:26–28, 1951.
97. Hörster W: Experience with functionally stable plate osteosynthesis after forward displacement of the upper jaw. *J Maxillofac Surg* 8:176–181, 1980.
98. Hullihen S: Case of elongation of the under jaw and distortion of the face and neck, caused by a burn, successfully treated. *Am J Dent Sci* 9:157, 1849.
99. Hunsuck EE: A modified intraoral sagittal splitting technique for correction of mandibular prognathism. *J Oral Surg* 26:250–253, 1968.
100. Jaboulay M, Berard L: Traitemente chirurgical du prognathisme inferieur. *Presse Med* 1898.
101. Jones BM: Paul Louis Tessier. *J Plast Reconstr Surg* 122:1297–1299, 2008.
102. Joseph J: Verbesserung meiner hangewangenplastik (melomioplastik). *Dtsch Med Wochenschr* 54:567, 1928.
103. Kazanjian VH: Surgical correction of deformities of the jaws and its relation to orthodontia. *Am J Orthod Oral Surg* 22:259, 1936.
104. Kazanjian VH: Jaw reconstruction. *Am J Surg* 3:249, 1939.
105. Kazanjian VH: The interrelation of dentistry and surgery in the treatment of deformities of the face and jaws. *Am J Orthod Oral Surg* 27:11, 1941.
106. Kazanjian VH, Converse EJ: *The surgical treatment of facial injuries*. Baltimore, 1949, The Williams and Wilkins Company.
107. Kazanjian VH: The treatment of mandibular prognathism with special reference to edentulous patients. *J Oral Surg Oral Med Oral Pathol* 4:680–688, 1951.
108. Kazanjian VH: Remembrance of things past. *Plast Reconst Surg* 35:(1)5–13, 1965.
109. Kingsley NW: *A treatise on oral deformities (as a branch of mechanical surgery)*. New York, 1880, Appleton & Co.
110. Kirchner L: Die bedeutung der gesichtsproportionen für die wiederherstellungschirurgie. *Fortschr Kiefer Gesichtschir* 7:5–14, 1961.
111. Köle H: Corticalisschwachung zur unterstutzung bei der kieferorthopadischen behandlung. *Fortschr Kiefer Gesichtschir* 4:208, 1958.
112. Köle H: Surgical operations on the alveolar ridge to correct occlusal abnormalities. *Oral Surg Oral Med Oral Pathol* 12:515–529, 1959.
113. Köle H: Results, experience and problems in the operative treatment of anomalies with reserve overbite. *Oral Surg* 18:427, 1965.
114. Köle H: Zur behandlung der luxationsfracturen des unterkiefers. *Zahnärztl Rundsch* 64:419–425, 1995.
115. Kostecka F: Die chirurgische therapie der progenie. *Zahnärztl Rundsch* 16:670–688, 1931.
116. Kufner J: Four-year experience with major maxillary osteotomy for retrusion. *J Oral Surg* 29:549–553, 1971.
117. Lane WA: *Cleft palate and hare lip*. London, 1905, London Medical Publishing Co Ltd.
118. Langenbeck BV: Beitrage zur osteoplastik—die osteoplastische resektion de oberkiefers. In Goschen A, editor: *Deutsche klinik*, Berlin, 1859, Reimer.
119. Le Fort R: Etude experimentale sur les fractures de la machoire superieure. *Rev Chir Paris* 23:214, 1901.
120. Le Fort R: Experimental study of fracture of the upper jaw [reprint]. *Plast Reconstr Surg* 50:497, 1972.
121. Levignac J: Traitment d'un enforcement consolide du tiers moyen de la face par osteotomies et greffes. *Rev Stomatol* 50:551–556, 1958.
122. Limberg AA: Treatment of open bite by means of plastic oblique osteotomy of the ascending rami of the mandible. *Dent Cosmos* 67:1191, 1925.
123. Limberg AA: New method of plastic lengthening of the mandible in unilateral micrognathism and asymmetry of the face. *J Am Dent Assoc* 15:851, 1928.
124. Lindorf HH: Functionally stable tandem screw fixation in sagittal ramus osteotomy: surgical technique. *Dtsch Z Mund Kiefer Gesichtschir* 8:367–373, 1984.
125. Lindorf HH: Sagittal ramus osteotomy with tandem screw fixation: technique and results. *J Maxillofac Surg* 14:311, 1986.
126. Loh FC: Surgical refinement of the anterior maxillary subapical osteotomy. *J Oral Maxillofac Surg* 51:332–334, 1993.
127. Luhr HG: Zur stabilen osteosynthese bei unterkiefer-frakturen. *Dtsch Zahnarztl Z* 23:754, 1968.
128. Luhr HG: Die operative behandlung von unterkieferfrakturen besonderer berucksichtigung der kompressionosteosynthese. *Zentralbl Chir* 94:819, 1969.
129. Luhr HG: The compression osteosynthesis of mandibular fracture in dogs. A histologic contribution to "primary bone healing." *Eur Surg Res* 1:3, 1969.
130. Luhr HG: *Die kompressionsosteosynthese bei unterkieferfrakturen*. Munich, 1972, Hanser.
131. Luhr HG, Busch W, Maerker R: Histologische untersuchungen zur frage de primaren knochenheilung nach kompressionosteosynthese. *Fortschr Kiefer Gesichtschir* 19:13, 1975.
132. Luhr HG: Skelettverlagernde operationen zur harmonisierung des gesichtsprofils—probleme der stabilen fixation von osteotomiesegmenten. In von Pfeifer G, editor: *Die esthetik von form und funktion in*

der plastischen und wiederherstellungs—chirurgie, Berlin, 1985, Springer-Verlag.
133. Luhr HG: A micro-system for craniomaxillofacial surgery. *J Cranio Maxillofac Surg* 16:312, 1988.
134. Luhr HG: In *Fortschritte der kiefer und gesichts-chirurgie*. Stuttgart, 1994, Thieme Verlag, pp 1–13.
135. Matasa CG, Graber TM: Angle, the innovator, mechanical genius, and clinician. *Am J Orthod Dentofacial Orthop* 117: 444–452, 2000.
136. Max D, Rotskoff K: A modified technique for the sagittal split osteotomy. *J Oral Maxillofac Surg* 51:1050, 1993.
137. McKinstry RE: *Cleft palate dental care: a historical perspective*. Arlington, Va, 2000, ABI Publications.
138. Mehnert H: Progenieoperation durch sagittalstufenformige osteotomie in horizontalen ast bei bezahntem unterkiefer. *Dtsch Zahn Mund Kieferheilkd* 4:88, 1967.
139. Michelet FX, Deymes J, Dessus B: Osteosynthesis with miniaturized screws and plates in maxillofacial surgery. *J Maxillofac Surg* 1:79, 1973.
140. Millard DR: Chin implants. *Plast Reconstr Surg* 13:70, 1954.
141. Mohnac AM: Maxillary osteotomy in the management of occlusal deformities. *J Oral Surg* 24:305, 1966.
142. Moos KF: Origins of orthognathic surgery. *Dent Hist* 37:5–18, 2000.
143. Moose SM: Correction of abnormal mandibular protrusion by intraoral operation. *J Oral Surg* 3:304–310,1945.
144. Muchnic HV: Spencer Roane Atkinson (1886-1970). *Am J Orthod* 77:580–582, 1980.
145. Nance HN: The limitations of orthodontic treatment. *Am J Orthod* 33:177–223, 1947.
146. Niederdellmann H, Schilli W: Zur platen osteosynthese bei unterkieferfrakturen. *Dtsch Zahnarztl Z* 28:638, 1973.
147. Niederdellmann H, Schilli W, Ewers R, et al: Photoelastic behavior of osteosynthesis plates with different arrangements of screw holes for mandibular fractures. *Int J Oral Surg* 4:27, 1975.
148. Niederdellmann H, Shetty V: Solitary lag screw osteosynthesis in the treatment of fractures of the angle of the mandible: a retrospective study. *Plast Reconstr Surg* 80:68, 1987.
149. Obwegeser HL: In Trauner R, Obwegeser H, editors: Zur operationstechnik bei der progenie und anderen unterkieferanomalien. *Dtsch Zahn- Mund- u Kieferheilk* 23:1–26, 1955.
150. Obwegeser HL: The surgical correction of mandibular prognathism and retrognathia with consideration of genioplasty. Part 1. *Oral Surg* 10:677, 1957.
151. Obwegeser HL. The indications for surgical correction of mandibular deformity by the sagittal splitting technique. *Br J Oral Surg* 1:157–171, 1964.
152. Obwegeser HL: Der offene biss in chirurgischer sicht. *Schweiz Monatschr Zahnhlkd* 74:668–687, 1964.
153. Obwegeser HL: Surgical correction of small or retrodisplaced maxillae. *Plast Reconstr Surg* 53:351, 1969.
154. Obwegeser HL: Zur korrektur der dysostosis otomandibularis. *Schweiz Monatsschr Zahnheilkd* 80:331, 1970.
155. Obwegeser HL: Kieferchirurgische aufgaben im bereich des mittelgesichtes. *Arch Otorhinolaryngol* 207:229, 1974.
156. Obwegeser HL: Orthognathic surgery and a tale of how three procedures came to be: a letter to the next generations of surgeons. *Clin Plast Surg* 34:331–355, 2007.
157. Paladin P, editor: *Orthodontic history: braced for a century*, St. Louis, 2005, American Association of Orthodontics.
158. Perko M: Maxillary sinus and surgical movement of maxilla. *Int J Oral Surg* 1:177, 1972.
159. Perko M: The history of treatment of cleft lip and palate. *Prog Pediatr Surg* 20:239–248, 1986.
160. Perthes G: *Ueber frakturen und luxationsfrakturen des kieferkopfchens und ihre operative behandlung. Verh Dtsch Gesell Chir*. Berlin, 1924, Springer, pp 418–433.
161. Peterson HW: Some observations on the progress of orthodontics during the past fifty years. *Am J Orthod* 39:289–290, 1953.
162. Proffit WR, White RP, Sarver DM: *Contemporary treatment of dentofacial deformity*. St. Louis, 2003, Mosby.
163. Proffit WR, Fields HW, Sarver DM, editors: *Contemporary orthodontics*, ed 4, St. Louis, 2007, Mosby/Elsevier.
164. Proffit WR, White RP, Jr: Development of surgeon-orthodontist interaction in orthognathic surgery. *Semin Orthod* 17:183–185, 2011.
165. Ricketts RM: Keystone triad: part 2. *Am J Orthod* 50:728–750, 1964.
166. Ring ME: A thousand years of dental implants: a definitive history, part 1. *Compend Contin Educ Dent* 16:1060–1069, 1995.
167. Robinson M: Prognathism corrected by vertical subcondylotomy. *J Oral Surg* 16:215, 1958.
168. Rosen HM: Surgical correction of the vertically deficient chin. *Plast Reconstr Surg* 82:247, 1988.
169. Rowe NL, Killey HC: *Fractures of the facial skeleton*, ed 2, Edinburgh and London, 1968, Livingstone Ltd.
170. Schmid E: Zue wiederherstellung des mittelgeisichtes nach entwicklungsstorungen und defekten der knochernen unterbaues. *Fortschr Kiefer Gesichtschir* II:240–243, 1956.
171. Schuchardt K: Ein beitrag zur chirurgischen kieferorthopadie unter berucksichtigung ihrer bedentung fur die behandlung angeborener und erworbener kieferdeformitaten bei soldaten. *Dtsch Zahn Mund Kieferhlkd* 9:73, 1942.
172. Schuchardt K: Die chirurgie als helferin der kieferorthopadie. *Fortschr Kieferorthop* 15:9, 1954.
173. Schuchardt K: Formen des offenen bisses und ihre operativen behandlungsmoglechkeiten. In Schuchardt K, editor: *Fortschritt in kiefer und gesichtschirurgie*, Stuttgart, Germany, 1955, Thieme.
174. Shankland WM: *The American Association of Orthodontists: the biography of a specialty organization*. St. Louis, 1971, The American Association of Orthodontists.
175. Simon P: *Fundamental principles of systematic diagnosis of dental anomalies*. Boston, 1926, Stratford Co.
176. Spanier F: Prognathie-operationen. *Z Zahnarztl Orthop Munchen* 24:76, 1932.
177. Spiessl B: Osteosynthese bei sagittaler osteotomie nach Obwegeser/Dal Pont. *Fortschr Kiefer Gesichts Chir* 18:145, 1974.
178. Spiessl B: The sagittal splitting osteotomy for correction of mandibular prognathism. *Clin Plast Surg* 9:496, 1982.
179. Steinhauser EW: Bone screws and plates in orthognathic surgery. *Int J Oral Surg* 11:209, 1982.
180. Stoker NG, Epker BN: The posterior maxillary osteotomy: a retrospective study of treatment results. *Int J Oral Surg* 3:153, 1974.
181. Strang RH: Highlights of sixty-four years in orthodontics. *Angle Orthod* 44:101–112, 1974.
182. Storum KA, Bell WH, Nagura H: Microangiographic and histologic evaluation of revascularization and healing after genioplasty by osteotomy of the inferior border of the mandible. *J Oral Maxillofac Surg* 48:210–216, 1988
183. Stubtelny JD, Sakuda M: Open bite: diagnosis and treatment. *Am J Orthod Dentofacial Orthop* 50:337, 1964.
184. Tessier P, Guiot G, Rougerie J, et al: [Cranio-naso-orbito-facial osteotomies. Hypertelorism]. *Ann Chir Plast* 12:103–118, 1967.
185. Tessier P: Demonstration operations at Hopital Foch. *Paris (Suresnes)* 1967.
186. Tessier P: Osteotomies totales de la face: syndrome de Crouzon, syndrome d'Apert: oxycephalies, scaphocephalies, turricephalies. *Ann Chir Plast* 12:273–286, 1967.
187. Tessier P: *Dysostosis cranio-faciales (syndromes de Crouzon et d'Apert) osteotomies totales de la face. Transactions of the Fourth International Congress of Plastic and Reconstructive Surgery*, Rome, 1967/Amsterdam, 1969, Excerpta Medica Foundation, pp 774–783.
188. Tessier P: Autogenous bone grafts taken from the calvarium for facial and cranial applications. *Plast Reconstr Surg* 48:224, 1971.
189. Tessier P: The definitive plastic surgical treatment of the severe facial deformities of craniofacial synostosis: Crouzon and Apert diseases. *Plast Reconstr Surg* 48:419, 1971.

190. Tessier P: Total osteotomy of the middle third of the face for faciostenosis or for sequelae of the Le Fort III fractures. *Plast Reconstr Surg* 48:533, 1971.
191. Tessier P: Traitement des dysmorphies faciales propres aux dysostoses craniofaciales (DGF), maladies de Crouzon et d'Apert. *Neurochirurgie* 17:295, 1971.
192. Tessier P: Recent improvement in the treatment of facial and cranial deformities in Crouzon disease and Apert syndrome. In *Symposium of plastic surgery of the orbital region*, St. Louis, 1976, CV Mosby Co, pp 271.
193. Tessier P: Relationship of craniosynostosis to craniofacial dysostosis and to faciosynostosis: a study with therapeutic implications. *Clin Plast Surg* 9:531, 1982.
194. Tessier P: Apert syndrome: acrocephalosyndactyly, type I. In Caronni P, editor: *Craniofacial surgery*, Boston, 1985, Little, Brown.
195. Tessier P: *Craniofacial surgery in syndromic craniosynostosis: craniosynostosis, diagnosis, evaluation and management*. New York, 1986, Raven Press, pp 321.
196. Tessier P: The monobloc and frontofacial advancement: do the pluses outweigh the minuses? [discussion]. *Plast Reconstr Surg* 91:988, 1993.
197. The Division of Plastic and Reconstructive Surgery, Department of Surgery, Washington University School of Medicine: *The legacy of Vilray Blair*. Available at http://surgerydept.wustl.edu/Surgery_M.aspx?id=2990&menu_id=284. Accessed December 27, 2012.
198. Trauner R, Obwegeser H: Zur operationstechnik bei der progenie und anderen unterkieferanomalien. *Deutsche Zahn-, Mund- und Kieferheilkunde* 23:1–26, 1955.
199. Trauner R, Obwegeser H: The surgical correction of mandibular prognathism and retrognathia with consideration of genioplasty. I. Surgical procedures to correct mandibular prognathism and reshaping of the chin. *Oral Surg Oral Med Oral Pathol* 10:677–689, 1957.
200. Trauner R, Obwegeser H: The surgical correction of mandibular prognathism and retrognathia with consideration of genioplasty. II. Operating methods for microgenia and distoocclusion. *Oral Surg Oral Med Oral Pathol* 10:787–792, 1957.
201. Von Eiselberg E: Cited in Limberg. *J Am Dental Assoc* 15:851, 1928.
202. Wahl N: Orthodontics in 3 millennia. Chapter 1: Antiquity to the mid-19th century. *Am J Orthod Dentofacial Orthop* 127:255–259, 2005.
203. Wahl N: Orthodontics in 3 millennia. Chapter 2: Entering the modern era. *Am J Orthod Dentofacial Orthop* 127:510–515, 2005.
204. Wahl N: Orthodontics in 3 millennia. Chapter 6: More early 20th-century appliances and the extraction controversy. *Am J Orthod Dentofacial Orthop* 128:795–800, 2005.
205. Wassmund M: *Frakturen und luxationen des gesichtsschadels*. Berlin, 1927.
206. Wassmund M: *Die totale mobilisation des oberkiefers. Lehrbuch der praktischen chirurgie des mundes und der kiefer*. Leipzig, 1935, Meusser, pp 282–284.
207. Wassmund M: *Die korrektur der mikrognathie. Lehrbuch der praktischen chirurgie des mundes und der kiefer*. Leipzig, 1939, Barth, pp 493–494.
208. Weinberger BW: *Orthodontics: a historical review of its origin and evolution, including an extensive bibliography of orthodontic literature up to the time of specialization*. St. Louis, 1926, The CV Mosby Company.
209. Weinberger BW: Historical resume of the evolution and growth of orthodontia. *J Am Dent Assoc* 21:2001–2021, 1934.
210. Weinberger BW: Dr Edward Hartley Angle: his influence on orthodontics. *Am J Orthod* 36:596–607, 1950.
211. Weinstein I: C-osteotomy for correction of mandibular retrognathia: report of cases. *J Oral Surg* 29:358, 1971.
212. Whipple JW: Double resection of the inferior maxilla for protruding lower jaw. *Dent Cosmos* 40:552–557, 1898.
213. Wikkeling OME, Tacoma J: Osteotomy of the pterygomaxillary junction. *Int J Oral Surg* 4:99, 1975.
214. Wolfe SA: Gunther Cohn-Stock, M.S., D.D.S., father of maxillary orthognathic surgery. *J Cranio Maxillofac Surg* 17: 331–334, 1989.
215. Wolfe SA: Paul Tessier, creator of a new surgical specialty, is recipient of Jacobson innovations award. *J Craniofacial Surg* 12(1):98–99, 2001.
216. Wolfe SA: Paul Louis Tessier, MD, 1917 to 2008. *J Plast Reconstr Surg* 122:1294–1296, 2008.
217. Worms FW, Meskin CH, Isaacson RJ: Open bite. *Am J Orthod Dentofacial Orthop* 59:589, 1971.
218. Wright CF: Status of orthodontic education prior to 1930. *Angle Orthod* 17:92–96, 1947.
219. Wunderer S: Die prognathie-operation mittels frontal gestieltem maxillofragment. *Oesterr Z Stomatol* 59:98–102, 1962.
220. Wunderer S: Erfahrungen mit der operativen behandlung hochgradiger prognathien. *Dtsch Zahn Mund Kieferheilkd* 39:451, 1963.

3

Definition and Prevalence of Dentofacial Deformities

JEFFREY C. POSNICK, DMD, MD

- Prevalence of Jaw Deformities and Malocclusion
- Orthognathic Procedures Performed on Hospitalized Patients in the United States
- Conclusions

The term *dentofacial deformity* refers to significant deviations from normal proportions of the maxillomandibular complex that also negatively affect the relationship of the teeth within each arch and the relationship of the arches with one another (occlusion).[2,4,5,8,11,14,15,17,18,21,22,25,27,31,36,37,40,52,54] The affected individual will have varied degrees of compromise in head and neck functions related to breathing, swallowing, speech articulation, chewing, and lip closure/posture. Effects on the temporomandibular joints, the periodontium, and the teeth themselves may also occur.[24,25,28,33,34,38,55] The presenting facial disproportion will, in general, have at least some negative effects on psychosocial health.[16,39,56,57]

Racial variations with regard to the incidence of facial dysmorphology and the resulting malocclusion are also known to occur.[59,64] Definitions of acceptable levels of deviation from normal continue to be questioned by both clinicians and patients.[7,26,30,32,35,58,61,62] Over the years, the National Center for Health Statistics has collected data and the Research Council has held multidisciplinary conferences to focus attention on these issues.[6,12,19,20,29,41-51,53,63]

Surgery to reposition the jaws (i.e., an orthognathic procedure) as part of an interdisciplinary approach is often recommended to manage the related skeletal, dental, and soft-tissue dysfunctions and concerns.[3,13,23,60,66] Speech therapy, dental work, orthodontics, and surgical procedures alone are generally inadequate as isolated treatment modalities.

A dental clinician who is asked to evaluate the affected individual must ask himself or herself, "Are the problems too severe to be most effectively managed with orthodontics and dentistry alone?" A cosmetic surgeon who evaluates the same patient must ask himself or herself, "Will limited soft-tissue or augmentation procedures alone be sufficient to properly manage the presenting dysmorphology and to address functional aspects?"

If the discrepancy in the size or position of the jaws as they relate to each other and to the upper facial skeleton results in significant facial disproportion and also negatively affects certain head and neck functions (e.g., speech, swallowing, chewing, lip closure and posture, breathing), then it should not be ignored. It would be misguided to consider orthodontics alone to alter the occlusion without fully informing the patient and his or her family of the preferred biologic approach to management (i.e., orthodontics and jaw surgery). Likewise, it would be avoiding the obvious for the cosmetic surgeon to recommend either soft-tissue aesthetic or skeletal contour procedures without discussion of the presenting malocclusion, airway dysfunction, and overall facial disproportion. The treatment recommended to the patient and his or her family should not be limited by the skill set of the initial consulting clinician.

Facial disproportion observed in a child may at times be self-correcting. For example, apparent mandibular deficiency that is present before the pubertal growth spurt may normalize. In some cases, the maxilla or mandible may be induced to grow a few millimeters, more or less, through dentofacial orthopedics. However, major transformation of the jaws with the use of growth-modification techniques cannot be expected. Proffit has pointed out that, even with the well-intended aim of dentofacial orthopedics to alter jaw growth, as a result of anchorage requirements and biologic realities coupled with the practical desire of the orthodontist to "correct the occlusion," the treatment generally results in the displacement of the teeth in the direction of correcting the occlusion rather than the jaw relationships.[53] The term *dental compensation for the skeletal discrepancy* is universally understood to explain this treatment approach. Orthodontic-introduced dental compensation for the occlusion will hinder the eventual skeletal (orthognathic) correction if this is later required or requested.

Informed consent from the patient or his or her family is strongly recommended before embarking on a compromised treatment plan. For example, if a child is recognized to have an underdeveloped mandible with a Class II malocclusion and standard growth modification is attempted, it may be difficult for the orthodontist to prevent at least some retraction of the upper incisors and the forward displacement of the lower teeth. This may result in an "improved" occlusion, but it may also potentially involve long-term negative effects on periodontal health (e.g., labial cortical bone stripping), the airway (e.g., retroglossal obstruction), and facial aesthetics (e.g., a weak profile).[39,56,57] It also compromises the option of an orthognathic correction with the need to first "undo" the dental compensations through "redo" orthodontics (Fig. 3-1).

In the growing child who presents with a Class II malocclusion pattern, an active treatment approach is often offered by the orthodontist. This approach may attempt

• **Figure 3-1** A 21-year-old woman with a primary mandibular deficiency growth pattern requested a surgical consultation for a "weak chin." During her early teenage years, she underwent unsuccessful growth modification in an attempt to stimulate the forward projection of the mandible. This was followed by an orthodontic camouflage approach. The mandibular anterior dentition was flared forward. The history was significant for restless sleeping and a degree of daytime fatigue, which are suggestive of obstructive sleep apnea. Examination confirmed a retrognathic mandible with a Class II malocclusion. The mandibular incisors were crowded and procumbent. The family had hoped that a "chin implant" would be effective to manage the aesthetic effects. A sleep study confirmed obstructive sleep apnea (respiratory disturbance index = 18/hour). An orthognathic approach (Le Fort I, Sagittal splits, Osseous genioplasty) with redo orthodontic treatment including lower bicuspid extractions was recommended as the preferred method to improve the airway, to achieve long-term dental health, and to enhance facial aesthetics. **A,** Frontal facial and occlusal views. **B,** Profile facial view and lateral cephalometric radiograph.

to alter jaw growth and to correct the occlusion by means of the following: (1) functional appliance use (e.g., Frankel, Twinblocks) to stimulate sagittal growth of the mandible; (2) the possible extraction of maxillary premolars with orthodontic incisor retraction; (3) the use of headgear to restrain maxillary sagittal growth; and (4) the orthodontic forward displacement of the lower anterior teeth (Fig. 3-2). With the use of this approach, favorable facial results will be seen in only a very specific patient subgroup that includes those patients with true maxillary dental protrusion and a limited degree of mandibular retrusion. In these cases, the extraction of maxillary premolars with the retraction of the incisors to a corrected inclination in combination with the minimal forward displacement of the lower teeth may result in both favorable occlusion and acceptable facial aesthetics.

For the experienced surgeon and orthodontist, the accurate diagnosis of a dentofacial deformity will usually be clear after the initial examination and a review of standard records. A favorable correction through the effective

• **Figure 3-2** A 20-year-old man with a primary mandibular deficiency growth pattern requested a surgical consultation for a "weak chin." During his early teenage years, he underwent unsuccessful growth modification in an attempt to stimulate the forward projection of the mandible. This was followed by an orthodontic camouflage approach that included maxillary first bicuspid extractions to retract the anterior teeth. In addition, the mandibular anterior dentition was flared forward. His history was significant for heavy snoring, restless sleeping, and a degree of daytime fatigue, all of which are suggestive of obstructive sleep apnea. Examination confirmed a retrognathic mandible with a molar Class II deep bite malocclusion. The mandibular incisors were crowded and procumbent. The family had hoped that a "chin implant" would be effective to manage the aesthetic effects. A sleep study was recommended. An orthognathic approach (Le Fort I, Sagittal splits, osseous genioplasty) with redo orthodontic treatment was suggested as the preferred method to improve the airway, to achieve long-term dental health, and to enhance facial aesthetics. **A,** Frontal facial and occlusal views. **B,** Profile facial view and lateral cephalometric radiograph.

orthodontic alignment of the teeth in combination with the surgical repositioning of the jaw(s) will be the preferred approach.

Prevalence of Jaw Deformities and Malocclusion

U.S. Population Survey

As part of a large-scale evaluation of the health of the U.S. population, a National Health and Nutrition Examination Survey (NHANES III) was carried out between 1989 and 1994.[53] Starting with a sampling of 14,000 individuals, estimates of the incidence of malocclusion and its severity were made. The sample of individuals was carefully selected to provide weighted estimates for an approximate 150,000,000 people between the ages of 8 and 50 years who were members of black, white, and Latino American racial and ethnic groups. Those individuals outside of that age range (i.e., those younger than 8 years and older than 50 years), Native Americans, those living on military reservations, and some other specific population groups were excluded from this study. Data collected included the following:

- The alignment of the incisor teeth
- The horizontal position of the incisors (i.e., overjet or reverse overjet)
- The vertical overlap of the incisors (i.e., deep bite or open bite)
- The presence of posterior crossbite
- The presence of maxillary midline diastema

The Horizontal/Sagittal Dimension

This study provides useful information about preadolescent children (8 to 11 years old), adolescents (12 to 17 years old), and adults (18 to 50 years old) with reference to how the teeth fit together and, by inference, the prevalence of dentofacial deformities. When interpreting the data collected for the NHANES III study, it is important to consider that at least some degree of dental compensation for an existing jaw deformity normally occurs during growth and is expected to have been present at the time that the study measurements were taken. Therefore, it is unlikely that either the moderate or greater values of positive overjet or the mild to moderate values of negative overjet measured in the NHANES III study were found in individuals with "normal" jaw relationships. It would be safe to assume that any individual in the study with more than 7 mm of positive overjet has a jaw discrepancy that is characterized by mandibular deficiency (see Chapter 19). In addition, those with 2 mm or more of reverse overjet are assumed to have elements of maxillary deficiency in combination with relative mandibular excess (see Chapter 20).

The NHANES III study documents that extreme positive overjet is more frequent among children and that reverse overjet becomes more common among adults (i.e., after growth completion). This data confirms that, in some children, a late mandibular growth spurt not only corrects the excess positive overjet but actually causes it to become negative. Fortunately, in at least some of the children with an excess overjet, "catch-up" mandibular growth corrects the occlusion without overshooting the mark. The NHANES III study also documents that significant reverse overjet is more prevalent among blacks and Latino Americans as compared with whites. This confirms the more frequent dentofacial deformity growth pattern of maxillary deficiency in combination with relative mandibular excess that is seen among blacks and Latinos as compared with whites.

According to the NHANES III study, only a third of the members of the U.S. population have ideal (horizontal) anteroposterior incisor relationships, and another third of these individuals have a moderate overjet discrepancy (i.e., borderline jaw dysharmony). The remaining third have either a severe positive overjet or a reverse overjet malocclusion. It is reasonable to assume that a significant percentage of this subgroup (i.e., those with a severe positive overjet or a reverse overjet) have a jaw discrepancy that would benefit from orthognathic surgery. In reality, many individuals in this subgroup will be treated with compromised orthodontics (i.e., dental compensation) in an attempt to neutralize the occlusion without the benefit of an orthognathic correction. This will result in suboptimal facial aesthetics and the potential for occlusal instability, long-term periodontal sequela, and compromise of the upper airway.

The Vertical Dimension

According to the NHANES III study, only half of the U.S. population has an ideal vertical relationship of the incisors (i.e., 0- to 2-mm overbite). In the others, a deep bite in combination with mandibular deficiency or maxillomandibular deficiency is more prevalent among whites, and an open bite in combination with bimaxillary dental protrusion is more frequent among blacks. Interestingly, either a severe deep bite (≥5 mm) or a marked open bite (≥2 mm) was present in approximately 20% of children and 13% of adults. The extreme values of open bite that were measured in the study group likely represent either a long face growth pattern (i.e., vertical maxillary excess or mandibular deficiency; see Chapter 21) or bimaxillary dental protrusion (see Chapter 24). Interestingly, significant anterior open bite as part of a long face growth pattern is more prevalent among white Americans. The extreme values of deep bite in the studied individuals are likely to represent either a short face growth pattern (i.e., maxillomandibular deficiency; see Chapter 23) or a primary mandibular deficiency. Significant degrees of vertical discrepancy (deep bite or open bite) at the incisors ideally benefit from orthognathic surgery. When dental compensating orthodontic treatment is instituted rather than orthognathic correction, compromised facial aesthetics, occlusal instability, and periodontal sequela are more likely to occur.

U.K. Population Survey

An Index of Treatment Need was developed by the Swedish Dental Board to classify the severity of dental findings by the worst presenting characteristic.[33] This method tends to downplay the specific alignment of individual teeth. It is a classification system that looks more globally at facial proportions and head and neck function than at isolated dental details. For example, if you have a mild irregularity of the incisors and only a mildly excessive overjet, you are judged to have only a mild problem. In other words, the impact on the individual is not judged to be more severe, because there is a combination of two mild occlusal deviations from normal. Alternatively, if you have a 10-mm overjet, even if the teeth are aligned in each jaw, you are judged to have a severe problem.

Brook and Shaw made modifications to the Swedish classification and developed the Index of Treatment Need for malocclusion to be used as a grading system of dental health and functional indications for treatment.[9,10] The first part of their study was derived from the direct examination of occlusion and dental alignment. They defined five grades of treatment need, with Grade 1 representing little or no need for treatment and Grade 5 representing great need for treatment. The authors attempted to establish meaningful values for cutoff points between grades for each occlusal trait and to determine the quantifiable threat to the dentition if no treatment is provided. Grades 4 and 5 have characteristics that can result in a more severe impact on both facial aesthetic and dental aspects. These characteristics include the following:

Grade 5:
- Defects of cleft lip and palate*
- Overjet of more than 9 mm*
- Reverse overjet of more than 3.5 mm with reported masticatory and speech difficulties*
- Extensive hypodontia*
- Impeded eruption of the permanent teeth
- Submerged primary teeth

Grade 4:
- Extreme lateral or anterior open bites (>4 mm)*
- Increased overjet (>6 mm but ≤9 mm)*
- Reverse overjet (>3.5 mm with no masticatory or speech difficulties)*
- Reverse overjet of more than 1 mm with reported masticatory or speech difficulties
- Posterior crossbite with no functional occlusal contacts in one or both buccal segments
- Increased and complete overbite with gingival or palatal trauma
- Anterior or posterior crossbites with more than 2 mm of centric relation/centric occlusion discrepancy
- Severe contact point discrepancy (>4 mm)
- Less extensive hypodontia (e.g., one tooth per quadrant requiring preprosthetic orthodontics)
- Partially erupted teeth that are tipped and impacted against adjacent teeth
- The presence of supernumerary teeth

*Occlusal traits consistently found in association with a dentofacial deformity.

Individuals with extremes in positive overjet, reverse overjet, crossbite, and open bite as well as those with associated cleft lip and palate are considered to have Grade 4 and 5 conditions and judged to have problems that are severe enough that definitive treatment is needed. By inference, it is likely that a significant number of individuals with Grade 4 and 5 issues would ideally receive both orthodontics and jaw-straightening surgery.

The second part of the authors' overall assessment of treatment priority was to record the aesthetic impairment contributed by the malocclusion. The authors used the Standard Continuum of Aesthetic Need (SCAN) index as a rating scale.[16] The SCAN scale was constructed with the use of dental photographs from 1000 12-year-old children that were collected as part of a large multidisciplinary survey. Six non-dental judges rated these photographs on a 10-point visual analog scale. Both the aesthetic impairment component and the dental health component were part of the overall study.

The distribution of ratings for the Dental Health Indications Study conducted by Brook and Shaw were obtained from examination subjects (n = 222) who, at minimum, were felt to have a malocclusion to the extent that referral to a regional orthodontic center for advice or treatment was recommended.[10] Each study patient was then examined, and their available radiographs were reviewed by skilled clinicians. Both components of the index (i.e., aesthetic and dental) were applied, and the patients were also asked to give their own rating according to the SCAN scale. Interestingly, there was a high correlation (confirmed by intraexaminer and interexaminer error testing of the findings) between both components (i.e., aesthetic and dental) of the study.

The study results indicated that 19.2% of the subjects (44 of 222) were considered to have Grade 5 conditions. Interestingly, the distribution of ratings from Grades 1 through 5 that were obtained from the examination of a matched, random, unselected group of schoolchildren (n = 333) indicated that only 5.1% fell in the Grade 5 category. A Grade 5 score indicates severe malocclusion with a high need for treatment to establish dental health. Most of the traits listed as Grade 5 cannot be corrected with orthodontics alone, and orthognathic surgery would likely be recommended or at least considered. The Shaw study indicates that, in the United Kingdom, more than 5% and as high as 19% of children who have been referred to an orthodontist for evaluation are likely to have a malocclusion to the extent that orthodontics alone would not be the first choice for full correction (i.e., there is a need for orthognathic surgery).[10]

Orthognathic Procedures Performed on Hospitalized Patients in the United States

Venugoplan and colleagues completed a study with the aim of providing a nationally representative estimate of the number and type of orthognathic procedures performed on hospitalized patients in the United States.[65] Their data was obtained from the Nationwide Inpatient Sample (NIS) database from 2008 and included all hospitalizations for orthognathic procedures. The procedures were identified via the procedure code listed in the *International Classification of Diseases, 9th Revision, Clinical Modification.*[1] The NIS database is sponsored by the Agency for Healthcare Research and Quality, a division of the Department of Health and Human Services of the U.S. government. Ten thousand three hundred and forty-five (10,345) hospitalizations for orthognathic procedures were identified as occurring during the 2008 calendar year. The average age of these patients was 26.7 years, and female patients comprised 56.2% of all those who were hospitalized for these procedures. Whites, blacks, Hispanics, Asians, Pacific Islanders, and Native Americans and other races constituted 71.9%, 4.9%, 12.6%, 5.6%, 0.4%, and 4.6% of the hospitalizations, respectively. Private insurance plans were the primary payers (77.3%). Government insurance plans (i.e., Medicare and Medicaid) accounted for 13.4% of the patient mix, whereas 3.5% of patient paid privately (i.e., out of pocket), without the benefit of medical insurance.

Le Fort I osteotomy without segmentation accounted for 15.3% of the procedures; 45.8% of the procedures were Le Fort I with segmentation; and 31.7% of the procedures were ramus osteotomies of the mandible (e.g., sagittal split ramus osteotomies or vertical oblique osteotomies). It was found that 53.3% of the patients underwent one jaw surgery, whereas 36.8% likely underwent bimaxillary osteotomies. It would appear that only 9.2% of the patients underwent combined upper jaw, lower jaw and chin procedures. Only 0.7% underwent additional other simultaneous procedures, including such possibility as septoplasty, inferior turbinate reduction, neck liposuction, and the removal of wisdom teeth.

The pattern of jaw deformity for which the patient underwent orthognathic surgery could to a certain extent be delineated. It would appear that 33.1% of the skeletal patterns were Class III anomalies, whereas 23.8% involved skeletal Class II anomalies. The vast majority of orthognathic procedures were for the correction of routine dentofacial deformities. Approximately 10% of the orthognathic procedures were for the correction of a syndromal form of jaw deformity.

Ninety-six percent of patients were discharged routinely without the need for a "home health care facility" or transfer to either a long-term or acute-care facility. The mean length of hospital stay was 2.95 days, and the mean hospital bill generated costs of $47,348.00. This was assumed to include all costs related to the hospital, operating room, and anesthesia, but it did not include the surgeons' fees. The hospital bill generated did not necessarily correlate with the amount actually paid by either the third-party insurance company or the self-paying patient. Data regarding the actual payments is not available. From the available data, the total estimated hospitalization charges for orthognathic procedures carried out in the United States during 2008 were estimated to be $466.8 million (not including the surgeons' fees). Surgeons in "teaching hospitals" performed 67.1% of the procedures, and surgeons in "large-bed hospitals" performed 70% of the procedures. Hospitals in urban areas accounted for 97.4% of the hospital admissions.

It must be understood that the study by Venugoplan and colleagues did not capture those orthognathic procedures carried out either in an outpatient setting (i.e., not requiring admission to hospital) or procedures that were performed exclusively in a "surgicenter." These parameters were outside of the NIS database. It is estimated that as many as another 20% of orthognathic procedures were likely completed in these outpatient or surgicenter settings during this time period, which could result in 2000 additional patients.

Conclusions

The term *dentofacial deformity* refers to significant deviations from the normal proportions of the maxillomandibular complex that also negatively affect the relationship of the teeth within each arch as well as the relationships of the archs with one another (i.e., occlusion). The affected individuals will have varying degrees of compromise in their head and neck functions related to breathing, swallowing, speech, chewing, and lip closure and posture. A negative impact of the maxillofacial dysmorphology on psychosocial health is also to be expected. Studies confirm that, at a minimum, 5% of both the U.S. and U.K. general populations will have dentofacial deformities that are associated with severe malocclusion and that require orthognathic surgery. It is likely that as high as 19% of individuals who present for orthodontic assessment would ideally require not just orthodontics but jaw-straightening procedures as well.

References

1. Agency for Healthcare Research and Quality: *NIS Database Documentation: The Nationwide Inpatient Sample (NIS)*. Available at www.hcup-us.ahrq.gov/db/nation/nis/nisdbdocumentation.jsp. Accessed November 29, 2012.
2. Allwright WC, Bundred WH: A survey of handicapping dentofacial anomalies among Chinese in Hong Kong. *Int Dent J* 14:505–519, 1964.
3. American Association of Oral and Maxillofacial Surgeons: *Residency procedure*

report, Chicago, 2007, American Association of Oral and Maxillofacial Surgeons.
4. Angle EH: Classification of malocclusion. *Dent Cosmos* 41:248–264, 1899.
5. Ast D-B, CMOS JP, Cons NC: The prevalence and characteristics of malocclusion among senior high school students in upstate New York. *Am J Orthod* 51:437–445, 1965.
6. Baume LJ, Horowitz HS, Summers CJ, et al: A method of examining occlusal traits developed by the FDI commission on classification and statistics for oral conditions (COCSTOQ). *Int Dent J* 23:530–537, 1973.
7. Bjork A, Krebs AA, Solow B: A method for epidemiological registration of malocclusion. *Acta Odontol Scand* 22:27–41, 1964.
8. Bolton WA: Disharmony in tooth size and its relation to the analysis and treatment of malocclusion. *Angle Orthod* 28:113–130, 1958.
9. Brook PH: The development of an orthodontic treatment priority index. MSc thesis. University of Manchester, 1987.
10. Brook PH, Shaw WC: The development of an index of orthodontic treatment priority. *Eur J Orthod* 11:309–320, 1989.
11. Carlos JP: Evaluation of indices of malocclusion. In *Transactions of the Second FDI Conference on Oral Epidemiology, Epidemiological Assessment of Dentofacial Anomalies* 20(4):606–615, 1969.
12. Cirainger RM: The statistical basis for international dental epidemiology. World Health Organization Document No. PA/98.6
13. DeLuke DM: Orthognathic surgery: the state of the art. *N Y State Dent J* 64:30, 1998.
14. Draker HL: Handicapping labio-lingual deviations, a proposed index for public health purposes. *Am J Orthod* 46:295–305, 1960.
15. Emrich RE, Brodie AG, Blayney JR: Prevalence of class I, class II, and class III malocclusions (Angle) in an urban population: an epidemiological study. *J Dent Res* 44:947–953, 1965.
16. Evans MR, Shaw WC: Preliminary evaluation of an illustrated scale for rating dental attractiveness. *Eur J Orthod* 9:314–318, 1987.
17. Foster TD, Walpole Day AJ: A survey of malocclusion and the need for orthodontic treatment in a Shropshire school population. *Br J Orthod* 3:73–78, 1973.
18. Gardiner JH: A survey of malocclusion and some aetiological factors in 1000 Sheffield school children. *Dent Practit* 6:187–201, 1956.
19. Grainger RM: *Indexing handicapping malocclusions. Working paper for meeting of Expert Committee on Dental Health*, Geneva, 1961, World Health Organization.
20. Grainger RM: *Orthodontic treatment priority index. Public Health Service Publication No. 1000, Series 2, No 25*, Washington, DC, 1967, U.S. Government Printing Office.
21. Gravely JF, Johnson DB: Angle's classification of malocclusion: an assessment of reliability. *Br J Orthod* 3:79–86, 1974.
22. Grewe JM, Cervenka J, Shapiro BL, et al: Prevalence of malocclusion in Chippewa Indian children. *J Dent Res* 47:302–305, 1968.
23. Hausamen JE: The scientific development of maxillofacial surgery in the 20th century and an outlook into the future. *J Craniomaxillofac Surg* 29:2–21, 2001.
24. Haynes S: Discontinuation of orthodontic treatment in the General Dental Service in England and Wales 1972-1979. *Br Dent J* 152:127–129, 1982.
25. Helm S, Kreiborg S, Barlebo J, et al: Estimates of orthodontic treatment need in Danish schoolchildren. *Community Dent Oral Epidemiol* 3:136–142, 1975.
26. Helm S: Intra-examiner reliability of epidemiological registrations of malocclusion. *Acta Odontol Scand* 35:161–165, 1977.
27. Horup N, Melsen B, Terp S: Relationship between malocclusion and maintenance of teeth. *Community Dent Oral Epidemiol* 15:74–78, 1987.
28. Jarvinen S: Traumatic injuries to upper permanent incisors related to age and incisal overjet. *Acta Odontol Scand* 37:335–338, 1979.
29. Kelley JE, Sanchez M, Van Kirk GE: *An assessment of the occlusion of teeth of children 6-11 years. U.S. Department of Health, Education, and Welfare, Public Health Service publication (HRA) No. 74-1612*, Washington, DC, 1973, National Center for Health Statistics.
30. Kelley JE, Harvey CR: *An assessment of the occlusion of teeth of youths 12-17 years. U.S. Department of Health, Education, and Welfare, Public Health Service publication (HRA) No. 77-1644*, Washington, DC, 1977, National Center for Health Statistics.
31. Kelly JF, Helfrick JF, Smith DW, Jones BL: A survey of oral and maxillofacial surgeons concerning their knowledge, beliefs, attitudes, and behavior relative to parameters of care. *J Oral Maxillofac Surg* 50:50–58, 1992.
32. Lau D, Griffiths G, Shaw WC: Reproducibility of an index for recording the alignment of individual teeth. *Br J Orthod* 11:80–84, 1984.
33. Linder-Aronson S: Orthodontics in the Swedish Public Dental Health System. *Trans Eur Orthod Soc* 233–240, 1974.
34. Lundstrom A: Need for treatment in cases of malocclusion. *Trans Eur Orthod Soc* 53:111–123, 1977.
35. Malmgren O: Studies on the need and demand for orthodontic treatment. *Swed Dent J Suppl* (6):1–121, 1980.
36. Massler M, Frankel JM: Prevalence of malocclusion in children aged 14-18 years. *Am J Orthod* 37:751–768, 1951.
37. Mills LF: Epidemiologic studies of occlusion: IV. The prevalence of malocclusion in a population of 1,455 school children. *J Dent Res* 45:332–336, 1966.
38. Mohlin B, Thilander B: The importance of the relationship between malocclusion and mandibular dysfunction and some clinical applications in adults. *Eur J Orthod* 16:192–204, 1984.
39. Montegi E, Hatch JP, Rugh JD, Yamaguchi H: Health-related quality of life and psychosocial function 5 years after orthognathic surgery. *Am J Orthod Dentofacial Orthop* 124:138–143, 2003.
40. Moyers RE, van der Linden FPGM, Riolo ML, et al: *Standards of human occlusal development*, Ann Arbor, MI, 1976, University of Michigan Center for Human Growth and Development, 1976.
41. National Center for Health Statistics: *Volume of dental visits, United States, July 1963-June 1964. Vital and Health Statistics. PHS Pub No 1000-Series 10-No 23. Public Health Service*, Washington, DC, 1965, U.S. Government Printing Office.
42. National Center for Health Statistics: *Decayed, missing, and filled teeth in adults. Vital and Health Statistics. PHS Pub No 1000-Series 11-No 23. Public Health Service*, Washington, DC, 1967, U.S. Government Printing Office.
43. National Center for Health Statistics: *Plan, operation and response results of a program of children's examinations. Vital and Health Statistics. PHS Pub No 1000-Series l-No 5. Public Health Service*, Washington, DC, 1967, U.S. Government Printing Office.
44. National Center for Health Statistics: *The orthodontic treatment priority index. Vital and Health Statistics. PHS Pub No 1000-Series 2-No 25. Public Health Service*, Washington, DC, 1967, U.S. Government Printing Office.
45. National Center for Health Statistics: *Plan and operation of a health examination survey of US youths 12-17 years of age. Vital and Health Statistics. PHS Pub No 1000-Series l-No 8. Public Health Service*, Washington, DC, 1969, U.S. Government Printing Office.
46. National Center for Health Statistics: *Decayed, missing, and filled teeth among children. Vital and Health Statistics. Series 11-No 106. DHEW Pub No (HSM) 72-1003. Health Services and Mental Health Administration*, Washington, DC, 1971, U.S. Government Printing Office.
47. National Center for Health Statistics: *Dental visits, volume and interval since last visit, United States, 1969. Vital and Health Statistics. Series 10-No 76. DHEW Pub No (HSM) 72-1066. Health Services and Mental Health Administration*, Washington, DC, 1972, U.S. Government Printing Office.
48. National Center for Health Statistics: *Plan and initial program of the health examination survey. Vital and Health Statistics. Series l-No 4. DHEW Pub No (HRA) 74-1038. Health Resources Administration*, Washington, DC, 1973, U.S. Government Printing Office.
49. National Center for Health Statistics: *An assessment of the occlusion of the teeth of children 6-11 years, United States. Vital and Health Statistics. Series 1l-No 130. DHEW Pub No (HRA) 74-1612. Health Resources Administration*, Washington, DC, 1973, U.S. Government Printing Office.
50. National Center for Health Statistics: *Decayed, missing, and filled teeth among youths 12-17 years, United States. Vital and Health Statistics.*

Series 1l-No 144. DHEW Pub No (HRA) 75-1626. Health Resources Administration, Washington, DC, 1974, U.S. Government Printing Office.
51. National Center for Health Statistics: *Vital statistics of the United States, 1971, Vol. I, Nationality. DHEW Pub No (HRA) 75-1113. Health Resources Administration*, Washington, DC, 1975, U.S. Government Printing Office.
52. Newman GV: Prevalence of malocclusion in children six to fourteen years of age and treatment in preventable cases. *J Am Dent Assoc* 52:566–575, 1965.
53. Proffit WR, Fields HW, Jr, Moray LJ: Prevalence of malocclusion and orthodontic treatment need in the United States: Estimates from the NHANES III survey. *Int J Adult Orthod Orthognath Surg* 13:97–106, 1998.
54. Salzmann JA: Handicapping malocclusion assessment to establish treatment priority. *Am J Orthod* 54:749–765, 1968.
55. Sandali T: Irregularities of the teeth and their relation to the periodontal condition with particular reference to the lower labial segment. *Trans Eur Orthod Soc* 319–333, 1973.
56. Shaw WC, Addy M, Ray C: Dental and social effects of malocclusion and effectiveness of orthodontic treatment: a review. *Community Dent Oral Epidemiol* 8:36–45, 1980.
57. Shaw WC, Addy M, Dummer PMH, et al: Dental and social effects of malocclusion and effectiveness of orthodontic treatment: a strategy for investigation. *Community Dent Oral Epidemiol* 14:60–64, 1986.
58. Singh IJ, Savara BS: Norms of size and annual increments of seven anatomical measures of maxillae in girls from 3 to 17 years of age. *Angle Orthod* 36:312, 1968.
59. Siriwat PP, Jarabak JR: Malocclusion and facial morphology: is there a relationship? An epidemiologic study. *Angle Orthod* 55:127–138, 1985.
60. Sullivan SM: Orthognathic surgery dilemma: increasing access. *J Oral Maxillofac Surg* 69:813–816, 2011.
61. Summers CJ: The occlusal index: a system for identifying and scoring occlusal disorders. *Am J Orthod* 57:552–567, 1971.
62. The American Association of Orthodontists: Study of the availability of orthodontists' services. *Am J Orthod* 68(3):326–338, 1975.
63. U.S. National Health Survey: *Dental care, interval and frequency of visits, United States, July 1957-June 1958. PHS Pub No 584-1314. Public Health Service*, Washington, DC, 1960, U.S. Government Printing Office.
64. Van Kirk LE, Jr, Pennell EH: Assessment of malocclusion in population groups. *Am J Pub Health* 49(9):1157–1163, 1959.
65. Venugoplan SR, Nanda V, Turkistani K, et al: Discharge patterns of orthognathic surgeries in the United States. *J Oral Maxillofac Surg* 70:e77–e86, 2012
66. Zins JE, Bruno J, Moreira-Gonzalez A, et al: Orthognathic surgery: is there a future? *Plast Reconstr Surg* 116:1442–1450, 2005.

4

Hereditary, Developmental, and Environmental Influences on the Formation of Dentofacial Deformities

JEFFREY C. POSNICK, DMD, MD

- Development of the Head and Neck
- Syndromes and Anomalies that Include Dentofacial Deformities
- Hereditary Tendencies in the Development of Dentofacial Deformities
- Environmental and Neuromotor Effects on Skeletal Growth
- Effects of Trauma on Skeletal Growth
- Effects of Tumors and Growths on Skeletal Growth
- Conclusions

The term *dentofacial deformity* is generally used to describe a significant disproportion of the jaws in association with malocclusion. It is estimated that, at a minimum, 5% of the population will have a discrepancy that falls into the developmental dentofacial deformity category. Another 45% of the population will fall into a much larger group of those with malocclusion but without what is considered a "notable" jaw discrepancy component.[2,3,5,56,65,72] After jaw growth is complete (i.e., 14 to 16 years of age in girls and 16 to 18 years of age in boys), the definitive reconstruction of a dentofacial deformity can go forward.

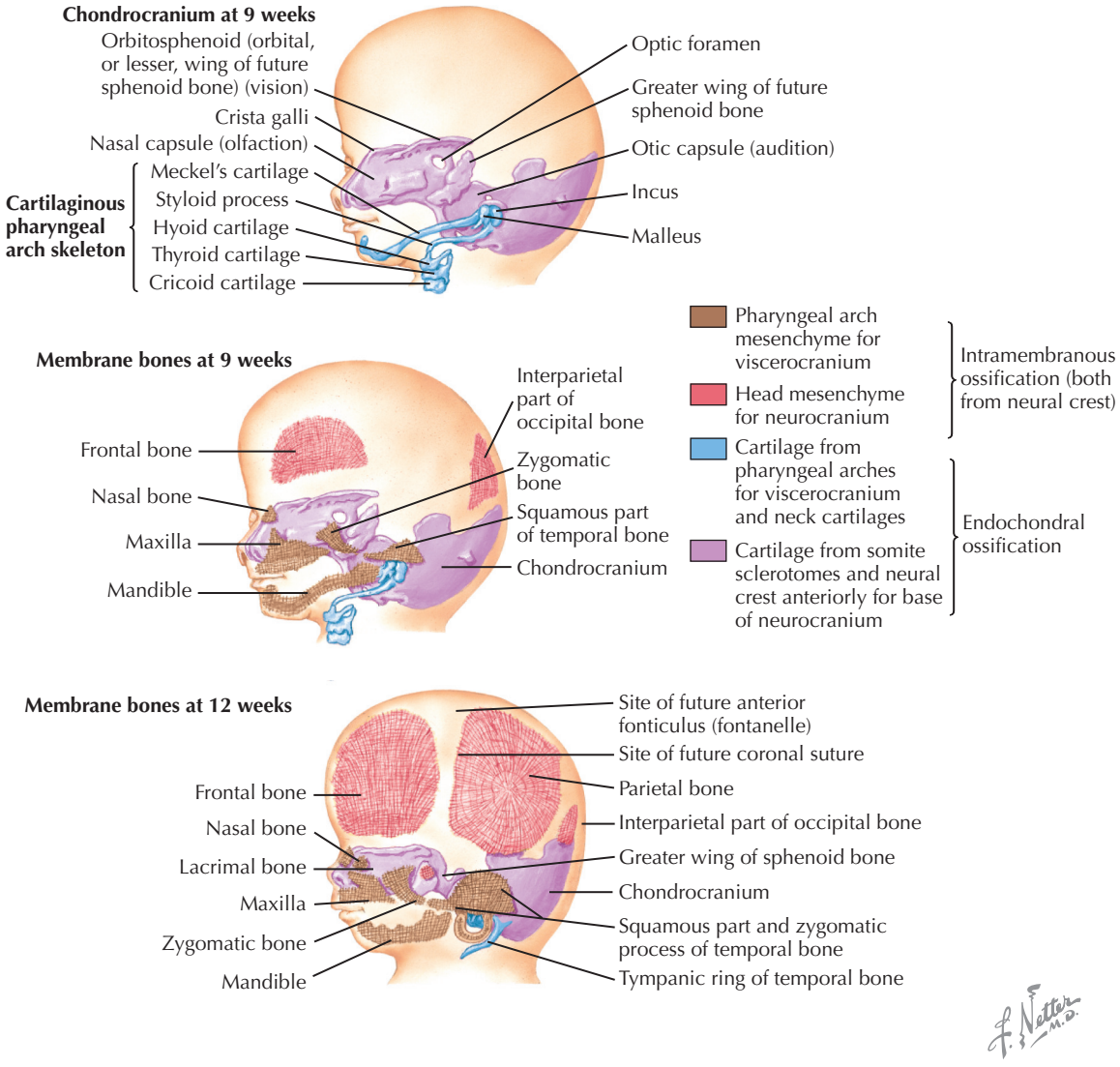
Knowing the etiology of a patient's jaw deformity can be important to the fundamental understanding of how and when to treat the problem. Knowledge of normal facial growth and development as well as the effect of any associated malformations (e.g., hemifacial microsomia with cervical spine anomalies) is useful both to limit complications during treatment and for family planning. Understanding the natural history of a condition that involves environmental influences may provide an opportunity to prevent or at least lessen the impact of the deformity. Knowledge of factors that influence facial skeletal growth and dental development (e.g., the muscles of the neck, tongue, and lips; breathing patterns) is important. Factors that are known to alter facial growth and cause jaw discrepancy can be subdivided to include the following: 1) known syndromes and anomalies; 2) hereditary tendencies; 3) environmental and neuromotor effects; 4) effects of trauma; and 5) effects of tumors or growths.

Development of the Head and Neck

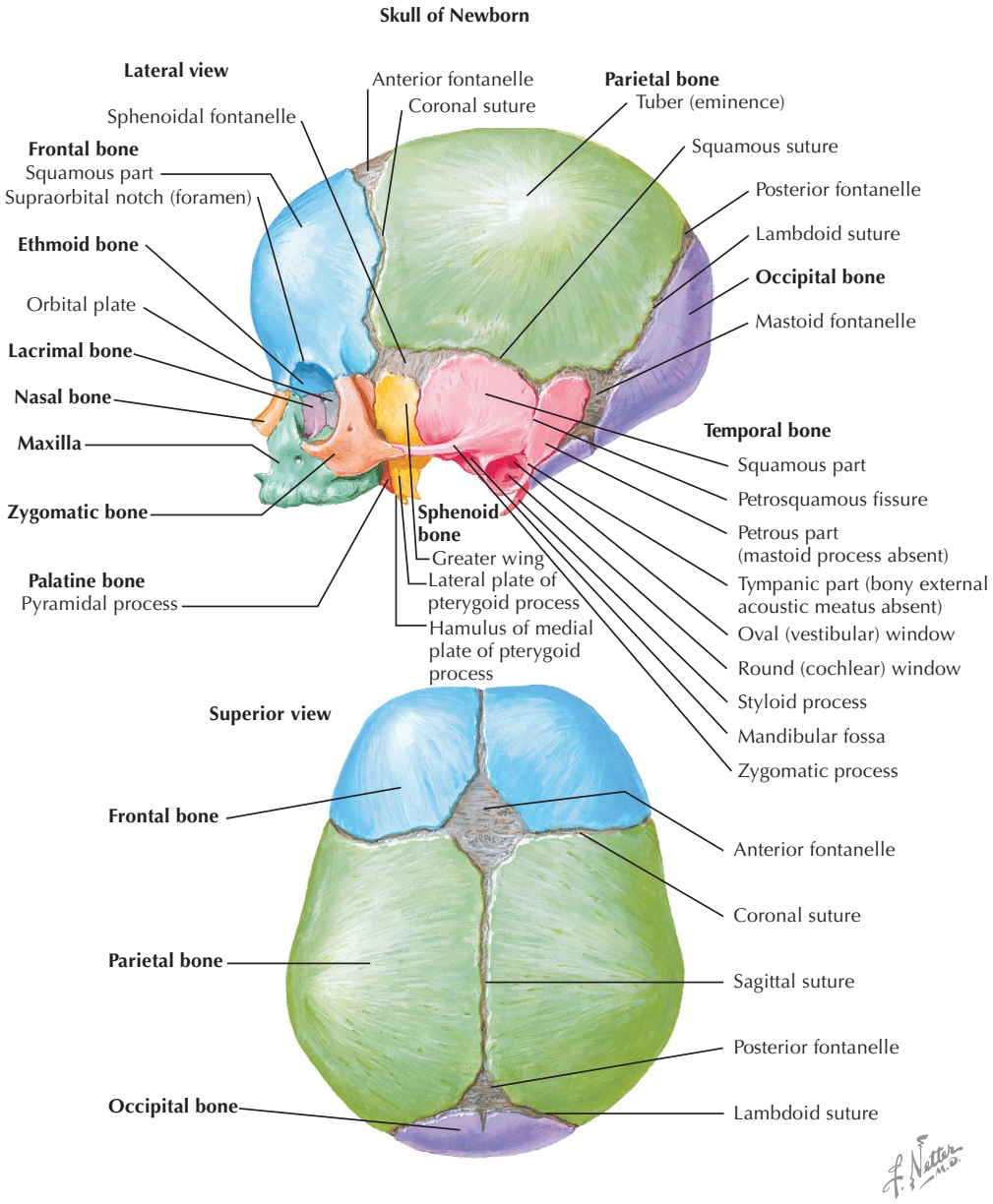
Skull

The *skull* is formed from the *lateral plate mesoderm* (the neck region), the *paraxial mesoderm,* and the neural crest (Fig. 4-1). The bony skull is formed by one of two mechanisms: *intramembranous ossification* or *endochondral ossification*. Skull development is divided into two parts: the *viscerocranium* and the *neurocranium*. The viscerocranium forms the bones of the face, whereas the neurocranium forms the bones of the cranial base and the cranial vault. The neurocranium can be divided into the membranous neurocranium and the cartilaginous neurocranium. The anterior fontanel (bregma) has a wide range of expected closure between 4 and 26 months of age. The posterior fontanel (lambda) generally closes between 1 and 2 months of age.

From the *first pharyngeal pouch* are formed the maxillary process, the mandibular process, and the second pharyngeal arch. Many bones are derived from the maxillary arch, including the maxilla, the temporal bones, the zygoma, the palatine bone, the lacrimal bone, the vomer, the nasal bones, and the inferior nasal concha. The *mandibular process* forms the mandible, the sphenomandibular ligament, the malleus, and the incus. From the *second pharyngeal arch* are



• **Figure 4-1** Illustrations of the development of the skull. *From* www.netterimages.com. *© Elsevier Inc. All rights reserved.*



• **Figure 4-1, cont'd**

formed the styloid process, the stapes, the hyoid bone, and the stylohyoid ligament.

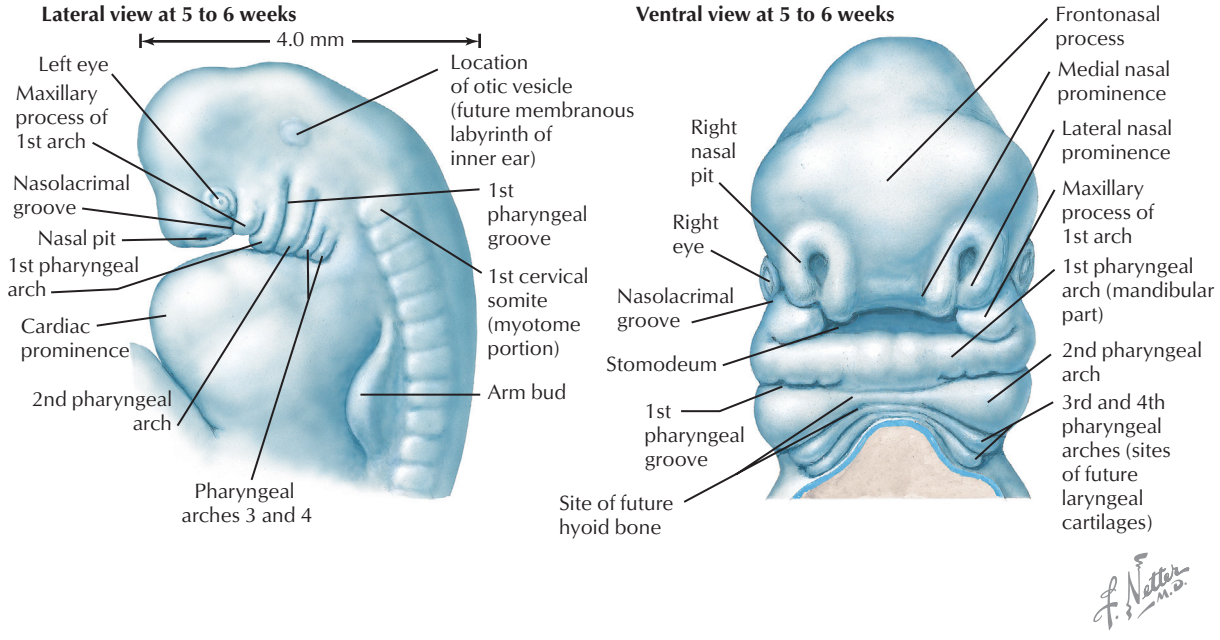
Face

The *face* is formed mainly from the *neural crest,* which makes three "swellings" that surround the stomodeum (Fig. 4-2). The three swellings are the *frontonasal prominence,* the *maxillary prominence* (from the first pharyngeal arch), and the *mandibular prominence* (from the first pharyngeal arch).

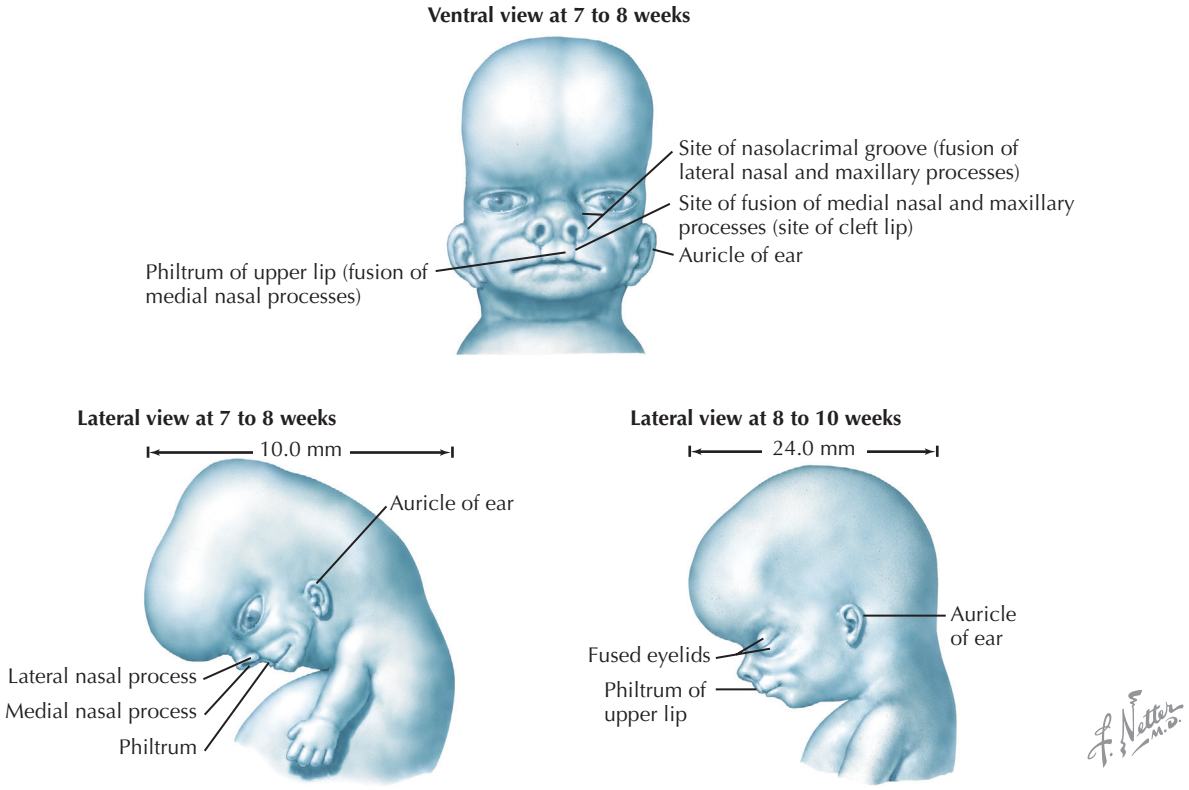
Lateral to the frontonasal prominence, there are two areas of ectoderm that form the *nasal placodes,* which invaginate in the center to form the *nasal pits.* The placodes that invaginate create ridges of tissues on either side of the pits. The ridges are called the *lateral nasal prominence* and the *medial nasal prominence*. The fusion of the medial nasal prominence at the midline results in the formation of the *intermaxillary segment*.

Palate

The *palate* is formed by the *primary palate* (intermaxillary segment) and the *secondary palate* (protrusions from the lateral prominences) (Fig. 4-3). The *intermaxillary segment* (primary palate) is the initial portion of the palate to develop. It contains the central and lateral incisors. Swellings of the *maxillary prominence* form shelves that project medially but that are separated by the tongue. When the tongue no longer occupies the space between the palatal shelves, these processes fuse together to form the secondary palate. The primary and secondary palatal tissues all meet at the *incisal foramen*. The primary and secondary palates and the nasal septum fuse to form the *definitive palate*.



• **Figure 4-2** Illustrations of the development of the face. *From* www.netterimages.com. *© Elsevier Inc. All rights reserved.*



• **Figure 4-2, cont'd**

Syndromes and Anomalies that Include Dentofacial Deformities

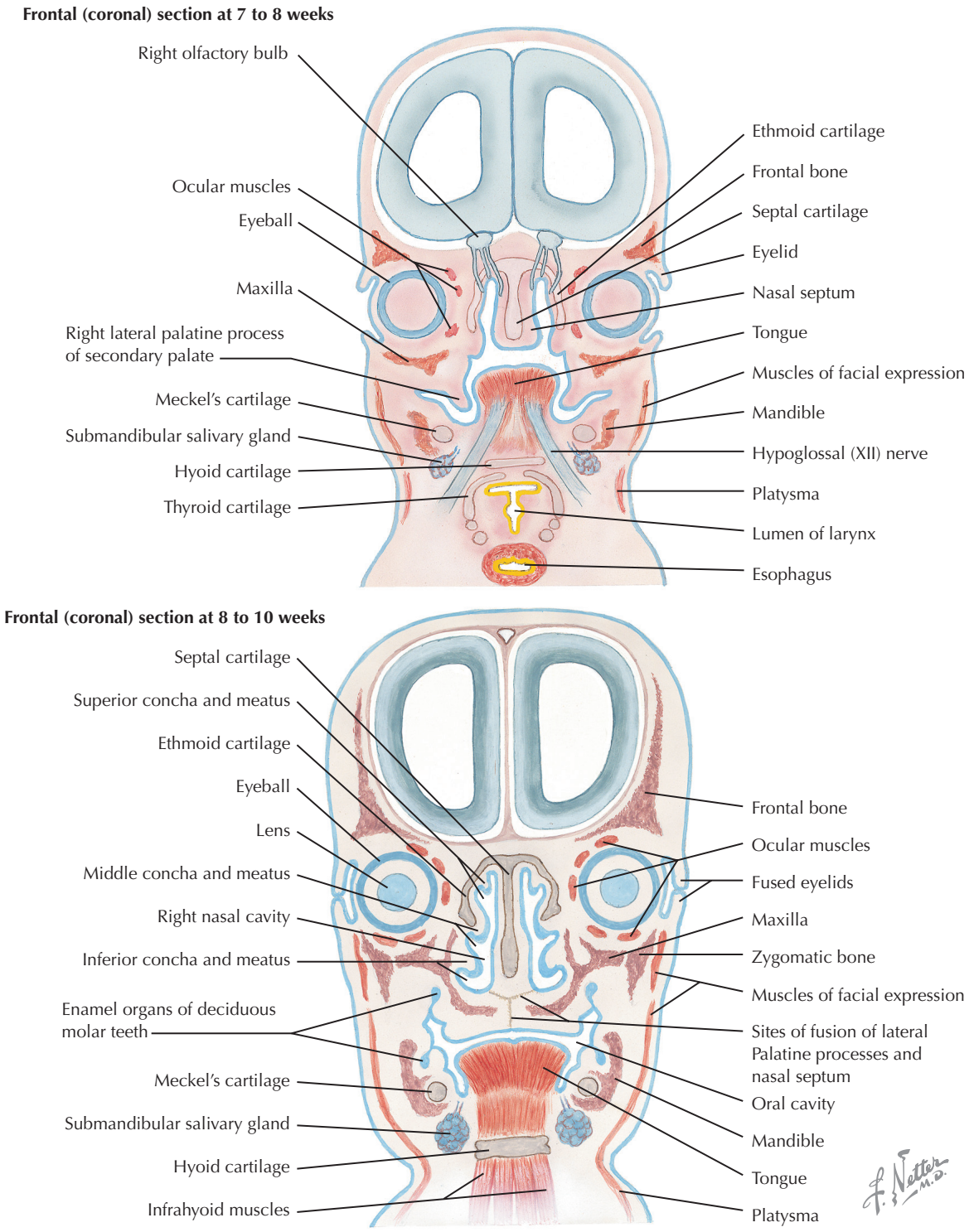
Many of the syndromes and anomalies that involve the cranio-orbito-zygomatic (upper face) region also affect the maxillomandibular (lower face) location.[1,20,124] These conditions may involve any of the tissue components, including the skin, the muscles, the nerves, the fat, the vessels, the bone, the teeth, the cartilage, and the associated viscera (i.e., brain, ears, eyes, salivary glands, sinuses). *Known syndromes and anomalies* comprise only a small proportion of the dentofacial deformity group (see Chapters 27 through 31).[19] These conditions can be further subdivided according to etiology or tissue of origin.

Deficiencies of Midline Tissue Structures

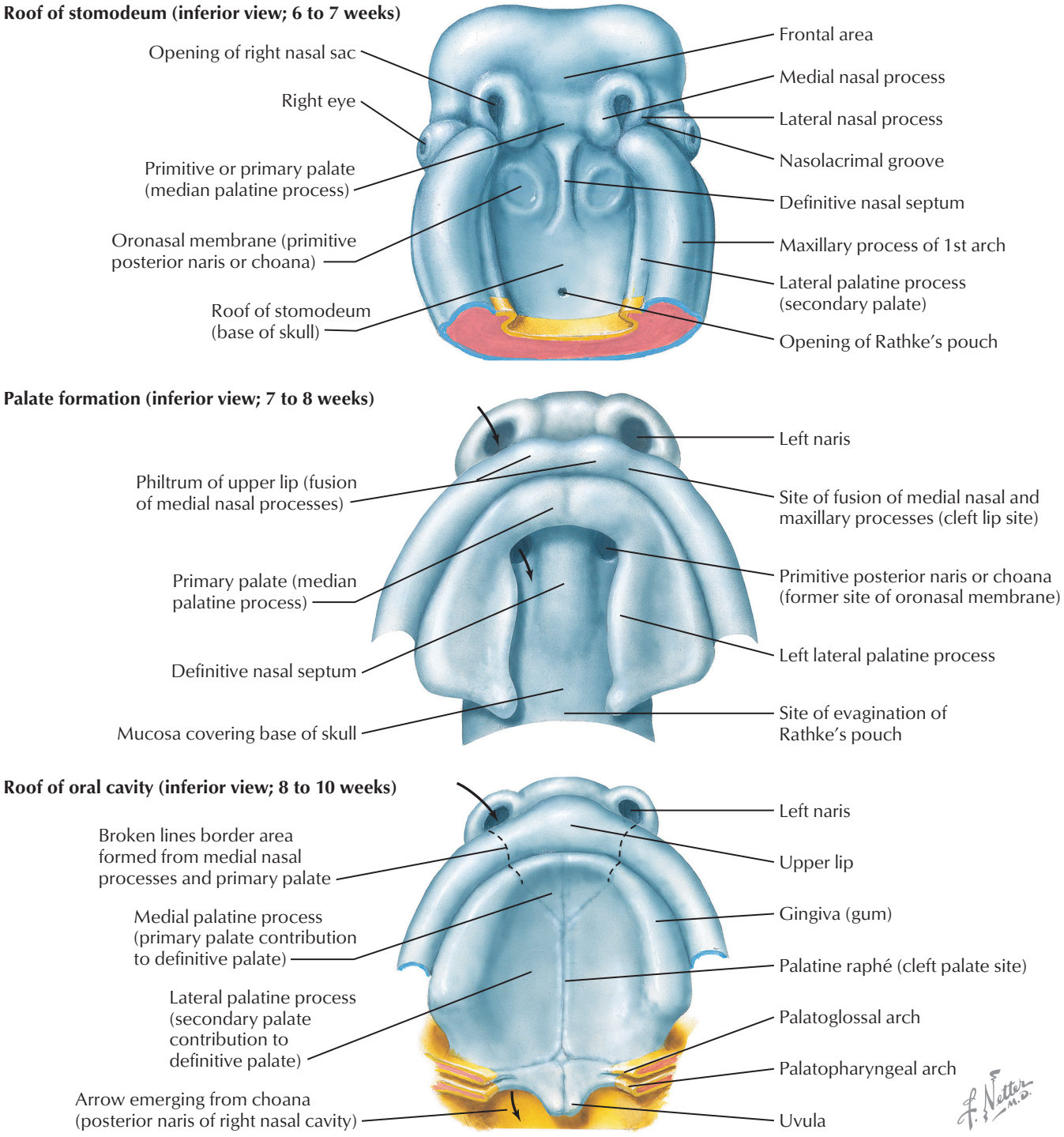
Holoprosencephalies involve variable deficiencies of midline tissues. These structural deficiencies have been documented to occur early during embryologic development.[16,18,23,27] They may range from severe brain anomalies to simple absence or hypoplasia of the corpus callosum. Severe facial anomalies such as cyclopia, ethmocephaly, and cebocephaly are always associated with severe holoprosencephaly, and they are incompatible with life. This can also be true of premaxillary agenesis; however, in some instances, patients may survive for several months or even years. There are reported cases of premaxillary agenesis with normal head circumference and no brain abnormalities (i.e., without holoprosencephaly), although these patients sometimes have mild mental deficiencies. Syntelencephaly (the absence or hypoplasia of the corpus callosum or absent or hypoplastic olfactory tracts and bulbs) is compatible with life and should thus be treated. Associated facial anomalies include retinal colobomas, cleft lip, cleft palate, single maxillary central incisor, and midface deficiencies. Holoprosencephaly has multiple causes and includes many chromosomal types, many monogenetic disorders, and at least three teratogens.

Deficiencies and Anomalies of Neural Crest Origin

Most of the tissues of the face, including the muscular and skeletal components, are derived from the *mesoderm*. Interestingly, throughout the rest of the body, these components (i.e., muscle and skeleton) are derived from an *ectodermal* origin.[19] The facial *mesodermal elements* develop from the neural crest and then migrate downward beside the neural tube and laterally under the surface ectoderm. When the *neural crest cells* have completed migration to their specific location, then facial development is dominated by specific growth centers, the formation of visceral structures (e.g., brain, eyes, sinuses), and the differentiation of the tissue layers. *Facial anomalies of neural crest origin* are thought to arise when the neuroepithelium has apoptosis (i.e., the cells die).[25] This results in less neuroepithelium to migrate through into the facial locations and to ultimately



• **Figure 4-3** Illustrations of the development of the palate. *From* www.netterimages.com. *© Elsevier Inc. All rights reserved.*

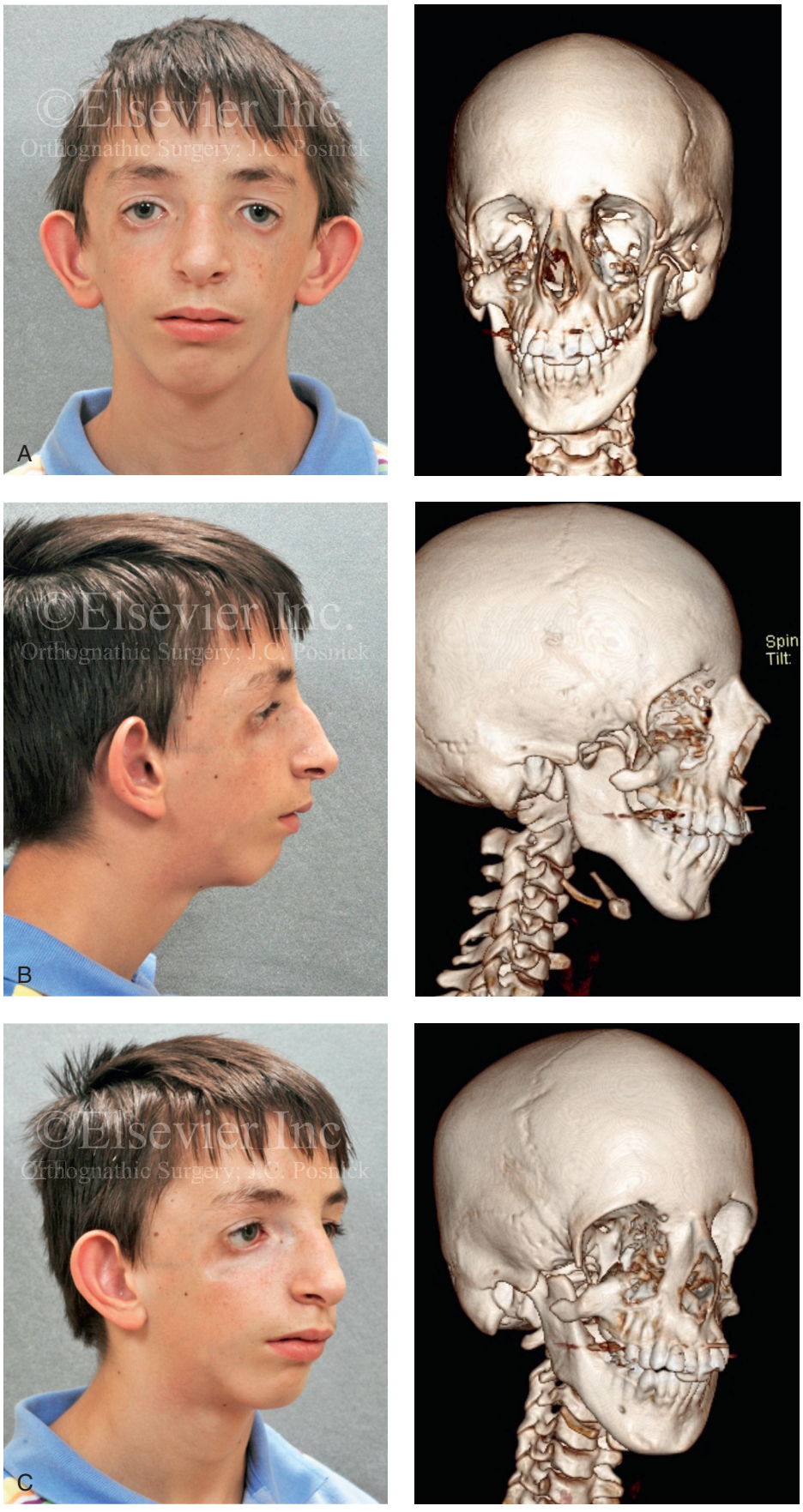


• **Figure 4-3, cont'd**

differentiate. Current knowledge of Treacher Collins syndrome indicates that this is not a migration problem but simply a lack of cells to migrate (Figs. 4-4 and 4-5).

Teratogens that are known to result in head and neck (neural crest) congenital anomalies include cytomegalovirus (microcephaly, hydrocephaly, microophthalmia); Dilantin (cleft lip and palate); vitamin D excess (premature suture closure); Valium (cleft lip and palate); Rubella virus (microophthalmia, cataracts, deafness); and thalidomide (variations of hemifacial microsomia and Treacher Collins syndrome). Despite these known relationships, teratogenic agents are not the most frequent causes of these syndromes.[1]

The *branchial arch syndromes* are neural crest anomalies that comprise an etiologically heterogeneous group of disorders.[14] They account for only a small percentage of the patients who present to the surgeon or orthodontist in need of jaw reconstruction. The best known of these are



• **Figure 4-4** An 11-year-old boy who was born with Treacher Collins syndrome has typical malformations of the soft tissues and the skeletal structures within the first and second branchial arches on both sides. There is significant hypoplasia of each zygomatic complex, the orbits, and the maxillomandibular structures (Kaban type II-A mandible). The soft tissues of the adnexal region are deficient. The external ears are malformed, but all parts are present. The central part of the face is fully formed. **A,** Frontal facial and computed tomography scan views. **B,** Profile facial and computed tomography scan views. **C,** Oblique facial and computed tomography scan views.

©Elsevier Inc.
Orthognathic Surgery, J.C. Posnick

• **Figure 4-5** This mother and daughter demonstrate the extent of variation of expression of Treacher Collins syndrome within a family. The mother was not aware that she carried the Treacher Collins gene until after the birth of her daughter. *From Posnick JC: Treacher Collins syndrome: perspectives in evaluation and treatment.* J Oral Maxillofac Surg *55:1120–1133, 1997.*

hemifacial microsomia, its variant Goldenhar syndrome, and Treacher Collins syndrome.[14] *Hemifacial microsomia* affects aural, zygomatic, and mandibular growth at a minimum (Fig. 4-6). The disorder may be mild or severe, and it is generally limited to one side of the face. However, bilateral involvement, with more severe expression on one side, is also known to occur. *Goldenhar syndrome,* which is a variant of hemifacial microsomia, also includes epibulbar dermoids and vertebral anomalies. Involvement is often but not always limited to the face. There may be cardiac, renal, skeletal, and central nervous system anomalies (see Chapter 28). At a minimum, patients with *Treacher Collins syndrome* demonstrate the physical findings of bilateral zygomatic hypoplasia, down-slanting palpebral fissures, malformed ears, and micrognathia (see Chapter 27). Inheritance occurs in an autosomal-dominant fashion, and expressivity varies (see Figs. 4-4 and 4-5). The syndrome maps to 5q32-q33.1, and mutations in the *TCOF1* gene are of the nonsense, insertion, deletion, or splice-site types.[14,30,139]

Clefting of the Lip and Palate

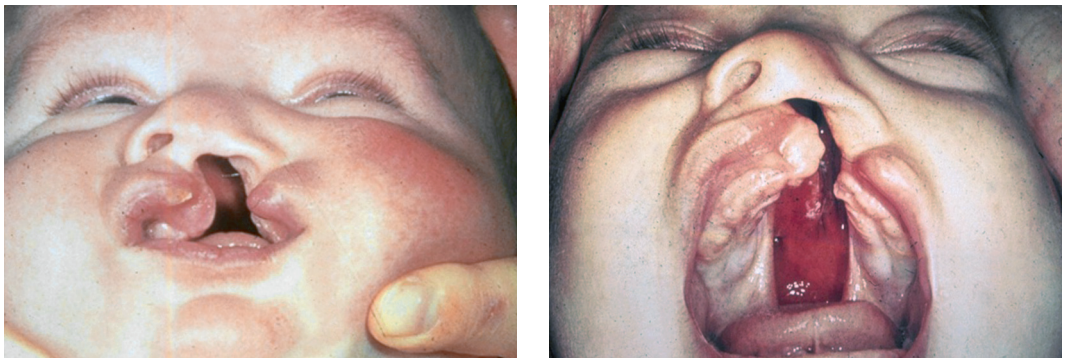
The most prevalent congenital defect of dentofacial development is *clefting of the lip, the palate, or both* (Figures 4-7 through 4-19). This condition occurs in approximately 1 in 1000 whites, 1 in 500 Asians, and 1 in 2000 blacks.[27] The etiology of clefts is often complex and multifactorial. Evidence supports the view that genetic factors are associated with orofacial clefting.[37] In twins with cleft lips and palates, concordance is far greater among monozygotic twins (40%) as compared with dizygotic twins (4.2%). In twins with isolated cleft palate, concordance is also higher among monozygotic twins (35%) as compared with dizygotic twins (7.8%).[138] Nevertheless, orofacial clefting is heterogeneous and variable, and it is likely determined by a number of major genes, minor genes, environmental factors, and a developmental threshold.[37] Some syndromes associated with clefting in which specific gene mutations have been identified include van der Woude syndrome *(IRF6)*, popliteal pterygium syndrome *(IRF6)*, autosomal-recessive cleft palate/ectodermal dysplasia *(PVRL1)*, hypodontia/clefting *(MSX1)*, Hay-Wells syndrome *(TP63)*, and cleft palate/ankyloglossia *(TBX22)*. These mutations explain only about 5% of clefting cases, with an additional 10% to 15% of cases explained by variations involving the *IRF6* gene.[110] Although orofacial clefting is caused by a malformation, the development of secondary deformities of the jaws after birth is well known (see Chapters 32, 33, and 34). For the most part, maxillary hypoplasia in patients with orofacial clefts is felt to be the result of palate surgical interventions carried out during infancy and early childhood and not from the primary congenital anomaly. Although van der Woude and Stickler are just 2 of more than 100 clefting syndromes, they demonstrate the importance of having an awareness of associated anomalies to assist with family counseling and to achieve effective reconstruction.

Van der Woude Syndrome

In 1845, Demarquay was the first to report the occurrence of congenital sinuses of the lower lip in combination with cleft lip and palate; this condition became known as *van der Woude syndrome*. This syndrome occurs in roughly 1% to 2% of patients with lip and palatal clefts. Van der Woude syndrome has an autosomal-dominant inheritance pattern;

Text continued on p. 91

• **Figure 4-6** A 7-year-old boy born with hemifacial microsomia and unilateral cleft lip and palate (left side) has typical malformations of the soft tissues and skeletal structures within the first and second branchial arches on the left side. There is an absence of the left zygomatic complex and hypoplasia of the orbit, the anterior cranial vault, and the maxilla. The mandibular malformation is a Kaban type III. The soft tissues of the left external ear, the ear canal adnexal structures, and the cheek region are markedly deficient. **A,** Frontal facial and computed tomography scan views. **B,** Oblique facial and computed tomography scan views.



• **Figure 4-7** Cleft lip and palate represents a spectrum of morphologic findings that are initially dependent on the individual's specific anomalies but that are then affected by surgical intervention during growth. A newborn with a complete (left side) unilateral cleft lip and palate is shown.

©Elsevier Inc.
Orthognathic Surgery: J.C. Posnick

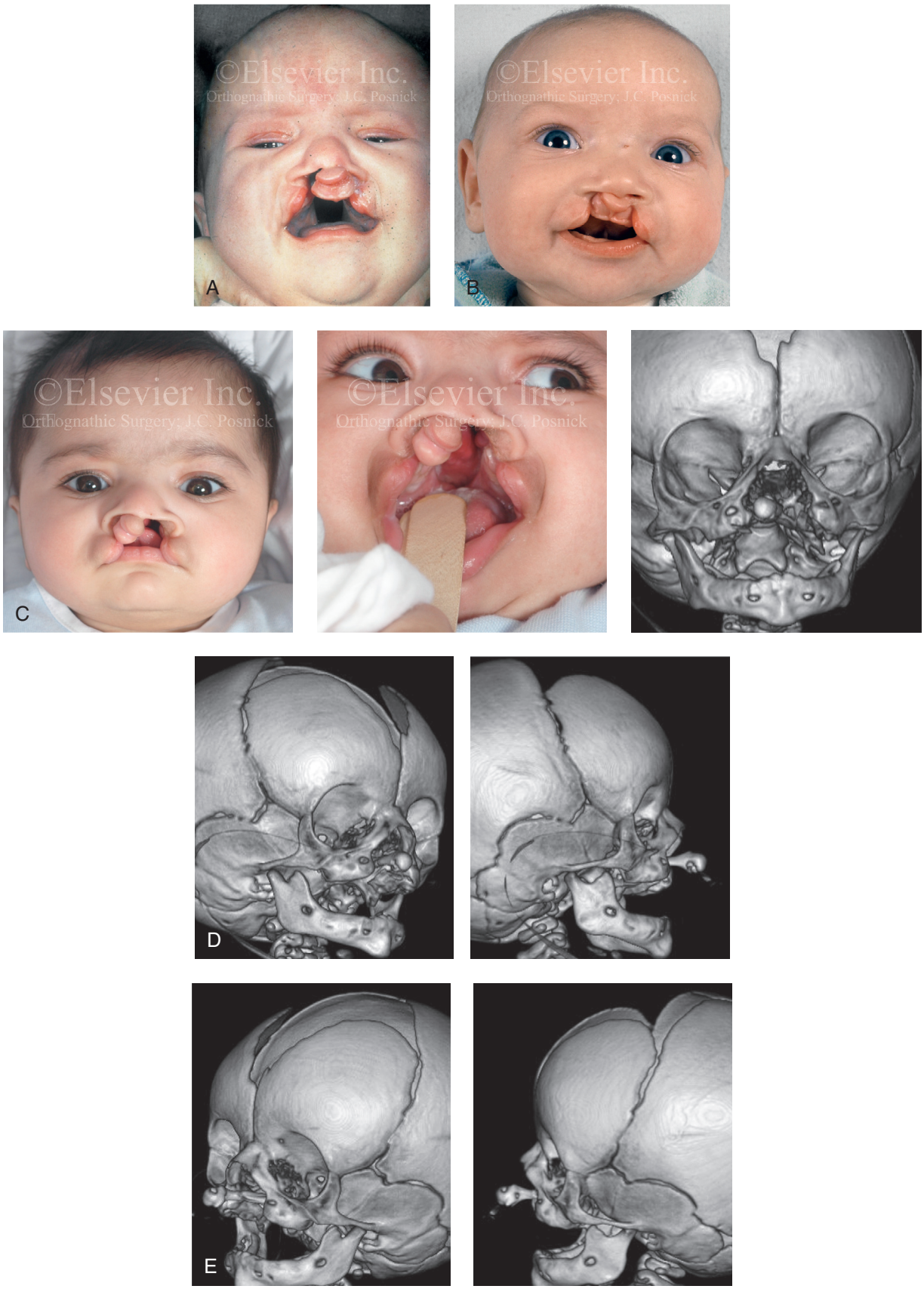
• **Figure 4-8** A Hispanic child was born with complete clefting of the left primary lip and palate. He was adopted when he was approximately 1 year old, before repair. He then underwent single-stage primary lip, nasal, and palate repair. He is shown before repair and then at the age of 2 years after single-stage repair.

©Elsevier Inc.
Orthognathic Surgery: J.C. Posnick

• **Figure 4-9** A child was born with an incomplete cleft of the left lip that also involved the alveolar ridge back to the incisal foramen. She is shown before primary lip and nasal reconstruction and then at the age of 5 years before alveolar cleft grafting.

©Elsevier Inc.
Orthognathic Surgery; J.C. Posnick

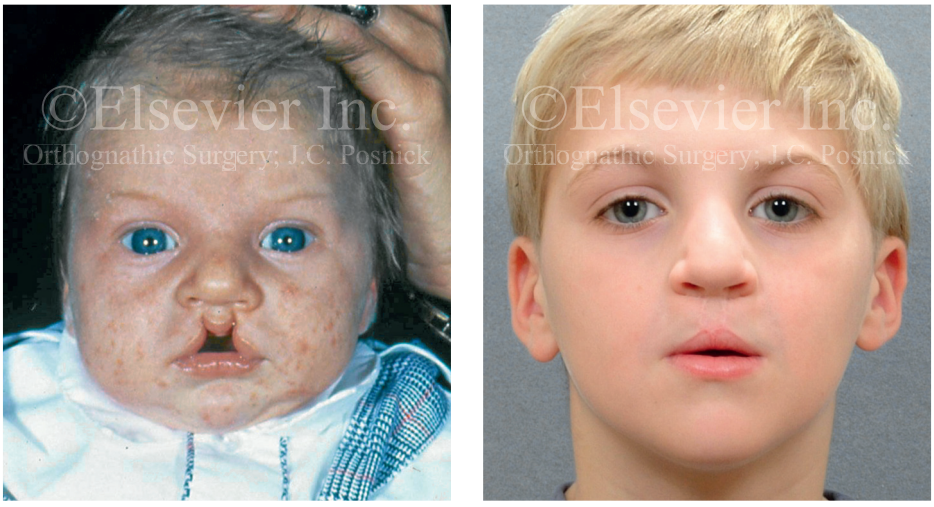
• **Figure 4-10** A child was born with a complete (left side) unilateral cleft lip and palate. He is shown before and at intervals after primary lip and palate repair. **A,** He is shown before primary lip and nasal reconstruction and **B,** at the age of 10 months, just before cleft palate repair. **C,** The same child at the age of 3 years and **D,** at the age of 8 years just before mixed dentition bone grafting.



• **Figure 4-11** Three newborns with bilateral cleft lip and palate (BCLP) are shown. **A,** A newborn with complete BCLP. **B,** A newborn with BCLP but with partial attachment of the lip and the nasal floor on the left side. **C,** A 3-month-old infant with complete BCLP is shown. The premaxilla is attached to the septum of the nose; it is forwardly projecting without attachment to the lateral lip segments. The wide separation of the palatal shelves is also demonstrated by the intraoral view. **D** and **E,** A series of computed tomography scan views of the maxillofacial complex of the infant shown in C demonstrate the skeletal anatomy associated with BCLP.

©Elsevier Inc.
Orthognathic Surgery: J.C. Posnick

• **Figure 4-12** A child born with complete bilateral cleft lip and palate. He is shown before and then 7 years after primary lip and palate repair, just before mixed dentition bone grafting.

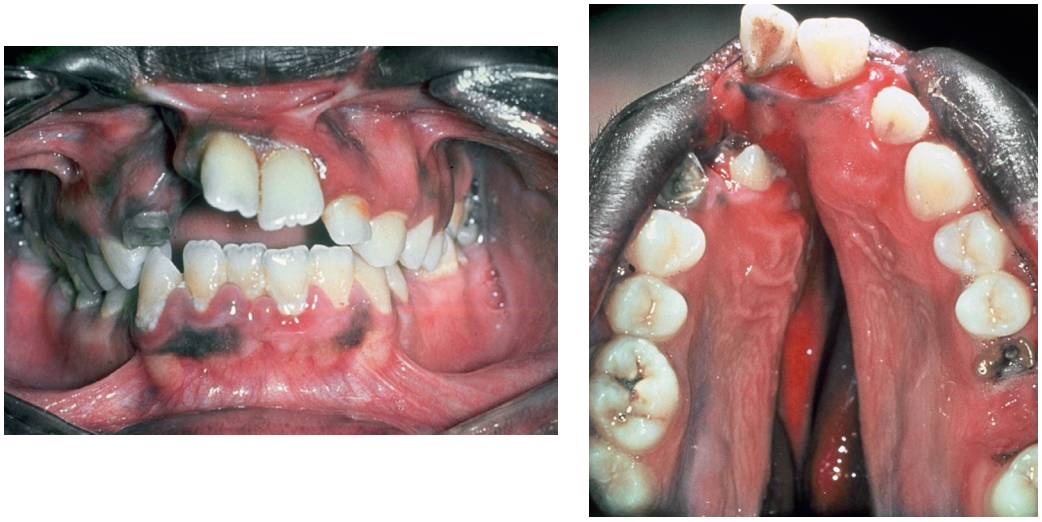

©Elsevier Inc.
Orthognathic Surgery: J.C. Posnick

• **Figure 4-13** A child born with incomplete bilateral cleft lip and palate. He is shown before and then 8 years after primary lip and palate repair, just before mixed dentition bone grafting.

©Elsevier Inc.
Orthognathic Surgery: J.C. Posnick

• **Figure 4-14** A child born with a complete cleft of the right lip and palate and incomplete clefting of the left lip. He is shown **A,** before and then **B,** at 2 and **C,** 8 years after primary lip and palate repair. The last photo was taken just before mixed dentition bone grafting.

• **Figure 4-15 A,** A child born with complete clefting of the secondary palate (i.e., incisal foramen through the uvula) is shown. **B,** An adult born with a complete unilateral cleft lip and palate is also shown. He underwent lip repair during childhood, but the cleft palate was neglected. Note the severe septal deviation that obstructs the right nasal valve.



• **Figure 4-16** A teenage boy from Jamaica who was born with a bilateral cleft lip and a unilateral cleft of the alveolar ridge and palate. He underwent rudimentary lip repair as a child. The unrepaired clefted alveolus and palatal anatomy are shown. Note the normal growth parameters of the upper jaw when the palate is unrepaired. With a lack of continuity of the alveolar ridge, there has been typical distorted growth of the maxilla.

• **Figure 4-17** A woman who was born with bilateral cleft lip and palate and pitting of the lower (central) lip. At the time of her pregnancy, an ultrasound confirmed twins, one of which was suspected of having bilateral cleft lip and palate and the other of having unilateral cleft lip and palate. This was documented at the time of delivery. **A,** A family with van der Woude syndrome, including a mother with a repaired bilateral cleft lip and palate and newborn twins. **B,** Twin A with bilateral cleft lip and palate. **C,** Twin B with unilateral cleft lip and palate.

• **Figure 4-18** A 16-year-old boy who was born with Stickler syndrome (type II collagen mutation). At the time of birth, Pierre Robin sequence was appreciated. The patient underwent repair of the cleft palate before he was 1 year old. He had positive eye findings, and he required the treatment of a retinal detachment during his teenage years. A small cataract is being followed. He arrived for the evaluation of a jaw deformity and malocclusion characterized by maxillomandibular deficiency with anterior open-bite malocclusion. This was combined with chronic obstructive nasal breathing and a long face growth pattern. Attempted growth modification and camouflage orthodontics earlier during life were ineffective. There was generalized root deficiency throughout the maxillary and mandibular dentition, likely as a result of the collagenopathy. **A,** Frontal views in repose before and after treatment. **B,** Frontal views with smile before and after treatment.

Continued

• **Figure 4-18, cont'd** The patient agreed to an orthodontic and surgical approach. Further orthodontic (dental) decompensation was cautiously carried out as a result of the compromised periodontal apparatus. The procedures included maxillary Le Fort I osteotomy (vertical intrusion, horizontal advancement, counterclockwise rotation, and arch expansion); bilateral sagittal split ramus osteotomies (horizontal advancement and counterclockwise rotation); osseous genioplasty (vertical shortening and horizontal advancement); and septoplasty, inferior turbinate reduction, and nasal floor recontouring. **C,** Oblique facial views before and after treatment. **D,** Profile views before and after treatment.

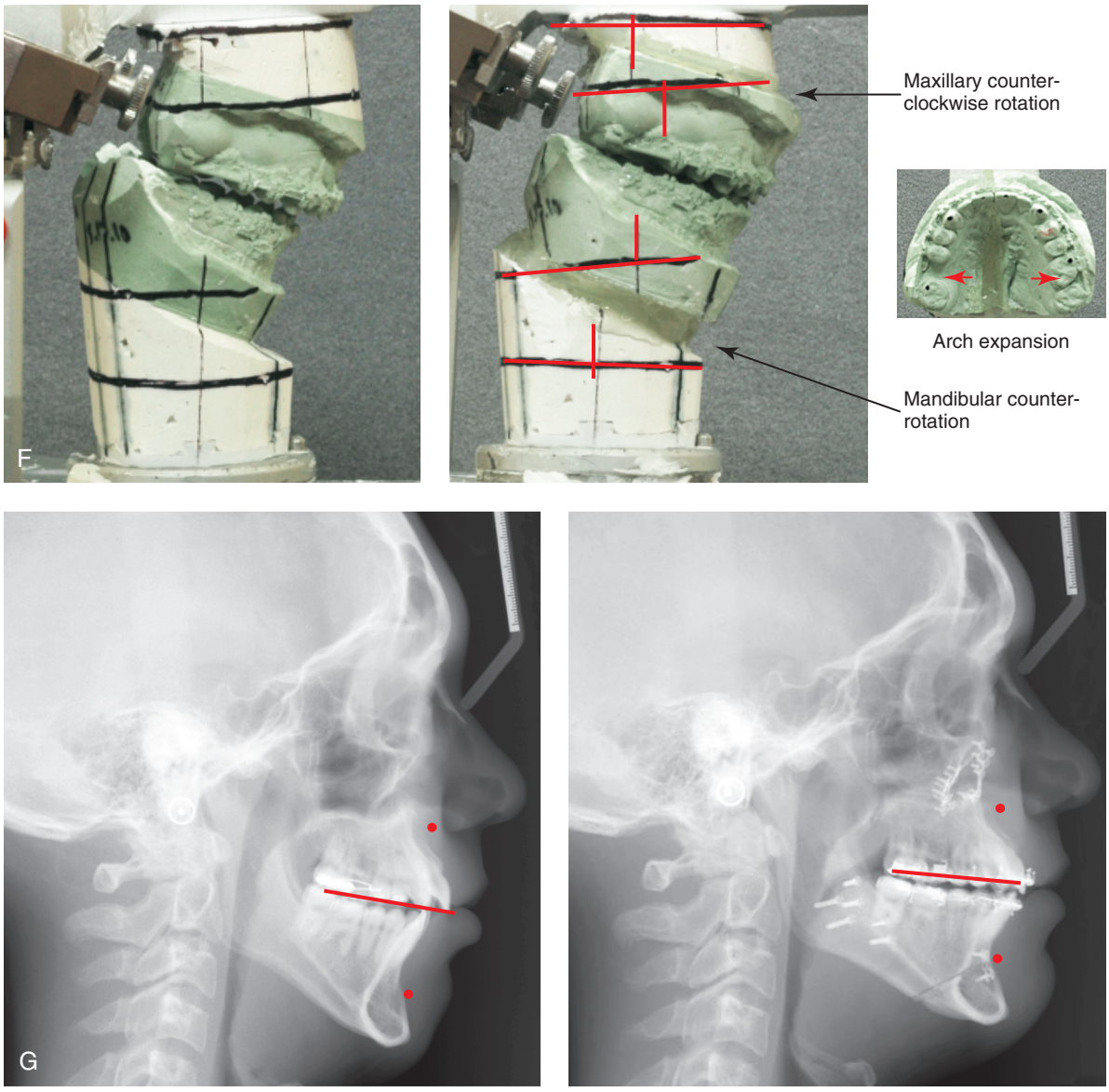
Prior to retreatment

Pre surgery

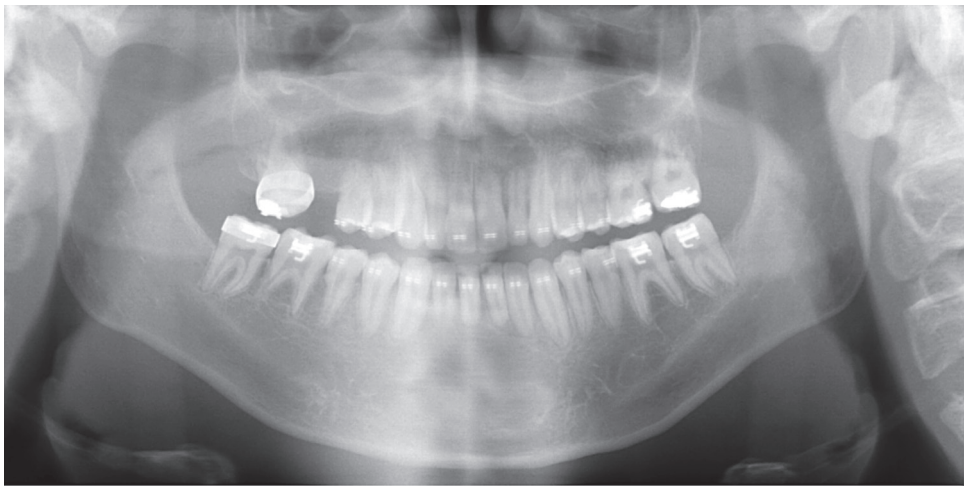
After treatment

• **Figure 4-18, cont'd E,** Occlusal views before retreatment, with orthodontics in progress, and after treatment.

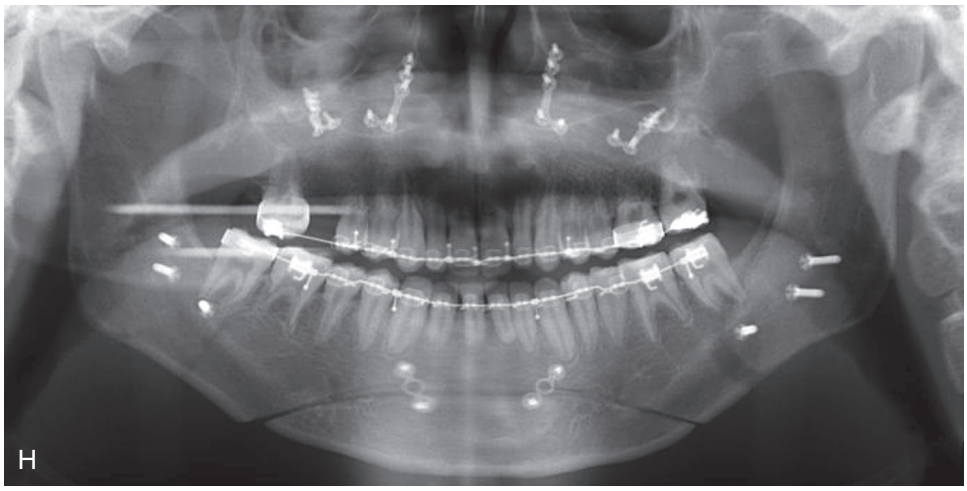
Continued



• **Figure 4-18, cont'd F,** Articulated dental casts that indicate analytic model planning. **G,** Lateral cephalometric radiographs before and after treatment.



Prior to retreatment



After surgery

• **Figure 4-18, cont'd H,** Panorex radiographs before and after treatment that demonstrate generalized limited root formation. The right maxillary first molar was lost 5 years earlier as a result of limited roots. The right maxillary first molar was lost 1½ years after surgery as a result of limited root support; the right mandibular first molar will likely be lost to similar pathology. Periodontal evaluation and treatment are ongoing. Future plans are for the placement of dental implants in the right posterior maxilla and mandible.

• **Figure 4-19** A child born with Robin sequence, which consists of retrognathia with glossoptosis, clefting of the secondary palate, and a degree of respiratory distress. **A,** Frontal facial and intraoral views demonstrating clefting of the secondary palate. **B,** Profile facial view indicating retrognathia. Computed tomography scan demonstrating retrognathia but with all of the components of the jaw present. For this child, the small mandible is the result of deforming forces during fetal development rather than the result of a malformation. Catch-up growth of the mandible is anticipated during childhood. **C,** Profile and occlusal views when the patient was 9 years old, with expected catch-up growth.

it has approximately 90% penetrance, and it is variably expressed (see Fig. 4-17). Manifestations of the syndrome outside of the oral and facial regions are unusual. Generally, the pits are characterized as depressions that are observed on the vermilion border of the lower lip; they are usually bilateral and symmetrically placed, although an asymmetrical pit or single central pit may occur. Roughly 33% of patients with van der Woude syndrome have pits without clefting, 33% have pits with cleft lip and palate, and 33% have lip pits with isolated cleft palate or submucous cleft palate. Approximately 10% of those individuals with van der Woude syndrome do not exhibit lip pits.

Stickler Syndrome

In 1967, Stickler and Pugh were the first to describe a series of patients who had a combination of eye findings, hearing loss, isolated cleft palate, a Marfanoid habitus, and long-bone changes.[1] It is now known that *Stickler syndrome* is associated with basic defects in collagen. These collagenopathies include mutations on type II collagen and mutations in type XI collagen.[25] The inheritance pattern with this syndrome is autosomal dominant with variable expressivity. Cohen and others have reviewed the relationship between high myopia and clefting of the secondary palate and the other features of Stickler syndrome.[19] High myopia (i.e., 8 to 18 diopters) is found in 75% of patients by the time they are 5 years old. The eye findings are progressive, with vitreous and chorioretinal degeneration and retinal detachment observed in 70% of patients by the time they are 20 years old. Additional eye findings include astigmatism, cataracts, strabismus, and glaucoma. Individuals with Stickler syndrome may manifest any or all of a range of craniofacial findings, including mild to moderate midface hypoplasia, shallow orbits with eye proptosis, epicanthal folds, a flat nasal bridge, and submucous or full clefting of the secondary palate. Some affected individuals will have progressive degeneration of the dental roots and the associated periodontium. This is primarily due to the inborn error in collagen production, but it may be exacerbated by malocclusion (secondary trauma) and orthodontic manipulations (see Fig. 4-18). Progressive neurosensory high-tone hearing loss has been reported in 80% of patients. Some patients have a Marfanoid body habitus, whereas others have short stature. There may be progressive early joint degeneration by the mid-adult years. Any newborn who is found to have Robin sequence should undergo an initial genetic and ophthalmologic assessment to rule out Stickler syndrome.

Robin Sequence

Robin sequence is commonly defined as cleft palate, micrognathia, and glossoptosis (see Fig. 4-19). This sequence of events can occur as a result of a variety of etiologic and pathogenetic conditions, and it involves a wide spectrum of phenotypes. The distinction must be made between a malformed mandible (micrognathia) and a deformed mandible that is simply retrognathic. The malformed mandible (i.e., Treacher Collins syndrome) may also be missing parts or sections of the jaw (i.e., Kaban types IIB and III) and cannot be expected to "grow out" and self-correct. This is different from a deformed mandible (i.e., retrognathic at birth) caused by fetal malpositioning, which can be expected to "grow out" and become relatively normal. The newborn's respiratory compromise that occurs with Robin sequence will vary according to the child's physical findings, and treatment should proceed accordingly. For example, with spondyloepiphyseal dysplasia congenita, the causes of respiratory compromise include a small and mechanically abnormal chest, tracheobronchial malacia, and central apnea as a result of cervical or medullary compression caused by cervical instability.[47] This will require a different level of airway management as compared with an isolated retrognathic mandible, which is simply deformed as a result of intrauterine compression. If the deformed (retrognathic) mandible is present at birth, it is expected to "catch up" during growth and basically to self-correct. For these infants, mandibular osteotomies with advancement carried out during childhood should be avoided.

Achondroplasia

Achondroplasia is the most common condition associated with severe disproportionate short stature.[22] The cause of this condition is the failure during development of the primary growth cartilages of the limbs and the cranial base. This results in short arms and legs as well as a characteristic midface deficiency or deformity that is most visually notable at the nasofrontal process.

Most affected individuals do well but attain a final height of only approximately 4 feet. The condition is known to be caused by mutations on the *FGFR3* (fibroblast growth factor receptor 3) gene.[22] Eighty percent of reported cases are sporadic, whereas 20% are familial. Although craniosynostosis is not a typical feature, three cases with craniosynostosis have been reported.

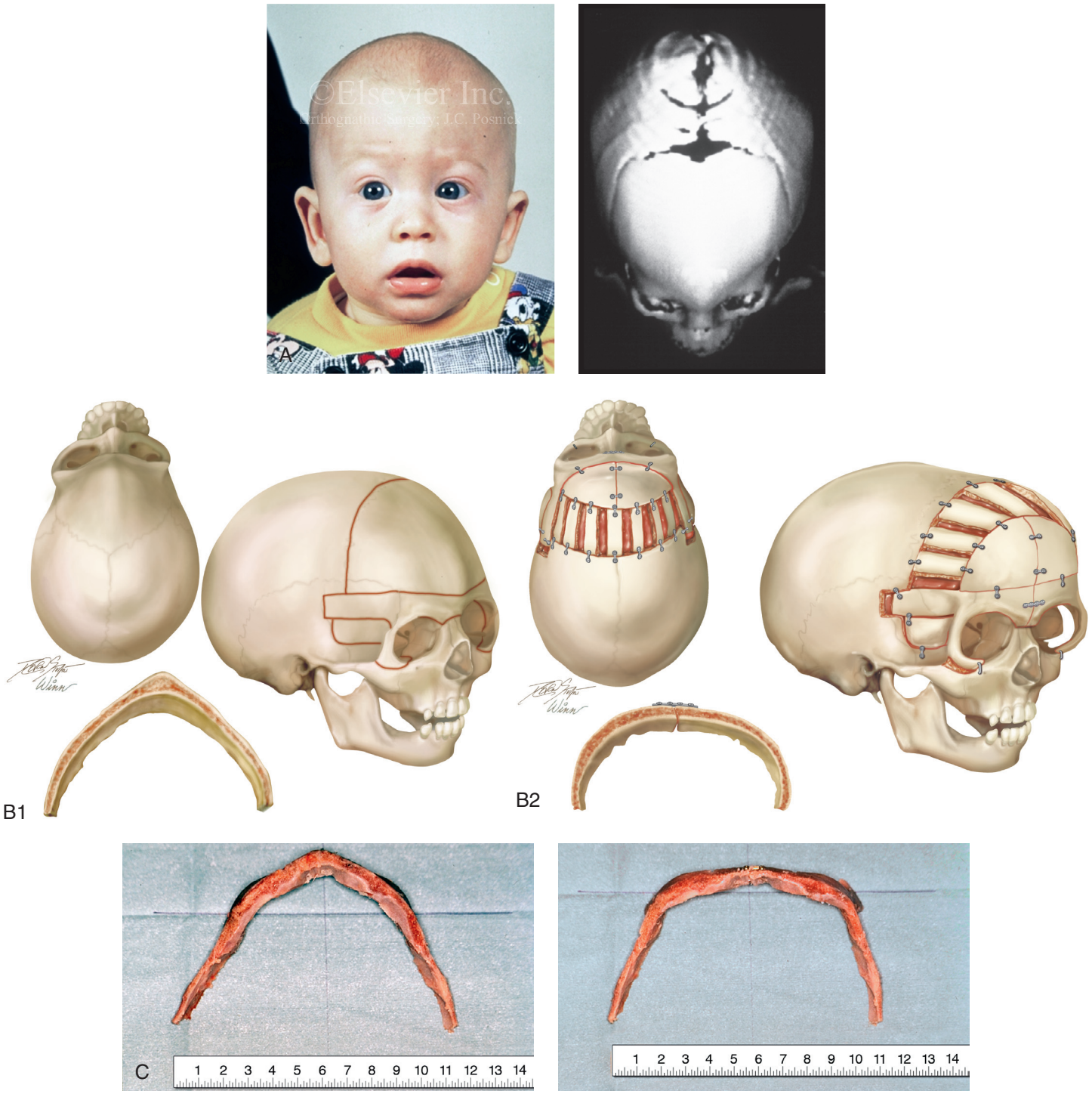
Premature Suture Closure of the Cranial Vault and Skull Base

The flat bones of the cranial vault develop from mesenchymal condensations over the developing brain (see Fig. 4-1). These bones grow primarily via the apposition of bone at their edges. The regions in which the cranial bony edges collide are called *sutures.* In addition to appositional growth at the edges (i.e., suture growth), cranial vault development also occurs through the process of remodeling (i.e., resorption and deposition).[17] Continued separation of the sutures during the process of the apposition of bone at the edges is also important to the normal growth of the bones of the cranial base and the midface.

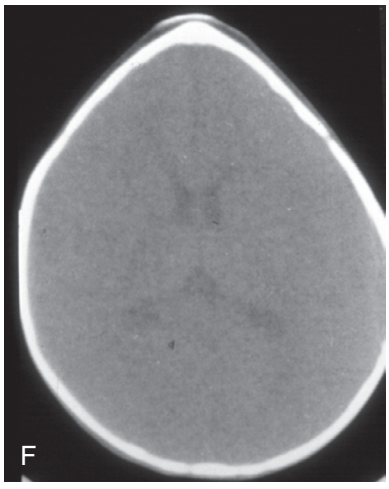
Synostosis is a condition of premature fusion (i.e., the arrest of appositional growth at the bone edges) that occurs before the associated visceral structures (e.g., brain, eyes) complete growth.[17] When this happens, there is distortion

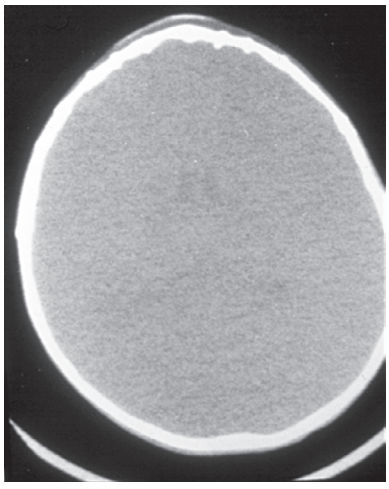
or deformity of the shape of the affected bones and possible compression of the underlying visceral structures (i.e., the brain). Premature closure of the cranial vault sutures (craniosynostosis) that do not extend into the cranial base will have minimal effects on the maxillofacial form (e.g., metopic, unilateral coronal, and sagittal suture synostosis; Figures 4-20, 4-21, and 4-22). However, prematurely fused cranial vault sutures that extend into the cranial base will also affect midfacial growth and development (e.g., Crouzon, Apert, and Pfeiffer syndrome; Figures 4-23 through 4-26; see Chapter 30).

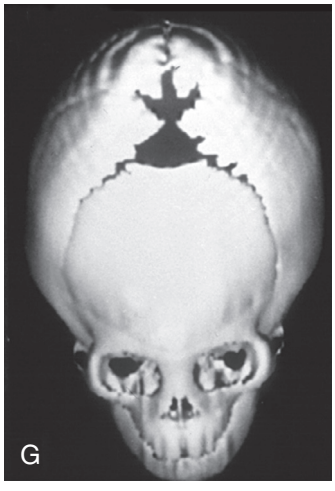
Text continued on p. 102



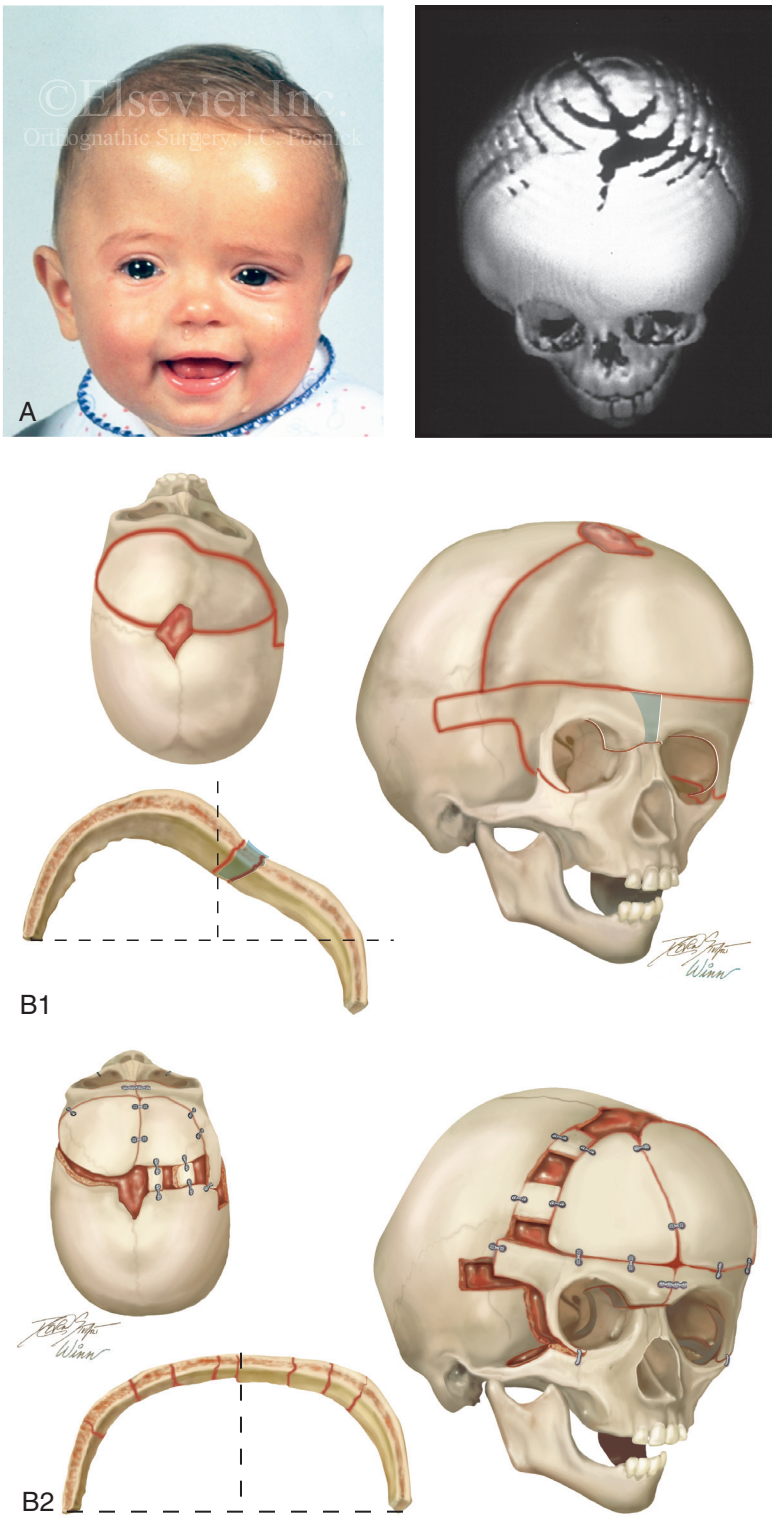
• **Figure 4-20** A child born with metopic synostosis that resulted in trigonocephaly. He underwent anterior cranial vault and three-quarter orbital osteotomies with reshaping when he was 10 months old. **A,** Facial and computed tomography scan views before surgery. **B,** Illustrations of the craniofacial skeleton in a child with metopic synostosis that resulted in trigonocephaly before and after anterior cranial vault and three-quarter orbital osteotomies. **C,** Bird's-eye view of removed orbital osteotomy unit before and after reshaping. *Part B modified from an original illustration by Bill Winn.*







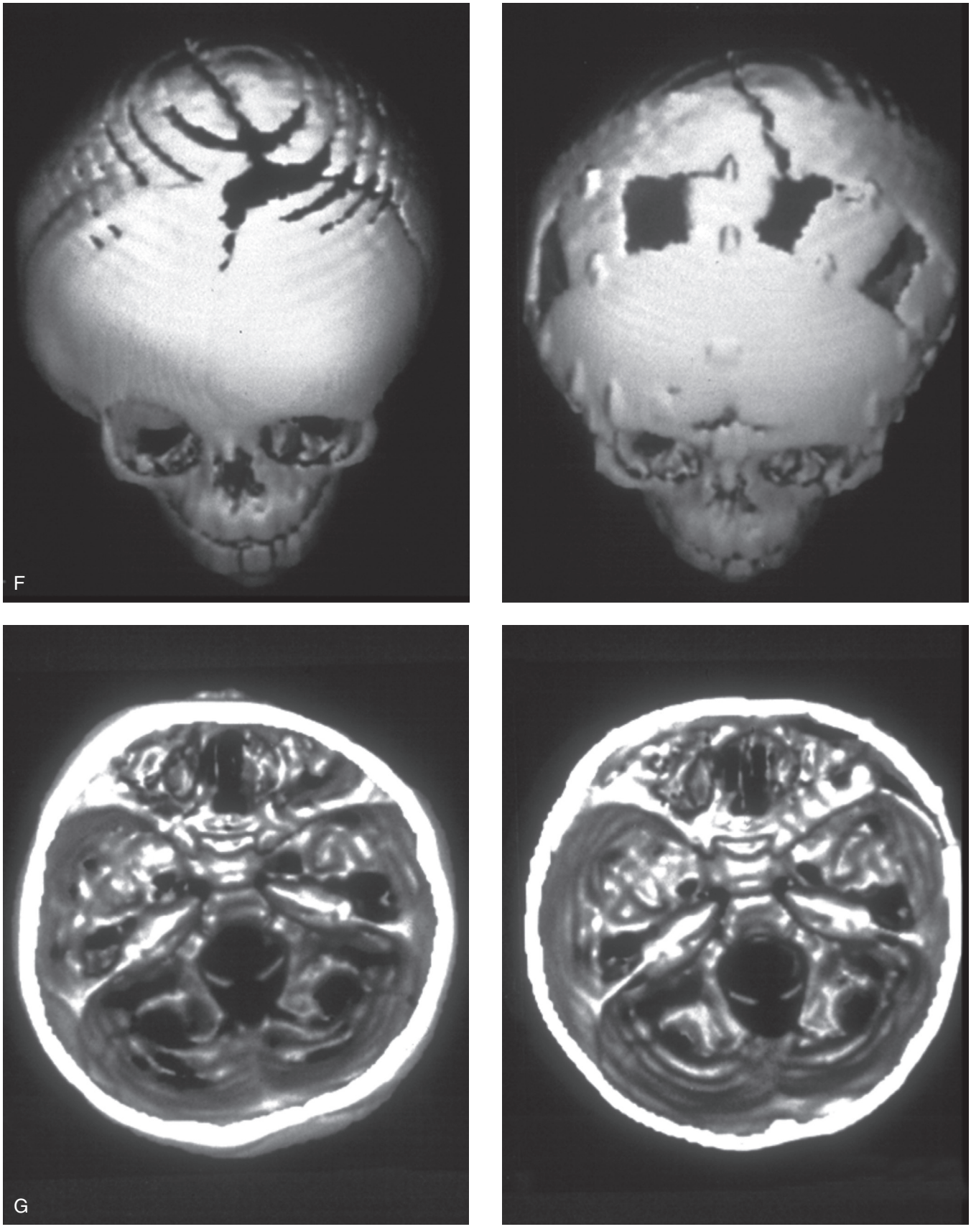
• **Figure 4-20, cont'd** **D,** Frontal views before and 1 year after reconstruction. **E,** Facial views of patient at 5 years old. **F** and **G,** Computed tomography scan views before and after reconstruction.



• **Figure 4-21** A child born with right unilateral coronal synostosis that resulted in anterior plagiocephaly. He underwent anterior cranial vault and three-quarter orbital osteotomies with reshaping when he was 9 months old. **A,** Facial and computed tomography scan views before reconstruction. **B,** Illustrations of the craniofacial skeleton in a child with unilateral coronal synostosis that resulted in anterior plagiocephaly before and after anterior cranial vault and three-quarter orbital osteotomies. *Part B modified from an original illustration by Bill Winn.*

• **Figure 4-21, cont'd C,** Bird's-eye view of removed orbital osteotomy units before and after reshaping. **D,** Frontal views before and 5 years after reconstruction. **E,** Oblique views 5 years after reconstruction.

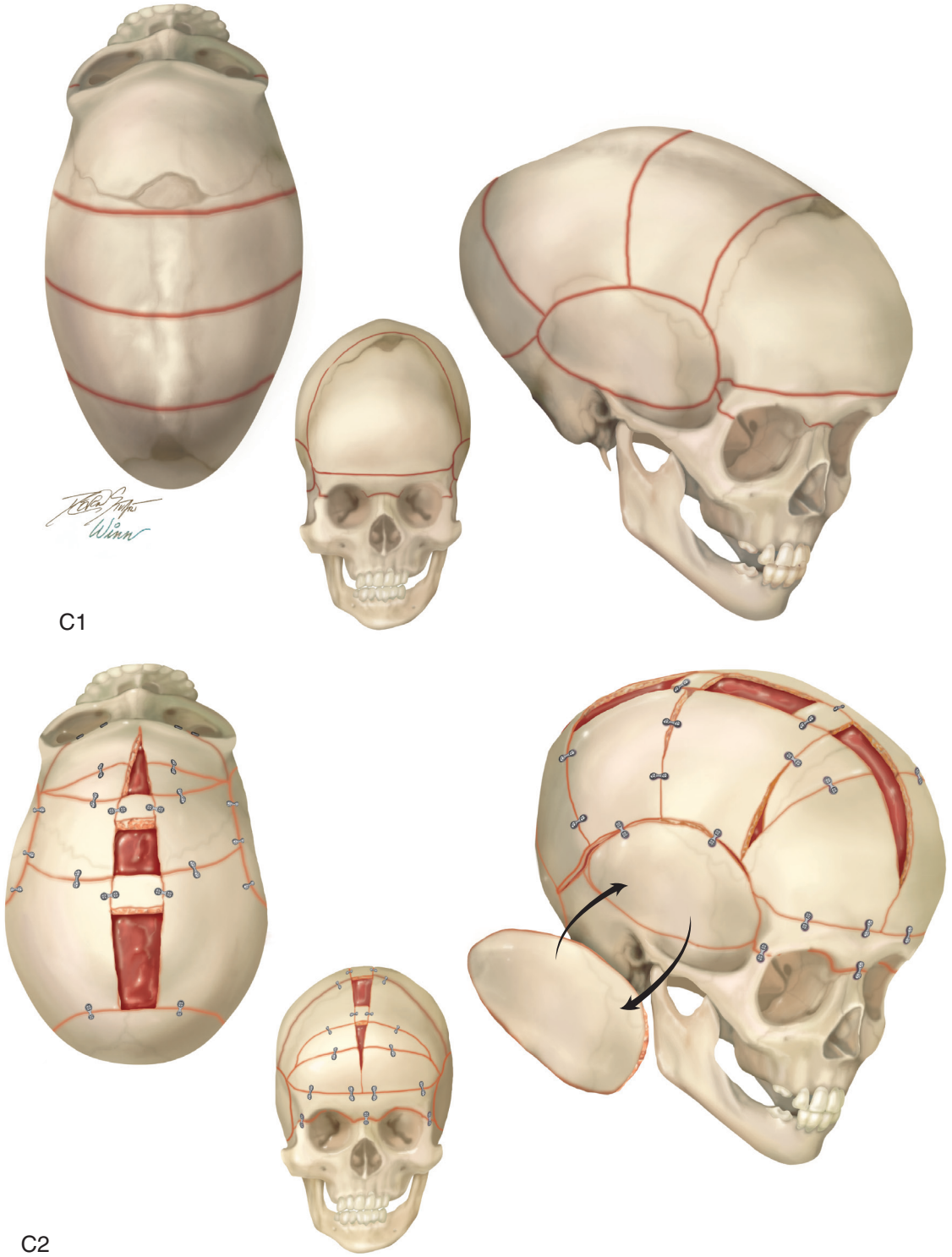
Continued



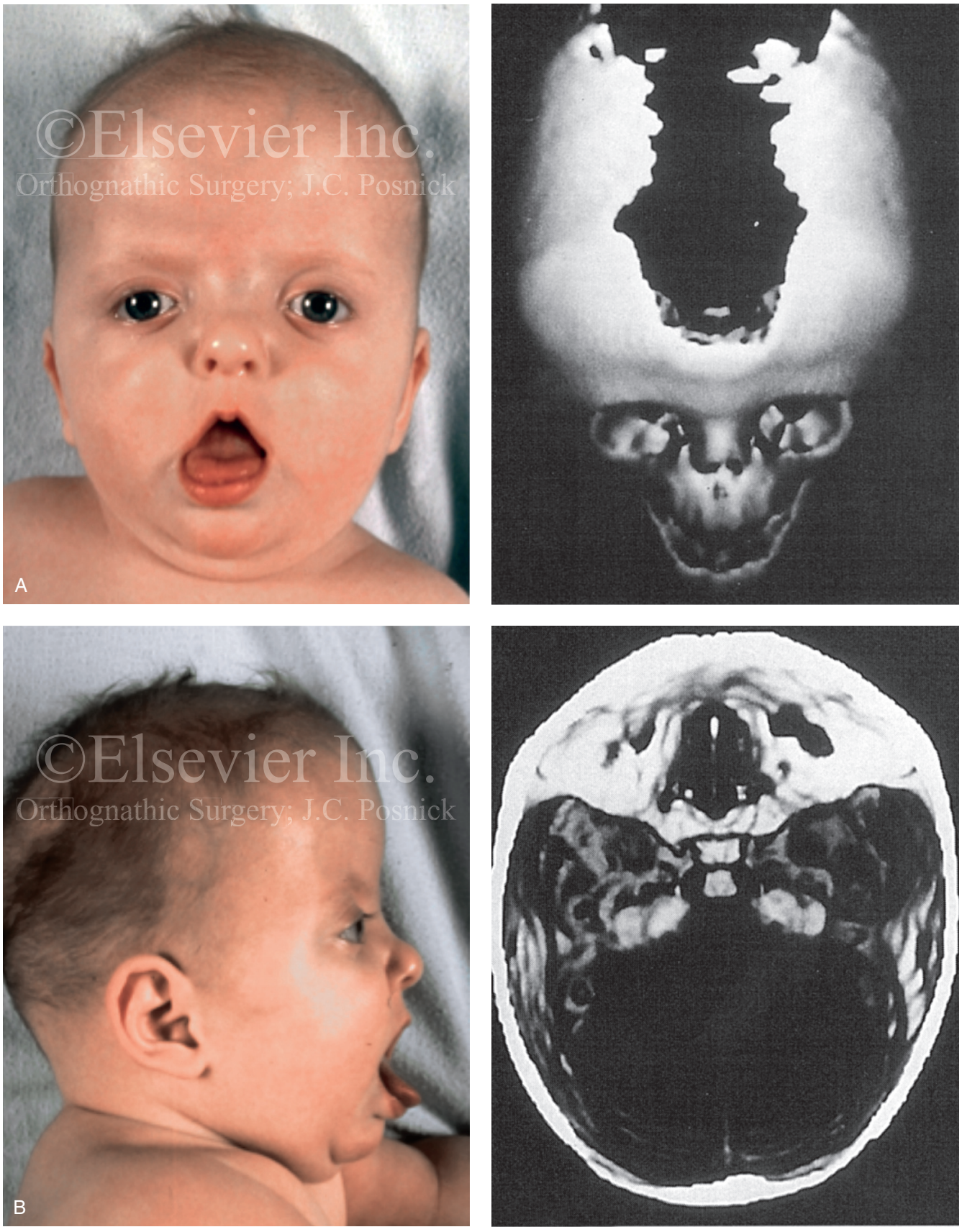
• **Figure 4-21, cont'd** **F** and **G,** Computed tomography scan views of the cranial vault before and just after reconstruction.

• **Figure 4-22** A child born with sagittal synostosis that resulted in a scaphocephalic shape of the cranial vault that was characterized by increased anteroposterior length and decreased bitemporal and biparietal width. It was not until the child was 2 years old that he was diagnosed with sagittal synostosis. After a comprehensive evaluation, he underwent total cranial vault reshaping through a coronal (scalp) incision. He is shown before and after a single-stage reconstruction. **A,** Frontal facial views before and after reconstruction. **B,** Profile facial views before and after reconstruction.

Continued



• **Figure 4-22, cont'd C,** Illustrations of the craniofacial skeleton in a child with sagittal synostosis that resulted in scaphocephaly before and after total cranial vault and upper orbital osteotomies with reconstruction. *Part C modified from an original illustration by Bill Winn.*



©Elsevier Inc.
Orthognathic Surgery; J.C. Posnick

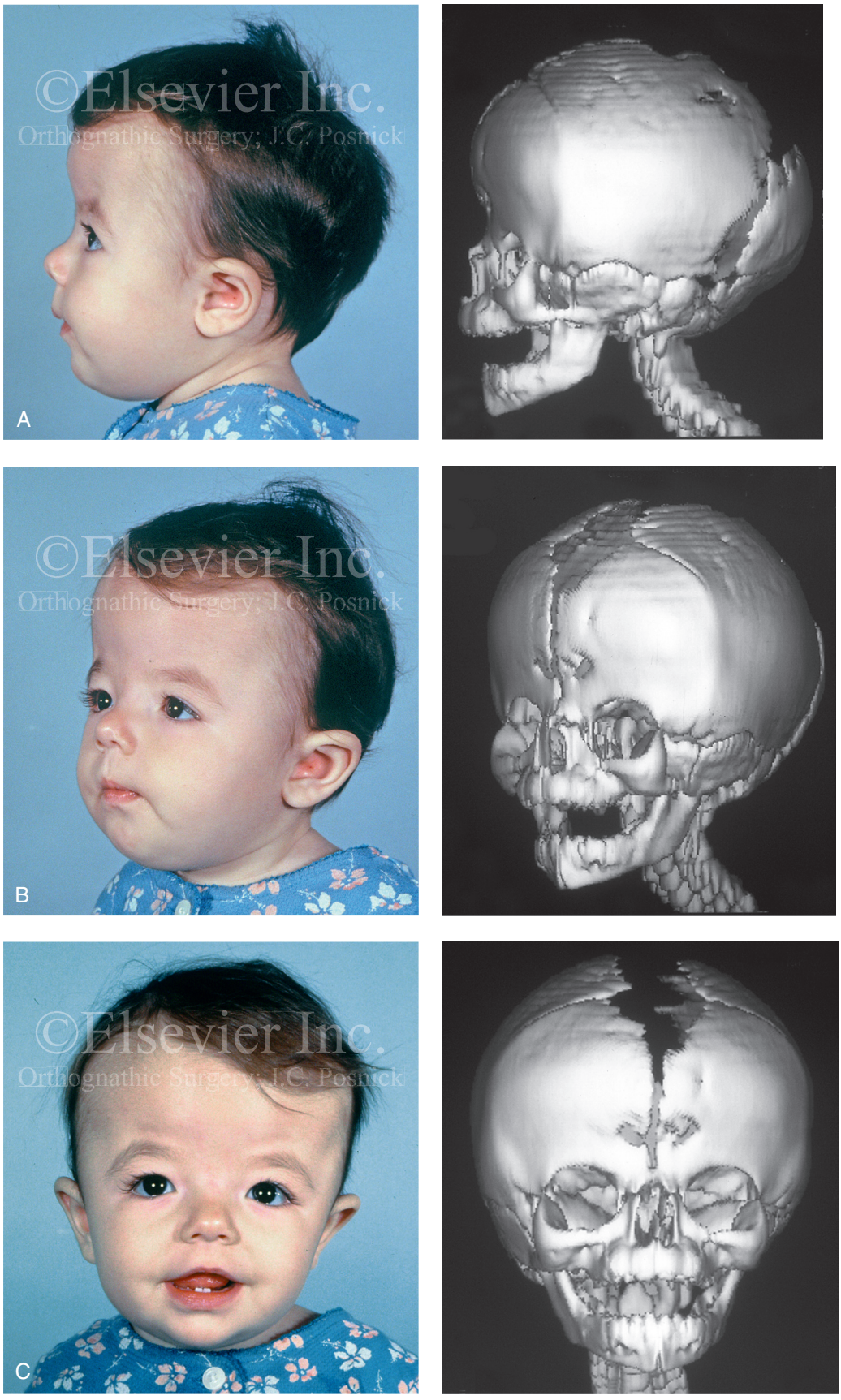
• **Figure 4-23** A 6-month-old girl who was born with Apert syndrome and bilateral coronal synostosis. **A,** Facial and computed tomography scan views without intervention. **B,** Profile and computed tomography scan views without intervention.

©Elsevier Inc.
Orthognathic Surgery; J.C. Posnick

• **Figure 4-24** A 28-year-old woman with uncorrected Apert syndrome. She was unable to undergo craniofacial or extremity reconstruction in her home country. Facial and extremity views are shown.

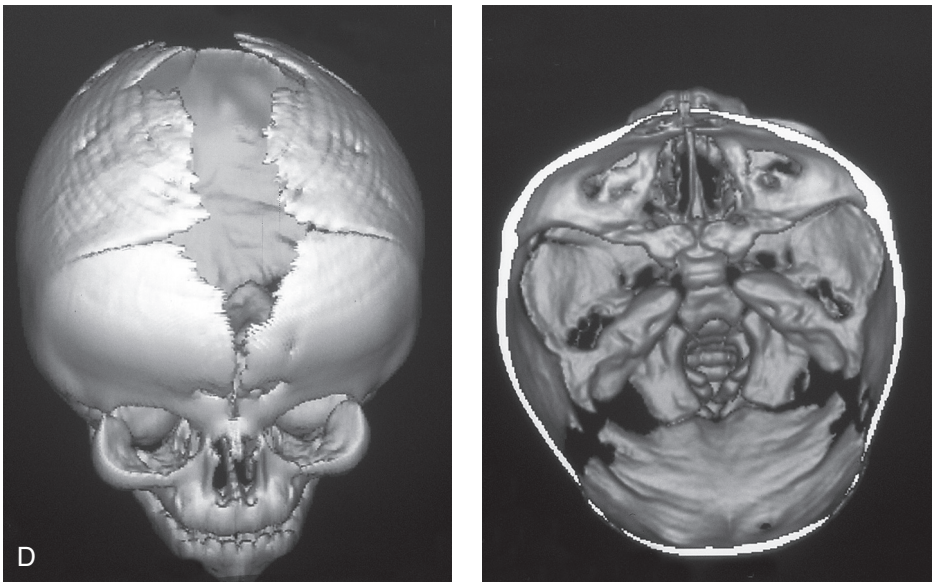
©Elsevier Inc.
Orthognathic Surgery; J.C. Posnick

• **Figure 4-25** A 50-year-old man who was born with Crouzon syndrome. His dysmorphic features include oxycephaly, orbital dystopia with eye proptosis, and midface deficiency with an Angle Class III malocclusion. Despite these difficulties, he is married and employed as a taxicab driver; he also has a son with Crouzon syndrome.



• **Figure 4-26** A 2-month-old child who was born with Pfeiffer syndrome (type I). She has bilateral coronal synostosis that resulted in brachycephaly but without the suggestion of midface deficiency. **A,** Profile facial and computed tomography (CT) scan views before intervention. **B,** Oblique facial and CT scan views before intervention. **C,** Frontal facial and CT scan views before intervention.

Continued



• **Figure 4-26, cont'd D,** Additional CT scan views before intervention.

Overgrowth Syndromes and Anomalies

Acromegaly is caused by an anterior pituitary tumor that secretes excessive growth hormone after the normal completion of growth.[21] The response of specific growth centers to abnormal serum levels of growth hormone is the overdevelopment of the hands, the feet, the supraorbital ridges, and the mandible. When there is excessive mandibular growth, the position of the teeth within the bone and the occlusion are also effected. An enlarged sella turcica (i.e., the resorption of bone in response to pituitary tumor expansion) can be seen on a lateral cephalometric radiograph or a computed tomography scan (Fig. 4-27). Excess growth hormone causes the proliferation of the condylar cartilage of the mandible, thereby resulting in the bilateral symmetric forward projection of the jaw. This overgrowth continues until the serum growth hormone levels return to normal. Unfortunately, the resultant skeletal deformities persist. Other examples of maxillomandibular overgrowth include hemifacial hypertrophy (see Chapter 31), hemimandibular hyperplasia, and hemimandibular elongation (see Chapter 22).

Hereditary Tendencies in the Development of Dentofacial Deformities

Jaw deformities are thought to be more common among individuals with genetic backgrounds that are crosses between different racial and ethnic groups as compared with those from isolated human populations.[10,32,40,57,59,60,62,83,85,86,91,93,108] It has been documented that crossing different breeds of dogs and other mammals can result in striking dentofacial deformities, particularly mandibular prognathism that resembles the human variant.[82,117,119] Harris and Johnson examined the longitudinal records from the Bolton Brush Growth Study and concluded that the heritability of maxillomandibular characteristics is relatively high[48] (Figures 4-28, 4-29, and 4-30). It is assumed that approximately one third of children who present with a severe Class III malocclusion will have a parent with the same problem and that one sixth of these children will have an affected sibling.[73] Interestingly, a short face growth pattern with a deep-bite malocclusion is found more frequently in whites (see Chapter 23). Bimaxillary dento-alveolar protrusion with anterior open-bite malocclusion is more prevalent among blacks (see Chapter 24).[112]

The Class III skeletal pattern of maxillary deficiency with relative mandibular excess is known to be more frequent in certain races.[2,73,84,123,139] Incidence rates of Class III malocclusion of as high as 13% have been reported in specific regions of Asia.[136] Lew and colleagues examined occlusal parameters in 1050 Chinese school children between the ages of 12 and 14 years. They found an incidence of 12.6% for Class III malocclusion, 58.8% for Class I malocclusion, and 21.5% for Class II malocclusion.[67] Samman and colleagues investigated 300 Chinese individuals residing in Hong Kong who were known to have a dentofacial deformity and who had been referred to an "orthognathic surgery clinic." After excluding those individuals with cleft lip and palate, 222 study patients remained. Forty-seven percent of these individuals (n = 104) were found to have a Class III skeletal growth pattern.[106]

Several of the common maxillomandibular disharmonies are known to result from environmental influences (see Chapters 10 and 21). The dental position in each arch and the resulting occlusal variations are felt to have an even greater environmental component.[3,11,29,41,64,92] For example,

• **Figure 4-27** A middle-aged woman who presented for the orthodontic evaluation of a progressive malocclusion. She had a normal occlusion, average facial features, and no distortions of the extremities earlier during life. Gradually, over the prior 5 years, malocclusion and changes in facial and extremity morphology had occurred. At presentation to the orthodontist, standard records were taken (lateral cephalometric and Panorex radiographs and dental models). The patient was then seen for surgical evaluation. A review of the lateral cephalometric radiograph showed an enlarged sella turcica. An endocrinology consult confirmed the diagnosis of acromegaly. Appropriate treatment was undertaken without further worsening of the facial or extremity deformities. **A,** Facial and occlusal views at presentation indicate growth hormone changes. **B,** Profile and lateral cephalometric views at presentation. Note the enlarged sella turcica. **C,** A view of the dorsal aspects of hands indicates growth hormone changes.

• **Figure 4-28** Biologic identical twin sisters are shown during their teenage years to confirm a hereditary tendency toward the occurrence of developmental jaw deformities with malocclusion. **A,** Facial and occlusal views of Twin A demonstrating bimaxillary dental protrusion with jaw disharmony and anterior open-bite malocclusion. **B,** Facial and occlusal views of Twin B demonstrating bimaxillary dental protrusion with jaw disharmony and anterior open-bite malocclusion.

dento-alveolar morphology is commonly affected by thumb sucking and other para-oral habits (i.e., environmental influences; Figures 4-31 and 4-32). The more severe the dentofacial deformity, the more likely that inheritance plays at least a partial role. Maxillary deficiency with relative mandibular excess (i.e., a Class III skeletal pattern) and maxillomandibular deficiency (i.e., the short face growth pattern) are both thought to have a significant hereditary component.[31,36,58,79,116,120,137]

Environmental and Neuromotor Effects on Skeletal Growth

The *mandible* can be viewed as a somewhat malleable block of hard tissue that is adjacent to the skull base. It is separated from the skull by working temporomandibular joints. It houses the inferior alveolar neurovascular bundle, and it provides a matrix for the formation, eruption, and eventual alignment of the teeth. Muscular activity in the head and neck can directly affect skeletal growth through one of the following mechanisms: 1) hyperactivity of muscles at the point of attachment may cause excessive skeletal remodeling or contour deformities (as discussed later in this chapter) 2) hypoactivity of the masticatory muscles may cause distortions of the normal skeletal growth (i.e., long face growth pattern; Fig. 4-33) and 3) hyperactivity of specific muscles such as the sternocleidomastoid (i.e., torticollis) may result in the deformation of the growing bones (i.e., cranial vault, orbit, and zygoma; Fig. 4-34).[71]

Muscle and Soft-Tissue Effects on Skeletal Growth

Experimental animal research and clinical observational studies confirm that the influences of the *mandibular rest posture,* the *neck rest posture,* and the *tongue and lip rest position* of the affected individual's eventual facial morphology are real and more important than the effects of intermittent muscle contracture (see Chapters 8 and 10).[128-130] It is also known that functional stresses that are placed on the skeleton will increase bone density.[113,114,118] A corollary is that, during periods of inactivity (e.g., mandibular and neck rest posture), skeletal demineralization occurs.

Erupting teeth carry alveolar bone with them. Therefore, forces that counteract the normal eruption of teeth can shape the dental arches and the alveolar processes of both the maxilla and the mandible.[5,101-105,107,109,111,126,140] Experimental studies confirm that pressure against the active eruption of the teeth that is maintained for 4 to 6 hours can affect tooth movement and initiate bone remodeling (see Chapter 5). Habits such as *finger or thumb sucking,* which apply consistent pressure against the teeth, can have the

Text continued on p. 113

• **Figure 4-29** A father and his biologic daughters are shown during the daughters' teenage years to confirm a hereditary tendency toward the occurrence of developmental jaw deformities with malocclusion. **A,** Facial and occlusal views of the father that demonstrate a Class III skeletal pattern.

Continued

• **Figure 4-29, cont'd** **B,** Daughter A with a similar class III skeletal pattern. **C,** Daughter B with a similar Class III skeletal pattern.

• **Figure 4-30** Biologic twin sisters are shown during their teenage years to confirm a hereditary tendency toward the occurrence of developmental jaw deformities with malocclusion. **A,** Facial and occlusal views of Twin A demonstrating a long face Class III growth pattern. **B,** Facial and occlusal views of Twin B demonstrating a similar long face Class III growth pattern.

©Elsevier Inc.
Orthognathic Surgery; J.C. Posnick

• **Figure 4-31** A 10-year-old girl with a severe anterior open bite. She demonstrates how a para-oral habit (thumb sucking) can exert the long duration of light force at the periodontal ligament that is required to move the teeth. This patient was a consistent and constant "thumb sucker" from soon after her birth and throughout the mixed dentition.

©Elsevier Inc.
Orthognathic Surgery; J.C. Posnick

• **Figure 4-32** A 12-year-old girl was referred by an orthodontist for surgical evaluation. She had persistently sucked her thumb since infancy. This parafunctional habit resulted in the severe deformity of her maxilla and mandible. The upper jaw has a constricted arch width and an excessive curve of Spee, whereas the mandible has a Class III reversed curve of Spee. A severe anterior open-bite malocclusion is also present.

©Elsevier Inc.
Orthognathic Surgery; J.C. Posnick

• **Figure 4-33** A teenage girl with a lifelong history of obstructed nasal breathing demonstrates the resulting long face growth pattern. The nasal obstruction resulted in forced mouth breathing with an open-mouth posture. This in turn is associated with maxillary dental hypererruption (i.e., hypererruption of the molars greater than the incisors), a steep mandibular plane, anterior open-bite Class II malocclusion, and increased lower anterior facial height. Clinically, there is a "gummy" smile, lip incompetence, and a flat, long face. **A,** Frontal views with smile before and after reconstruction. **B,** Profile views before and after reconstruction.

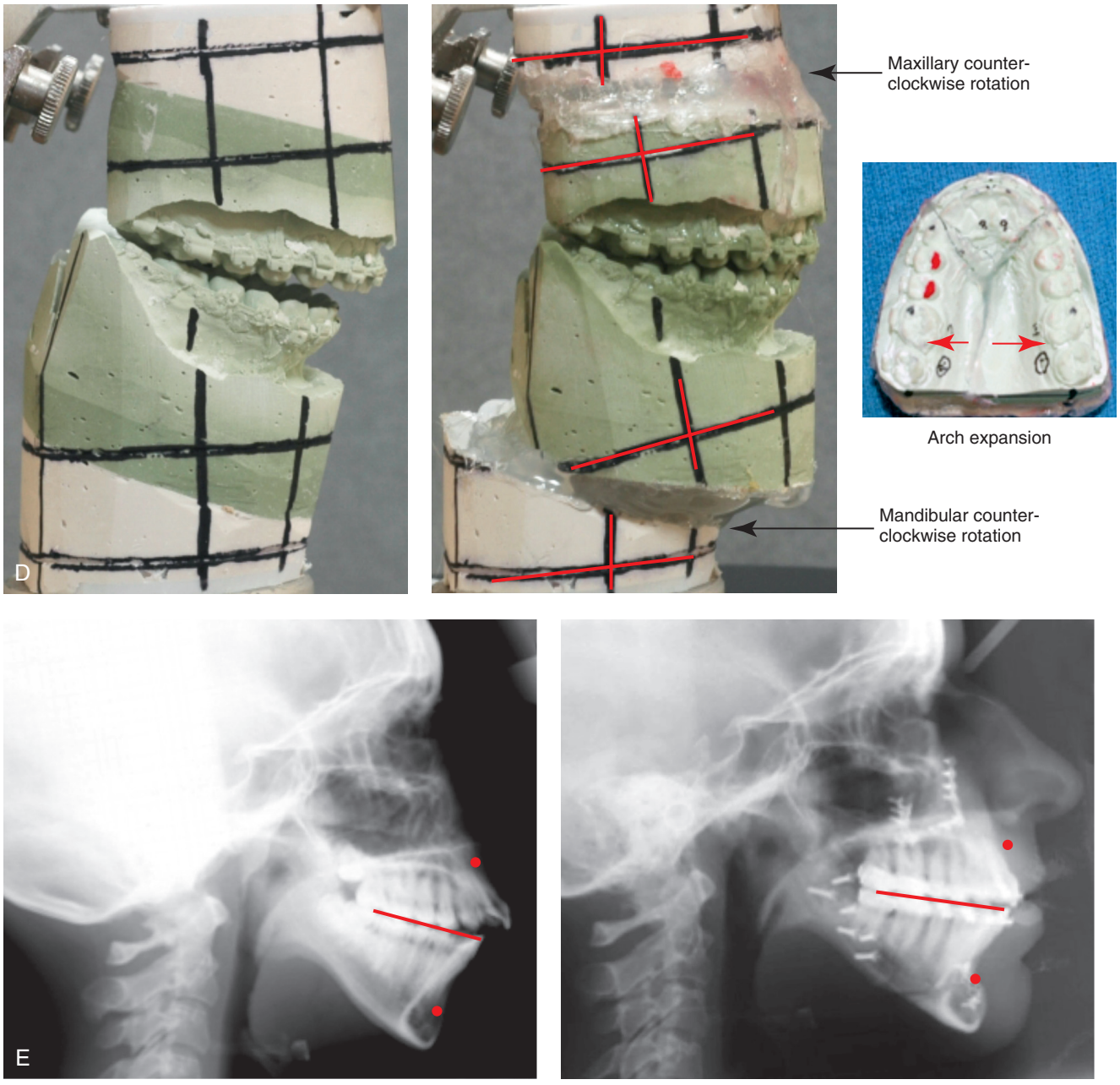
Continued

Prior to retreatment

Pre surgery

After treatment

• **Figure 4-33, cont'd C,** Occlusal views before retreatment, with orthodontic decompensation in progress, and after the completion of treatment.



• **Figure 4-33, cont'd** **D,** Articulated dental casts that indicate analytic model planning. **E,** Cephalometric radiographs before and after surgery.

• **Figure 4-34** A child with right-sided congenital muscular torticollis and C1-C2 rotary subluxation. Ipsilateral right fronto-orbital flattening and contralateral left occipitoparietal flattening were noted. The right sternocleidomastoid muscle (SCM) remained tight when the patient was 2 years old. He underwent surgical release of the SCM, and this was followed by neck range-of-motion exercises with good maintenance of function. A degree of right fronto-orbital flattening (skull molding) remains. **A,** Frontal view with the neck in a relaxed position before treatment indicated a head tilt. Computed tomography scan views indicate fronto-orbitozygomatic asymmetry as a result of skull molding. **B,** Intraoperative view of the tight SCM and the proposed incision site, and a close-up view of the exposed SCM before release. **C,** Frontal views before and 2 years after neck muscle release. Residual fronto-orbital (skull molding) flattening remains. *From Slate RK, Posnick JC, Armstrong DC, et al: Cervical spine subluxation associated with congenital muscular torticollis and craniofacial asymmetry.* Plast Reconstr Surg *91:1187–1195, 1993.*

same effect on the shaping of the dental arches (see Figs. 4-31 and 4-32). It seems as though the intensity (i.e., the amount of force) against the teeth is less important than the duration (i.e., the number of hours per day) with regard to causing this effect.

It has been confirmed that the *postural position of the tongue at rest* (e.g., the mandible open with the constant protrusion of the tongue) rather than where the tongue is placed during function (e.g., a tongue-thrust swallowing habit) is most important for determining the eventual position of the teeth and the morphology of the dento-alveolar complex.[61,121] The intermittent forces that are generated against the teeth during chewing, swallowing, and speaking are documented to be more than heavy enough to produce tooth movement. However, during these normal functions, the force is not maintained long enough at any point in time to have an appreciable effect on the position of the teeth or the morphology of the bone.[35] Even repetitive intermittent "abnormal" tongue and lip contraction with resulting pressure on the teeth during swallowing and speaking (i.e., tongue thrusting)—despite their frequency—do not sum up to the number of hours of force required to produce a pathologic effect. By contrast, even very light forces sustained for a long duration (i.e., the tonic contracture of masticatory muscles to maintain mandibular posture) can affect both the path of tooth eruption and jaw development.[8,94]

It is known that muscles of different size and force that attach to the mandible at specific locations will result in characteristic ridges and contours (e.g., gonial angles, coronoid process, genial tubercle).[12,13] For patients with the condition known as *masseteric muscle hypertrophy*, there is excess bone proliferation at the masseter muscle's point of attachment along the inferior border of the mandible (i.e., the gonial angle ridge) in response to greater than average muscle forces.[43] By contrast, in patients with *muscular dystrophies* (i.e., hypotonic cerebral palsy, Moebius syndrome with cranial nerve palsy), the ramus may be short, and the gonial angles appear to be underdeveloped as a result of the limited muscle forces applied to these same regions.[100]

The lips, cheeks, and tongue contain muscles in their deep layers that exert varied pressures against the teeth and the alveolar process. They normally do so with a constant level of tone while at rest as well as during dynamic function. These light (tone) forces applied for a long duration are known to cause tooth movement and remodeling of the alveolar process. It is the "resting" postural pressures of these structures (i.e., lips, cheeks, tongue, and muscles), the presence of para-oral habits (i.e., thumb sucking, pencil biting; see Figs. 4-31 and 4-32), and the man-made and controlled orthodontic pressure that can exert the long duration of light force at the periodontal ligament that is required to move teeth.[55] When the difference between applied orthodontic forces and pressures of the tongue, cheek, and lips that counteract those forces applied to the periodontal ligament becomes great enough for sustained periods of time, tooth movement will occur. Another example of dental adaptation is seen when the constant strong force of the tongue is juxtaposed with intrinsic weaker bilabial (i.e., upper and lower lip) muscle tone forces.[135] This results in the predictable dental compensations (i.e., proclined maxillary and mandibular incisors) observed in patients with *bimaxillary dento-alveolar protrusion* (see Chapter 24). This also explains the consistent dental compensations that are typically observed in patients with specific developmental dentofacial deformities such as long face growth pattern (see Chapter 21 and Fig. 4-33) or isolated mandibular deficiency (see Chapter 19).[122]

Severe *scar contracture of the anterior neck and lower lip* soft tissues from a burn that occurred during childhood will also affect mandibular morphology. The soft-tissue contracture will distort ongoing skeletal growth and cause the mandible to be retrusive, with an obtuse angle. The chin region of the mandible will grow with increased vertical length and without sagittal prominence, and there will be an anterior open-bite malocclusion. This deformity and its surgical correction were first described by Simon Hullihen in 1849 (see Chapter 2 and Fig. 2-19).

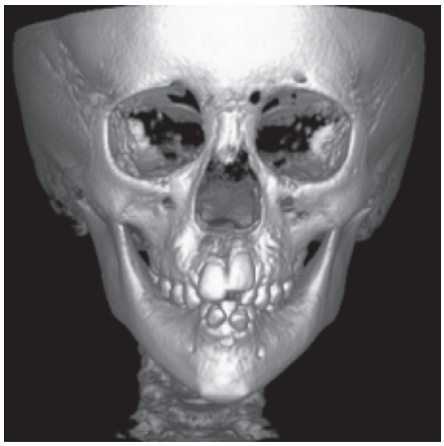
When an *electric burn to an oral commissure* occurs during childhood and results in soft-tissue contracture with microsomia, the constant soft-tissue forces against the teeth will distort the shape of the dental arch and the alveolar process. There will be anterior dental crowding, tipping of the incisors, and malocclusion.

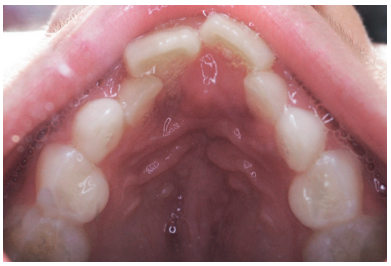
With the *congenital absence of part or most of the tongue,* there will be a characteristic V-shaped deficiency or dysmorphology of the mandibular dental arch (e.g., hypoglossia–hypodactylia syndrome; Fig. 4-35).[15] In the presence of a volumetrically enlarged tongue (e.g., vascular malformation, hemangioma, neurofibromatosis, Proteus syndrome, Beckwith-Wiedemann syndrome), there will be dento-alveolar effects that result in spaces between the teeth (Fig. 4-36). The lower anterior teeth will be flared, and the overall mandibular arch form will be enlarged. The important factor that causes these dental compensations and arch form deformities is the sustained and constant rest forces of the oversized tongue on the teeth and bone and not what the tongue does when it is in motion.

In the individual with a normal tongue volume and neuromotor capability, it is known that the rest position of the tongue in the mouth is primarily influenced by *respiratory factors*.[73] It is the rest position of the tongue that influences the pattern of dentofacial development rather than the position of the tongue during function.

In the presence of *chronic nasal obstruction,* a typical long face growth pattern with anterior open-bite malocclusion is likely to occur. In these circumstances, a speech therapist may have success with teaching the individual altered movement of the tongue for the formation of more accurate voluntary independent speech sounds.[90] However, unless the anterior open bite and the nasal obstruction are corrected, the myofunctional therapy will not allow the individual to reliably position his or her tongue, lips, teeth, and mandible for correct articulation during rapid conversational speech (see Chapter 8).

©Elsevier Inc.
Orthognathic Surgery; J.C. Posnick





Palatal view | Facial-occlusal view | Lingual view

• **Figure 4-35** A 10-year-old girl who was eventually diagnosed with hypoglossia–hypodactylia syndrome. In this photo, she demonstrates how the congenital absence of part or most of the tongue will result in a characteristic V-shaped deficiency and dysmorphology of the mandibular dental arch and how this will negatively affect the maxillary arch form.

©Elsevier Inc.
Orthognathic Surgery; J.C. Posnick

• **Figure 4-36** A 3-year-old child with a congenitally enlarged tongue is shown. The secondary effects on the oral cavity include a Class III anterior open-bite malocclusion and a lateral crossbite malocclusion.

In the skeletal Class III adult patient (i.e., a retrusive maxilla with relative mandibular excess) who undergoes successful orthognathic correction, a normal oral cavity volume and a normal occlusion are established. Interestingly, in most cases, the tongue position and lip posture will then naturally adapt to the new morphology.[80,81]

Clinical observation confirms that, when an individual with a longstanding dentofacial deformity undergoes successful reconstruction, it is generally not necessary to retrain the tongue with active myofunctional therapy. This only needs to occur under the following conditions: 1) when the tongue volume is truly excessive (e.g., vascular

malformation, neurofibromatosis) 2) when there is the presence of intrinsic neuromotor dysfunction (e.g., cerebral palsy) or 3) when the nasal airway remains obstructed and a failure of tongue adaptation is seen.[63]

Respiration Patterns and Their Effects on Skeletal Growth

Harvold conducted primate experiments that were designed to test hypotheses regarding *the relationship between nasal obstruction, mouth breathing, mandibular rest posture, tongue rest position, jaw growth, and dental malocclusion*.[49-54] He created an experimental model in growing rhesus monkeys *(Macaca mulatta)* to study these relationships. To do this, he completely obstructed the nasal passages in the study subjects with silicone nose plugs. The experiments showed that the monkeys adapt to total nasal obstruction in two different ways. Predominantly, the experimental animals maintained an open-mouth posture with tongue protrusion. Interestingly, some adapted to a forced oral airway by holding the upper and lower teeth close together but with wide lip separation. Nevertheless, both adaptive mandibular, tongue, and lip positioning patterns gradually resulted in maxillomandibular dysmorphology and dental malocclusion that was notably different from that seen in the control animals.

In the experimental animals, morphologic changes that occurred in the lips, tongue, dentition, jaws, and mandibular posture were documented by the research team through cephalometric measurements as well as through electromyographic and behavioral findings. Harvold's cephalometric findings documented changes in facial height (e.g., of the naso-palatal plane and the symphysis-palatal plane) and alterations in mandibular morphology.

As stated, a minority of animals adapted to the nasal airway blockage by holding their teeth together with wide lip separation (rather than maintaining an open-mouth posture with tongue protrusion) while still breathing through their mouths. This subgroup showed the least change in jaw morphology and dental alignment.

The observed morphologic and occlusal changes were most extreme when the mandible constantly remained open, with tongue protrusion (i.e., an open-mouth posture). The adaptive lowering of the mandible (with clockwise rotation) to achieve effective oral respiration was followed by secondary changes in the maxilla, including a downward displacement (i.e., vertical lengthening) of the alveolar process in conjunction with excess extrusion of the teeth. When molar (posterior) hypereruption was significantly greater than incisor hypereruption, an anterior open-bite malocclusion developed (Fig. 4-37).[127]

It became apparent to Harvold that the changes in mandibular shape and the direction of mandibular growth (i.e., clockwise rotation) observed in a majority of the experimental monkeys were dependent on the adaptive activity of the facial, tongue, jaw, and neck muscles in response to respiratory need. Interestingly, in Harvold's experimental animal model, there was no indication of abnormal bony apposition on the condyles in association with the forced oral respiratory pattern. The apposition of new bone on the condyles continued (in the usual way) independently of aberrant mandibular positioning or open-mouth posture.[107]

In review, the growing experimental animals studied by Harvold coped with nasal obstruction by changing neck, tongue, facial, and masticatory muscle tone, thereby resulting in alterations in mandibular posture and tongue position.[44,45,55] The animals typically did this in one of two different ways but always with the goal of maintaining adequate respiration. This resulted in varied but consistent secondary changes in the maxillomandibular morphology (i.e., developmental jaw deformity) and dental compensations (i.e., malocclusion). The measured lower facial height increase that occurred in the presence of a constant open-mouth posture was primarily the result of maxillary molar—and, to a lesser extent, incisor dental—hypereruption (e.g., long face growth pattern).

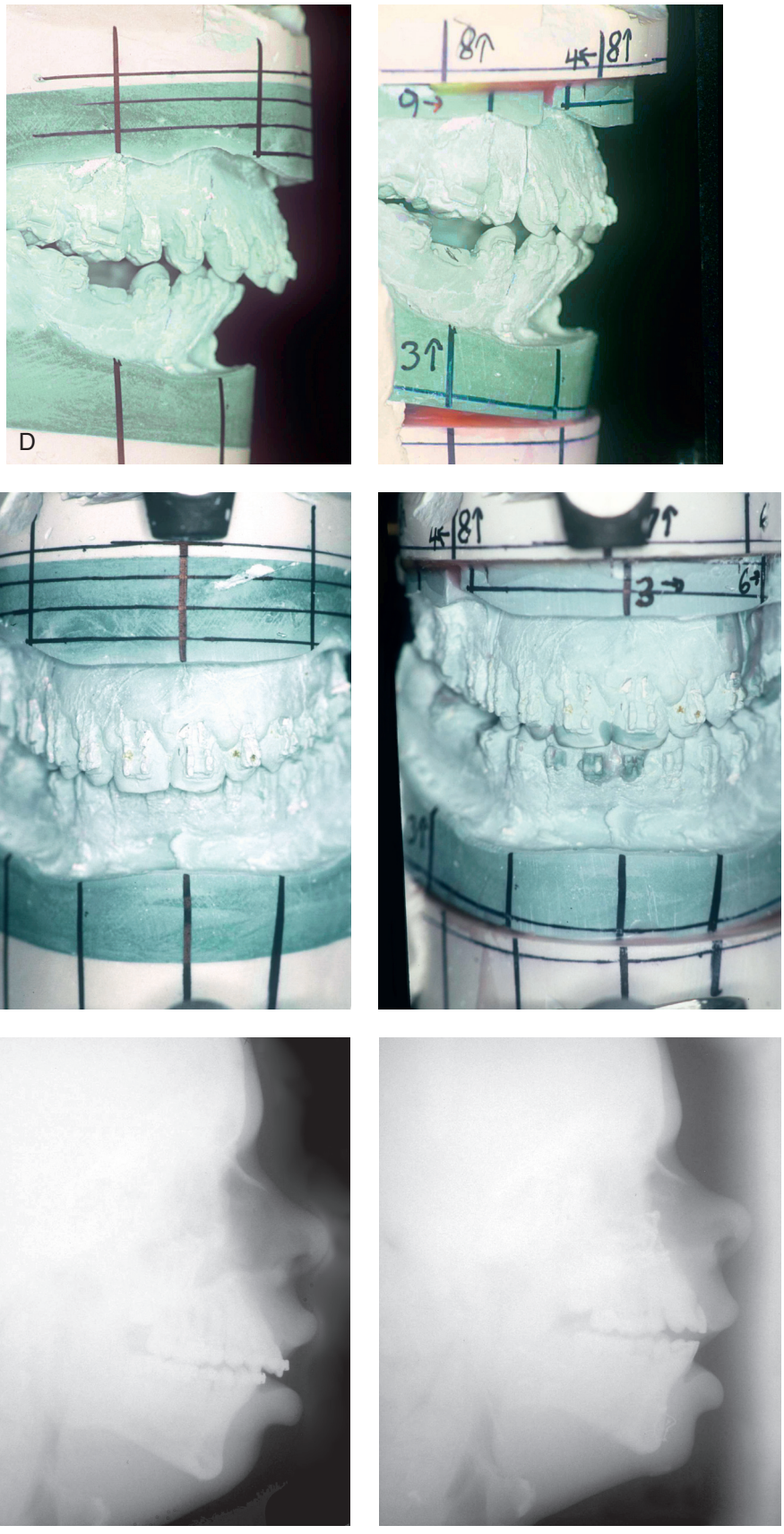
Harvold also reviewed the human observation studies of Linder-Aronson and colleagues and found that they paralleled his experimental animal research.[68-70,75-78] Their clinical research established that *nasal obstruction with forced mouth breathing* (i.e., open-mouth posture) in humans is frequently associated with maxillary dental extrusion, a steeper than normal mandibular plane, anterior open-bite malocclusion (i.e., the hypereruption of the molars is greater than that of the incisors), and increased lower anterior facial height. Similar to Harvold's experimental monkeys, growing humans may develop different neuromotor adaptations to cope with nasal obstruction and the need to maintain adequate respiration. The secondary skeletal and dental morphologic deviations will therefore vary, but they tend to occur in a predictable long face growth pattern (see Chapter 21).[39,125,133,134]

Humans with severe hypoplasia of the maxilla, a retropositioned palate and a restricted nasal airway, as are seen in patients with Crouzon or Apert syndrome, also become dependent on oral respiration (see Chapter 30).[4] With Crouzon or Apert syndrome, the individual's neck is often maintained in hyperextension with an open-mouth posture, the protrusion of the tongue, and the clockwise rotation of the mandible; this head and neck posture is compensatory to support respiration. During the day, this compensatory posture may be sufficient, but it becomes inadequate at night and results in obstructive sleep apnea (see Chapter 26).[4] When these compensatory adaptive changes occur during childhood, they contribute to additional secondary maxillomandibular skeletal dysmorphology with growth that further affects the occlusion (see Chapter 30).[7,33,115] This is an example of a congenital midface deformity that is further influenced by the environment.

Congenital Myopathies and Effects on Skeletal Growth

Individuals with *congenital myopathies* constitute a heterogeneous subgroup of patients with congenital

• **Figure 4-37** A 16-year-old with a nonprogressive congenital myopathy that resulted in a constant open-mouth posture. He developed a long face growth pattern that was characterized by severe maxillary vertical excess and protrusion, a retrusive mandible, and an Angle Class II malocclusion. He underwent a combined orthodontic and surgical approach. Orthodontic manipulation was limited as a result of hygiene control. The surgery included a maxillary Le Fort I osteotomy (vertical intrusion, first bicuspid extractions, and alveolar bone removal with premaxillary setback and cant correction) and sagittal split osteotomies of the mandible (clockwise rotation and asymmetric correction). **A,** Frontal views with smile before and after reconstruction. **B,** Oblique facial views before and after reconstruction. **C,** Occlusal views before and after reconstruction.



• **Figure 4-37, cont'd** **D** and **E,** Articulated dental casts that indicates analytic model planning. **F,** Lateral cephalometric radiographs before and after reconstruction.

neuromuscular disorders in which the pathology is attributable to defects in the muscle fibers rather than in the neurons. Congenital fiber-type disproportions are rare congenital myopathies that are considered slow or nonprogressive. Patients with these conditions, like those with other types of primary myopathies, will generally exhibit facial features that often result in what is described as a *"myopathic face."* The "myopathic face" has many similarities to the *"adenoid face"* that is seen in children with a blocked nasal airway due to enlarged adenoids and other intranasal obstructions, which result in an open-mouth posture. These abnormal human maxillomandibular growth patterns are also consistent with the findings reported by Harvold when rhesus monkeys were forced to maintain an open-mouth posture during jaw growth, as described previously in this chapter. The "myopathic face" findings described by Lehman and colleagues include characteristics of an increased lower anterior facial height, incompetent lips, a high-arched palate, and often anterior open-bite malocclusion (Figs. 4-37 and 4-38).[66] Clinical human and animal model studies confirm that these features are to be expected in the presence of an open-mouth posture during jaw growth, no matter what the cause. Although blocked nasal breathing is the most common cause of an open-mouth posture during growth, clearly congenital myopathies that also result in an open-mouth posture will produce the same facial dysmorphology.

Effects of Trauma on Skeletal Growth

Intrauterine compressive force against the developing face is a form of trauma that can lead to *deformations* of the craniofacial skeleton that are visually present at the time of birth. For example, when there is a decrease in the volume of amniotic fluid, the fetus' head is typically flexed tightly against his or her chest in utero. The compressive forces to the chin will prevent the mandible from achieving its full prenatal growth potential. The normally developing tongue is then forced to rest high in the mouth, thereby preventing approximation and fusion of the lateral maxillary processes. At the time of birth, clefting of the secondary palate is recognized. A pathologic process continues after birth, and this includes retrognathia, a cleft of the secondary palate, and varying degrees of respiratory distress as a result of the otherwise normal tongue resting superior and posterior in the mouth. This intrauterine deformation is responsible for approximately two thirds of those individuals with observed *Robin sequence* at the time of birth. Significant but incomplete catch-up mandibular growth is expected. In general, there will be sufficient mandibular growth in combination with an expected degree of maxillary growth inhibition from cleft palate repair during childhood that results in maxillomandibular harmony but at least mild horizontal deficiency and the clockwise rotation of both jaws. Individuals with these features are typically managed with orthodontics to achieve an acceptable occlusion, but this does result in a degree of facial dysmorphology (see Fig. 4-20). Unless upper airway obstruction with obstructive sleep apnea is confirmed, the degree of maxillomandibular deficiency is such that a surgical consult is generally not requested.

An infant, child, or young adolescent who sustains *injury to the craniofacial skeleton* has the potential for growth disturbance of the injured bones. This is in addition to any skeletal deformity that remains if the fracture was not fully reduced with satisfactory initial healing (see Chapter 35). During some difficult deliveries through the vaginal canal, especially those that require forceps, damage to one or both of the temporomandibular joints or fracture of the mandible can occur. In modern times, with good prenatal care and skilled obstetrics support being available at the time of delivery, birth trauma to the mandible has become a rare event.

A childhood injury to the *cartilage of the nasal septum* may result in nasal deformity. It may also lead to deficient midface growth (see Fig. 35-2).[38] This has been confirmed in experimental animals and documented through clinical observation in humans.[28] In experimental animals, when the cartilage of the nasal septum is removed before growth is complete, there will be a dramatic decrease in the horizontal and vertical growth of the midface. Whether this is the result of a mechanical collapse with a secondary loss of growth velocity of the maxilla or an intrinsic growth restriction of the upper jaw remains open to question.

Trauma to the growing mandible that is severe enough to cause fracture and the displacement of the fragments is also known to result in secondary progressive growth deformities, at least in some cases. There are thought to be three major sites of mandibular growth that may be affected:

1. The mandibular condyle: Proliferating cartilage at the base of the fibrocartilage layer that covers the articular surface results in mandibular growth. At the completion of mandibular growth, the fibrocartilage layer is replaced by bone.
2. The outer surfaces of the ramus: Extensive remodeling of the mandible through the apposition of bone on the posterior and outer surfaces along with resorption on the anterior and inner surfaces is known to occur and is influenced by muscle forces and the compression of the associated soft tissues (e.g., tongue volume) within the mouth.
3. The alveolar process: New alveolar bone is added only as individual teeth develop and erupt into the arch.

In individuals who sustain a *displaced condylar neck fracture,* a sequence of events generally occurs that includes the contraction of the lateral pterygoid muscle with the displacement of the condylar fragment anteriorly and medially (Fig. 4-39). The displaced fragment (i.e., the medial pole of the condyle or the total condyle) has a tendency toward a degree of resorption and malunion. This may then result in a loss of posterior facial height, with a shift of the ipsilateral

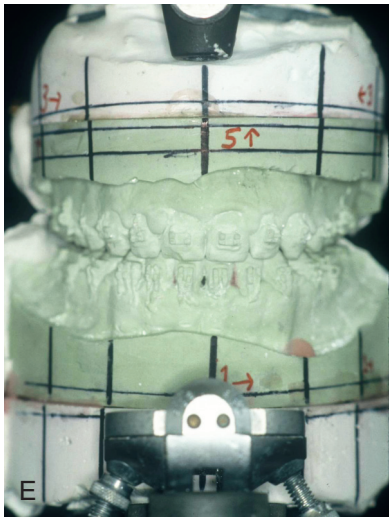
Text continued on p. 124

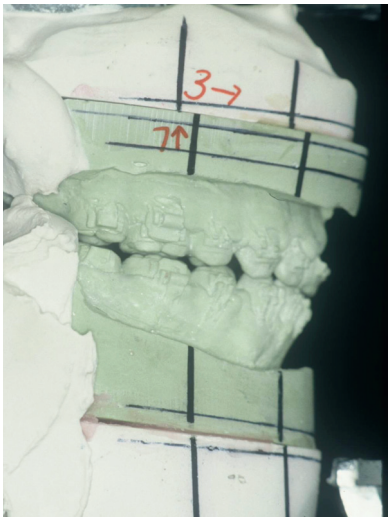
• **Figure 4-38** A 17-year-old boy who was born with a nonprogressive congenital myopathy was referred by an orthodontist for surgical evaluation. The lack of masticatory muscle strength resulted in a constant open-mouth posture. The long face growth pattern is characterized by vertical maxillary excess, a clockwise rotated retrusive mandible, a vertically long and retrusive chin, and an anterior open-bite malocclusion. The patient underwent a combined orthodontic and surgical approach. Four bicuspid extractions relieved dental crowding. This was followed by surgery that included a maxillary Le Fort I osteotomy (vertical intrusion and horizontal advancement); bilateral sagittal split osteotomies of the mandible (horizontal advancement); and osseous genioplasty (vertical reduction and horizontal advancement). **A,** Frontal views in repose before and after reconstruction. **B,** Frontal views with smile before and after reconstruction. **C,** Profile views before and after reconstruction.

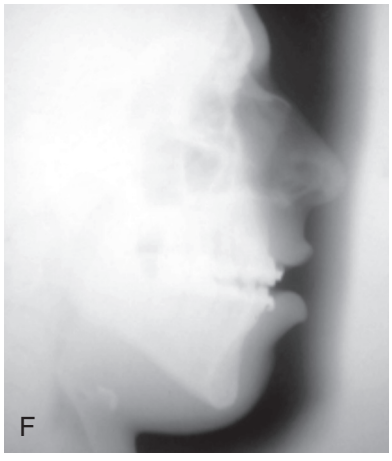
Continued

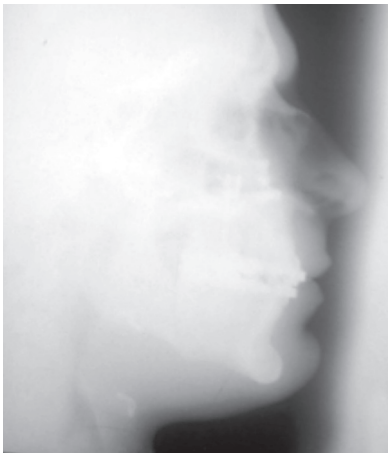
Prior to treatment

After treatment including 4 bicuspid extractions

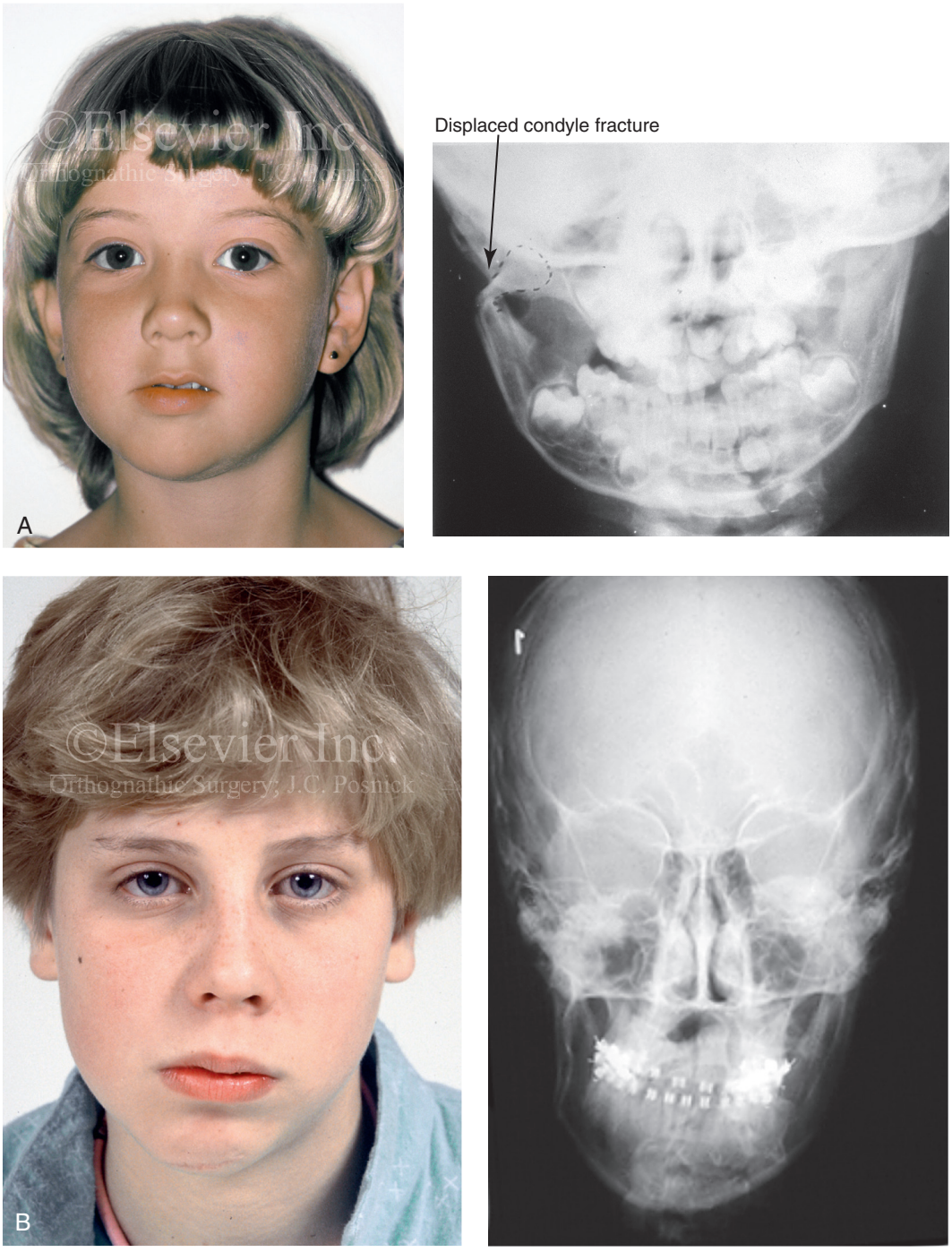






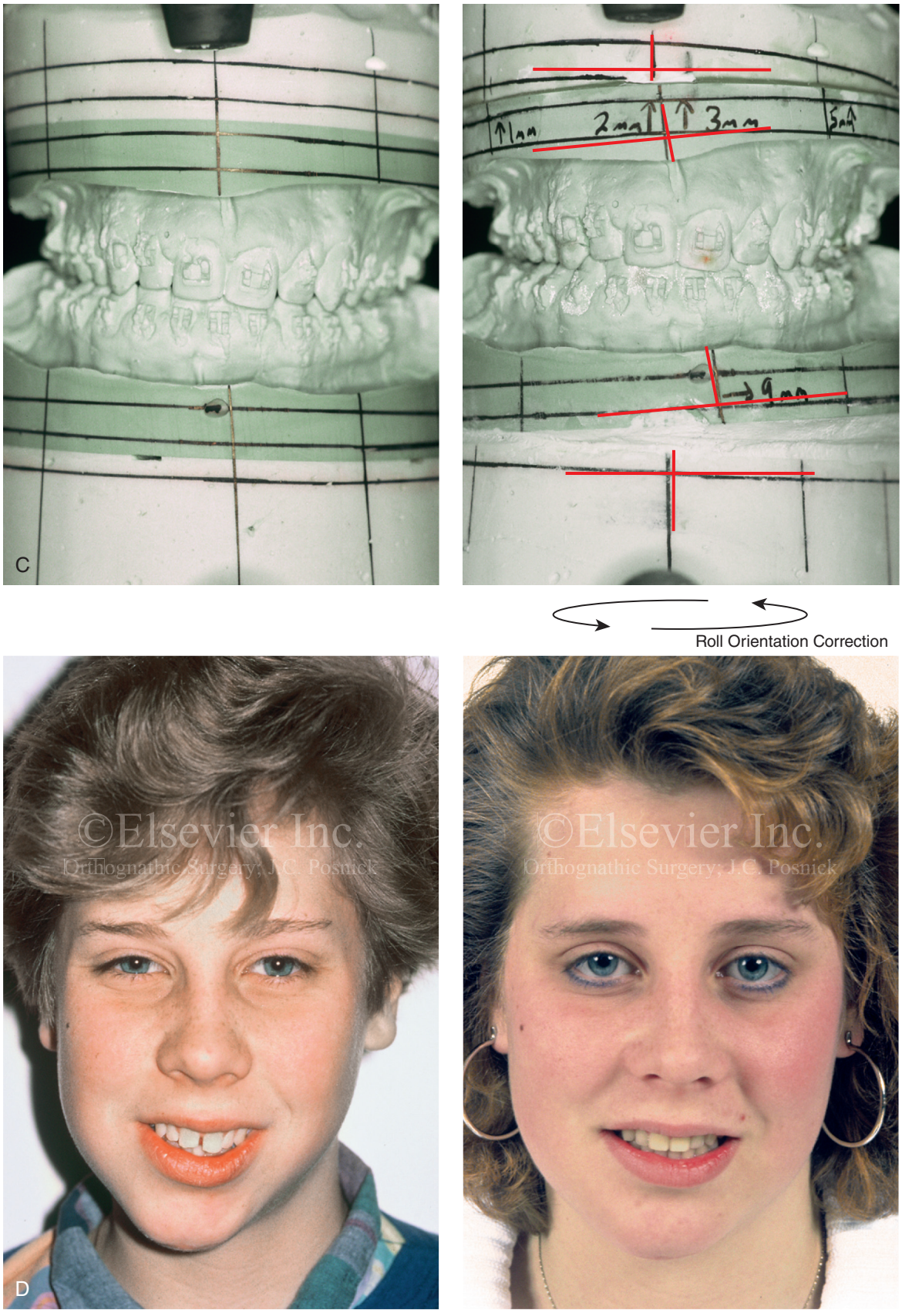


• **Figure 4-38, cont'd** **D,** Occlusal views before and after reconstruction. **E,** Articulated dental casts that indicate analytic model planning. **F,** Lateral cephalometric radiographs before and after surgery.



• **Figure 4-39** A 5-year-old girl sustained a right condyle fracture of the mandible and then presented as a teenager with resulting facial asymmetry involving the maxilla and the mandible. She had full painless mandibular mobility. She then underwent a combined orthodontic and orthognathic approach that included a Le Fort I osteotomy (cant correction), bilateral sagittal split ramus osteotomies (asymmetry correction), an osseous genioplasty (horizontal advancement), and intranasal procedures (septoplasty, inferior turbinate reduction, and nasal recontouring) during her teenage years (see Chapter 35). **A,** Frontal view when the patient was 6 years old, which was 1 year after the right condyle fracture. The anteroposterior facial radiograph indicates a medially displaced right condyle fracture. **B,** Frontal view when the patient was 15 years old. An anteroposterior cephalometric radiograph confirms the resulting facial asymmetry that primarily involved the mandible but that now also involves the secondary deformity of the maxilla and the chin.

Continued



• **Figure 4-39, cont'd C,** Articulated dental casts that indicate analytic model planning. **D,** Frontal views before and after reconstruction.

• **Figure 4-39, cont'd** **E,** Profile views before and after reconstruction. **F,** Occlusal views before and after reconstruction. **G,** Posteroanterior cephalometric radiographs before and after reconstruction.

mandible toward the affected side. In these cases, an asymmetric retrusive lower jaw and predictable malocclusion can be documented when the jaw is placed in centric relation. Experimental and clinical studies confirm that the ability of the individual's mandible and masticatory muscles to adapt will determine the degree of eventual jaw deformity and residual malocclusion as well as what treatment is needed (see Chapter 35).

When significant *condylar trauma* occurs during childhood, with or without fracture, there will have been injury to the fibrocartilage, with a potential effect on growth. If in fact the "condylar cartilage–push theory" of mandibular growth is all important, a devastating growth distortion should occur over time in all affected individuals. Walker tested this theory by deliberately producing condylar fractures in young monkeys and then observing the effect of these fracture on subsequent growth.[131,132] He was unable to document a consistent progressive mandibular deficiency or deformity with malocclusion. Mandibular growth that approximated normal occurred in the majority of experimental animals. Late postmortem gross and histologic evaluation confirmed that there was regeneration of the condylar process with a new layer of cartilage over the articular surface that was identical to the contralateral (noninjured) side. Clinical studies on a series of human children who sustained similar condylar neck fractures confirmed the potential to regenerate bone that approximated the lost (injured) condyle adverse effects on growth in many of the cases.[42,74] It also became evident from these studies that a notable minority (approximately 25%) of affected individuals do sustain significant growth disturbances (i.e., mandibular hypoplasia or asymmetry) after a condylar injury, often with secondary deformity in the growing maxilla (see Fig. 4-39). The explanation for this variation in human response (i.e., repair versus deformity) is likely dependent on the following: 1) the extent of initial injury; 2) the extent of resulting scarring or the lack of regenerative potential; 3) the variable importance of the condyle as a growth center in each individual child; and 4) the variable adaptive ability of the muscles of mastication (see Chapter 35).

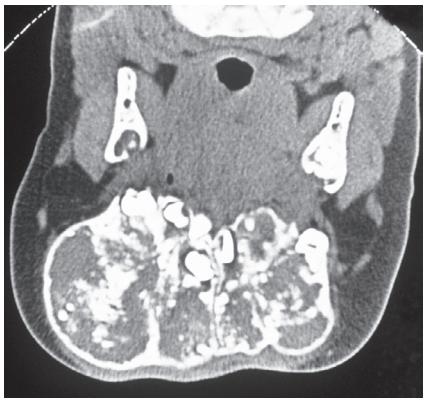
Over the decades, clinicians have confirmed the work of Gilhuus-Moe [42] and Lund[74] by documenting a spectrum of possible outcomes after condylar fracture. Reports confirm instances in which the fracture dislocation of a condylar process has occurred and is followed by a continued normal downward and forward translation of the mandibular body with regeneration of a neocondyle back to baseline. Other clinical reports show a complete loss of the condylar morphology without regeneration followed by decreased posterior facial height, ipsilateral mandibular hypoplasia, and secondary deformity of the contralateral side of the mandible, the maxilla, and the chin region. This sequence of events results in significant maxillomandibular asymmetry and disproportion (see Fig. 35-8). There are also numerous reported examples of bony ankylosis of the condylar stump to the glenoid fossa after the fracture of the condyle (see Fig. 35-1).

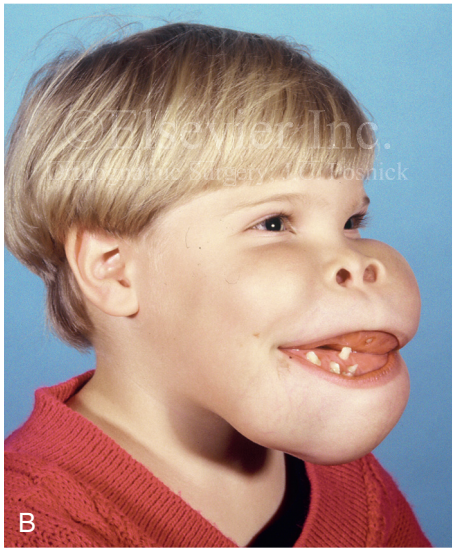
Effects of Tumors and Growths on Skeletal Growth

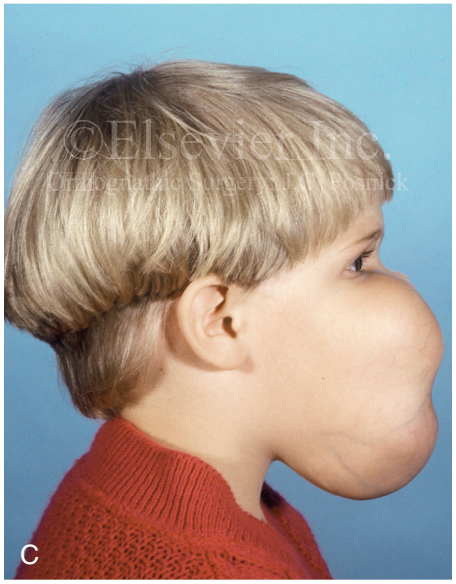
The presence of a sustained *expansile mass* (e.g., cyst, cellular tumor, skeletal mass) will distort the normal growth and morphology of the bones that are being compressed or expanded.[1,6,9,20,24,26,34,46,87-89,95-99] Frequent examples of this occurrence in the maxillofacial region include the effects of neurofibromatosis, vascular malformations, Proteus syndrome, fibrous dysplasia, and odontogenic tumors and cysts (Fig. 4-40). In the adult, the *compressive forces* of a tumor mass may cause resorption and even result in pathologic fracture. The tumor mass may also compress visceral structures and cavities, thereby causing an impairment of function. Depending on the tumor's location, volume, and velocity of expansion, there may be alterations in cognitive function, vision, speech, breathing, swallowing, chewing, lip control, hearing, neck range of motion, or mandibular range of motion. When radiation therapy is required for the management of a malignancy, damage to the adjacent soft tissues and skeletal structures frequently occurs. The arrest of growth in irradiated immature bones may result in orbital dysplasia, zygomatic hypoplasia jaw deformity with malocclusion, and damage to the developing teeth and periodontium (Fig. 4-41).

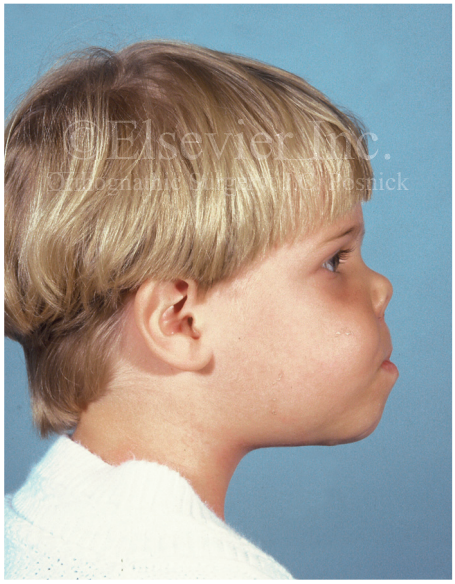
Conclusions

The term *dentofacial deformity* is generally used to describe a significant disproportion of the jaws that is associated with malocclusion. Knowing the etiologic factors of a jaw deformity can be important to the fundamental understanding of how and when to treat the problem. According to current knowledge, factors that are known to cause or result in dentofacial deformities can be subdivided to include known syndromes and anomalies, hereditary tendencies, environmental and neuromotor effects, effects of trauma, and effects of tumors and growths.



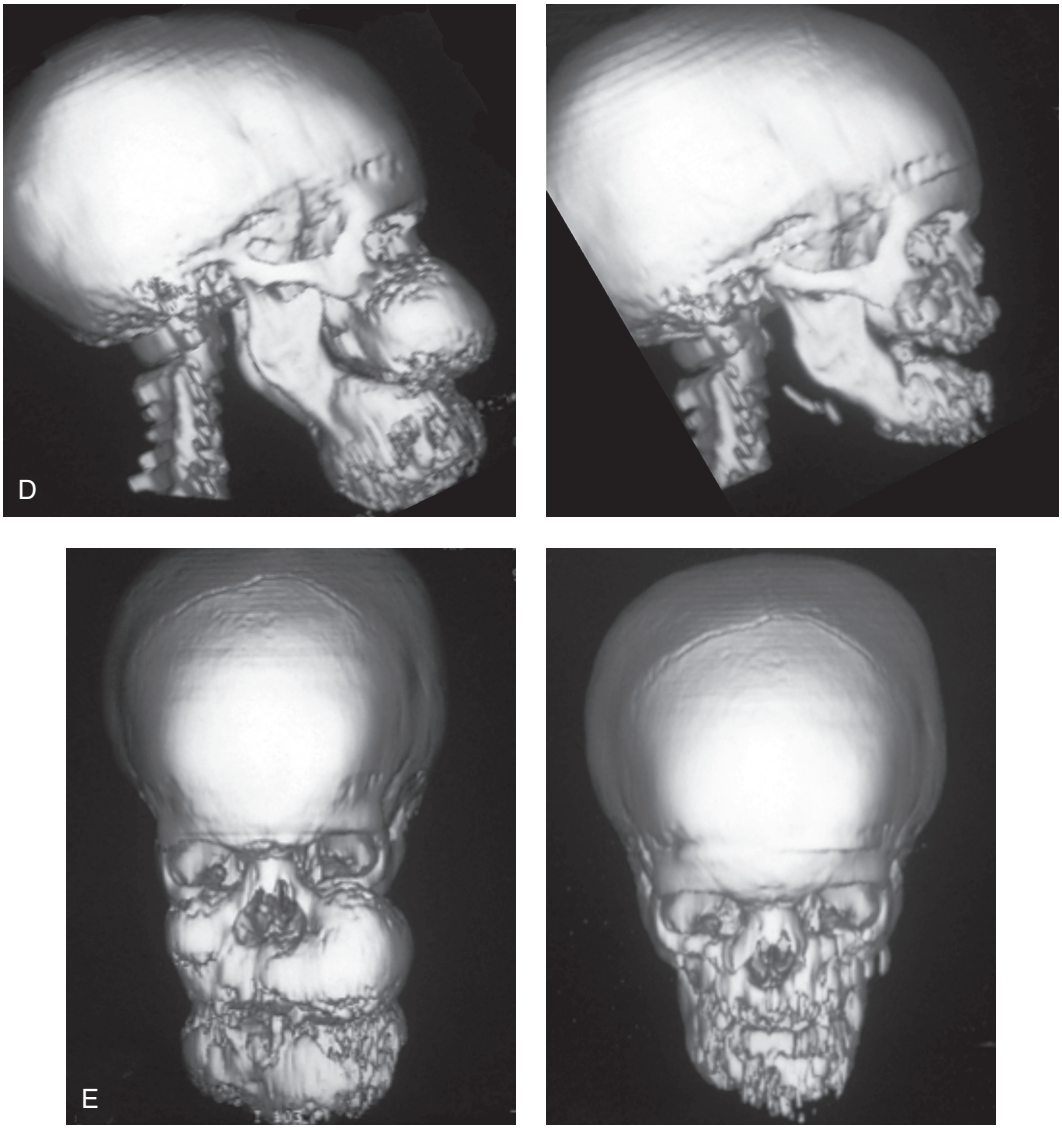






• **Figure 4-40** A 5-year-old girl with polyostotic fibrous dysplasia with massive involvement of the maxilla and mandible bilaterally. An asymmetric swelling of the mandible (left greater than right) was first recognized when the child was 18 months old; it rapidly enlarged, and maxillary involvement became evident with the deterioration of breathing, chewing, speech, and swallowing. Pathologic fractures were sustained in all four extremities as a result of the involvement of fibrous dysplasia. **A,** Frontal and computed tomography scan views before surgery. **B,** Oblique facial views before and after reconstruction. **C,** Profile facial views before and after reconstruction.

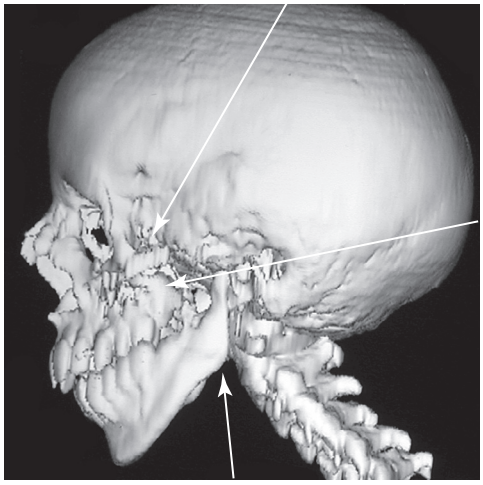
Continued

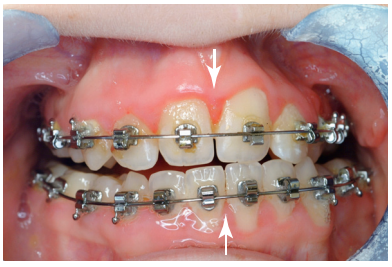


• **Figure 4-40, cont'd D** and **E,** Computed tomography views before and after a debulking procedure. *From Posnick JC, Hughes CA, Milmoe G, et al: Polyostotic fibrous dysplasia: an unusual presentation in childhood.* J Oral Maxillofac Surg *54:1458–1464, 1996.*

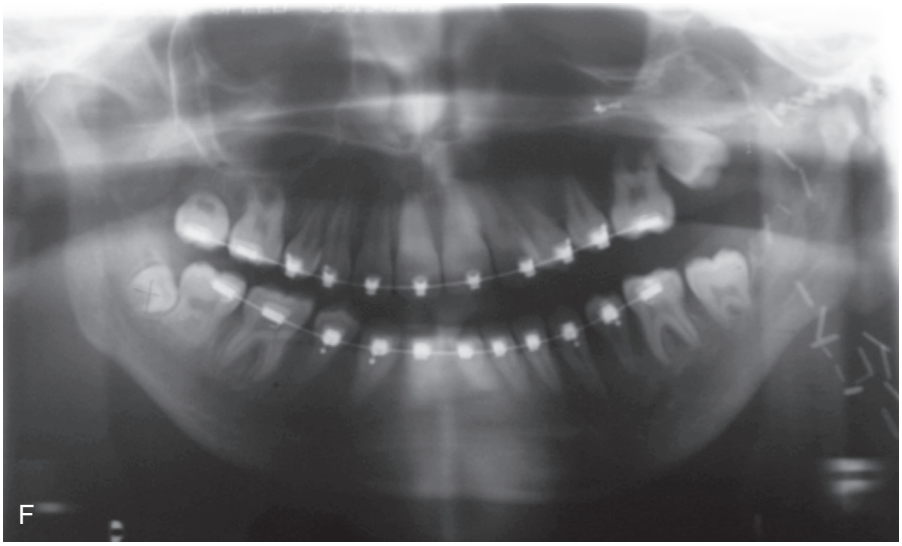
• **Figure 4-41** A teenage girl who at 9 years of age was found to have a malignancy of the parameningeal region. Biopsy confirmed the diagnosis of rhabdomyosarcoma. She was treated with surgical debulking, chemotherapy, and radiation according to intergroup protocols. There was resulting radiation growth disturbance of the skeletal and soft-tissue structures. The patient underwent staged reconstruction, including stage I serratus muscle microvascular transfer to the left temporal region in combination with full-thickness autogenous cranial graft reconstruction of the left zygomatic orbital complex when she was 13 years old. This was followed when she was 16 years old by further staged orthognathic reconstruction to manage the jaw deformity and the malocclusion. The orthodontic portion of her treatment was carried out cautiously, because there was radiation injury to her left posterior maxillary and mandibular dentition. The procedures included maxillary Le Fort I osteotomy in two segments with interpositional autogenous iliac (hip) grafting, bilateral sagittal split ramus osteotomies of the mandible, and an osseous genioplasty. Septoplasty, inferior turbinate reduction, and recontouring of the floor of the nose were also carried out to open the airway. **A,** Facial and occlusal views are shown when the patient was 12 years old, before reconstruction. **B,** Oblique facial and computed tomography scan views when the patient was 12 years old, before reconstruction.

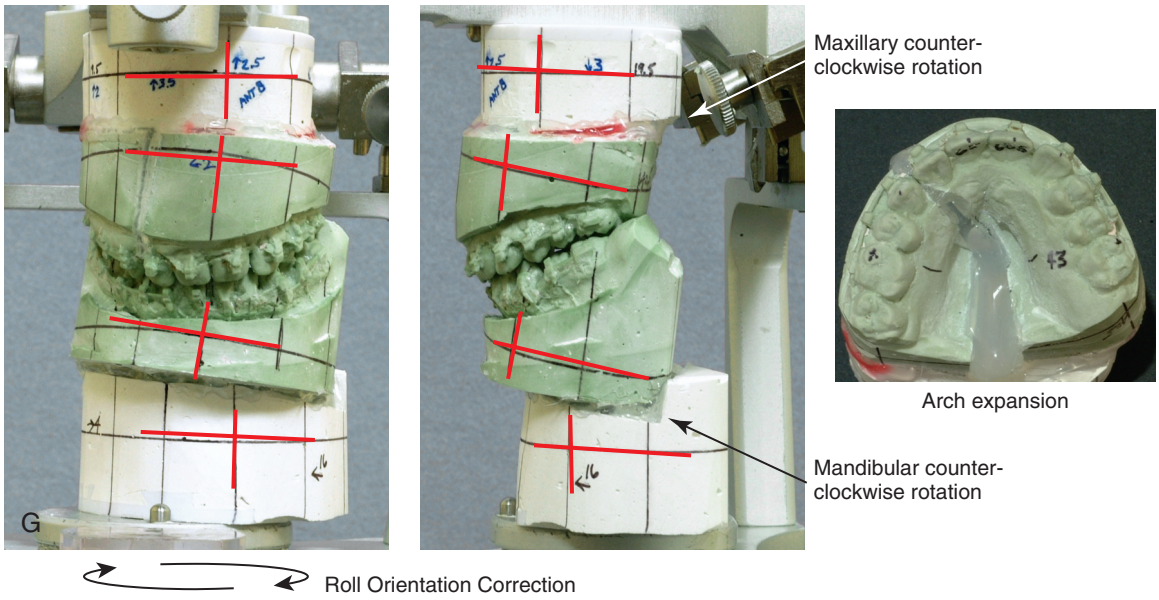
Continued





• **Figure 4-41, cont'd** **C,** Profile facial and computed tomography scan views when the patient was 12 years old, before reconstruction. **D,** Before and 1 year after left zygomatic and orbital cranial graft reconstruction and serratus muscle free-tissue transfer reconstruction. **E,** Frontal and occlusal views when the patient was 13 years old, with orthodontics in progress in preparation for jaw reconstruction.





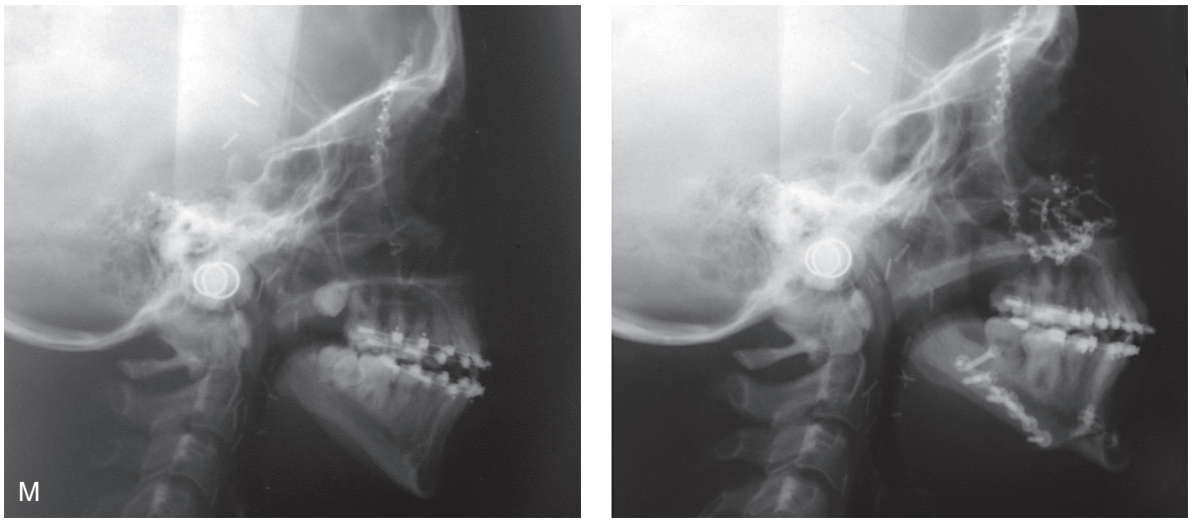
• **Figure 4-41, cont'd** **F,** Panorex radiograph when the patient was 13 years old, with preoperative orthodontics complete in preparation for jaw reconstruction. Note the short dental roots on the left side. **G,** Articulated dental casts that indicate analytic model planning. **H,** Frontal facial views in repose before and after reconstruction.

Continued

• **Figure 4-41, cont'd I,** Frontal views with smile before and after reconstruction. **J,** Oblique facial views before and after reconstruction. **K,** Profile facial views before and after reconstruction.

Prior to retreatment

After treatment



• **Figure 4-41, cont'd L,** Occlusal views before and after orthodontics and orthognathic surgery. **M,** Lateral cephalometric radiographs before and after reconstruction.

References

1. Adamides IP, Spyropoulos MN: The effects of lymphadenoid hypertrophy on the position of the tongue, the mandible and the hyoid bone. *Eur J Orthod* 5:287–294, 1983.
2. Allwright WC, Bundred WH: A survey of handicapping dentofacial anomalies among Chinese in Hong Kong. *Int Dent J* 14:505–519, 1964.
3. Bachrach FH, Young M: A comparison of the degree of resemblance in dental characteristics shown in pairs of twins of identical and fraternal types. *Br Dent J* 21:1293–1304, 1927.
4. Bacon WH, Turlet JC, Krieger J, Stierle JL: Craniofacial characteristics in patients with obstructive sleep apnea syndrome. *Cleft Palate J* 25:374–378, 1988.
5. Behrents RG: The biological basis for understanding craniofacial growth during adulthood. *Prog Clin Biol Res* 187:307–319, 1985.
6. Bradley JP, Zide BM, Berenstein A, Longaker MT: Large arteriovenous malformations of the face: aesthetic results with recurrence control. *Plast Reconstr Surg* 103:351–361, 1999.
7. Bresolin D, Shapiro PA, Shapiro GG, et al: Mouth breathing in allergic children: its relationship to dentofacial development. *Am J Orthod* 84:334–340, 1983.
8. Brudevold F: A basic study of the chewing forces of a denture wearer. *J Am Dent Assoc* 43:45–51, 1951.
9. Burns AJ, Kaplan LC, Mulliken JB: Is there association between hemangioma and syndromes with dysmorphic features? *Pediatrics* 88:1257–1267, 1991.
10. Burton PR, Tobin MD, Hopper JL: Key concepts in genetic epidemiology. *Lancet* 366:941–951, 2005.
11. Chung CS, Runck DW, Niswander JD, et al: Genetic and epidemiologic studies of oral characteristics in Hawaii's schoolchildren. I. Caries and periodontal disease. *J Dent Res* 49:1374–1385, 1970.

12. Cleall JF: Deglutition: a study of form and function. *Am J Orthod* 51:566–594, 1965.
13. Close RJ: Dynamic properties of mammalian skeletal muscle. *Physiol Rev* 52:129–197, 1972.
14. Cohen MM, Jr, Rollnick BR, Kaye CL: Oculoauriculovertebral spectrum: an updated critique. *Cleft Palate J* 26:276–286, 1989.
15. Cohen MM, Jr: *The child with multiple birth defects*, ed 2, New York, 1997, Oxford University Press.
16. Cohen MM, Jr: Holoprosencephaly survival and performance. *Am J Med Genet (Semin Med Genet)* 89:116–120, 1999.
17. Cohen MM, Jr, MacClean RE: *Craniosynostosis: diagnosis, evaluation, and management*, New York, 2000, Oxford University Press.
18. Cohen MM, Jr: Problems in the definition of holoprosencephaly. *Am J Med Genet* 103:183–187, 2001.
19. Cohen MM, Jr: Perspectives on craniofacial anomalies, syndromes, and other disorders. In Lin KY, Ogle RC, Jane JA, editors: *Craniofacial surgery: science and surgical technique*, Philadelphia, 2002, W.B. Saunders Company, Chapter 1, pp 3–38.
20. Cohen MM, Jr: Vasculogenesis, angiogenesis, hemangiomas, and vascular malformations. *Am J Med Genet* 108:265–274, 2002.
21. Cohen MM, Jr, Neri G, Weksberg R: *Overgrowth syndromes*, New York, 2002, Oxford University Press.
22. Cohen MM, Jr: FGFs/FGFRs and associated disorders. In Epstein CJ, Erickson RP, Wynshaw-Boris A, editors: *Inborn errors of development*, New York, 2004, Oxford University Press, Chapter 33, pp 380–400.
23. Cohen MM, Jr: Holoprosencephaly: clinical, anatomic, and molecular dimensions. *Birth Defects Res A Clin Mol Teratol* 76:658–673, 2006.
24. Cohen MM, Jr: Vascular update: morphogenesis, tumors, malformations, and molecular dimensions. *Am J Med Genet* 140A:2013–2038, 2006.
25. Cohen MM, Jr: Craniofacial disorders. In Rimoin DL, Conner JM, Pyeritz RE, Korf BR, editors: *Emory and Rimoin's principles and practice of medical genetics*, ed 5, Philadelphia, 2007, Elsevier, Chapter 151, pp 3303–3348.
26. Cohen MM, Jr: Craniofacial abnormalities. In Gilbert-Barness E, editor: *Potter's pathology of the fetus, infant, and child*, ed 2, vol 1, Philadelphia, 2007, Mosby/Elsevier, Chapter 20, pp 885–918.
27. Cohen MM, Jr: Holoprosencephaly: a mythologic and teratologic distillate. *Am J Med Genet Part C Semin Med Genet* 154C:8–12, 2010.
28. D'Ascanio L, Lancione C, Pompa G, et al: Craniofacial growth in children with nasal septum deviation: a cephalometric comparative study. *Int J Pediatr Otorhinolaryngol* 74:1180–1183, 2010.
29. Downs WG: Studies in the causes of dental anomalies. *J Dent Res* 16:367–379, 1928.
30. Edwards SJ, Gladwin AJ, Dixon MJ: The mutational spectrum in Treacher Collins syndrome reveals a predominance of mutations that create a premature termination codon. *Am J Hum Genet* 60:515–524, 1997.
31. El-Gheriani AA, Maher BS, El-Gheriani AS, et al: Segregation analysis of mandibular prognathism in Libya. *J Dent Res* 82:523–527, 2003.
32. Emrich RE, Brodie AG, Blayney JR: Prevalence of class I, class II, and class III malocclusions (Angle) in an urban population: an epidemiological study. *J Dent Res* 44:947–953, 1965.
33. Enache AM, Nimigean VR, Mihaltan F, et al: Assessment of sagittal and vertical skeletal patterns in Romanian patients with obstructive sleep apnea. *Rom J Morphol Embryol* 51:505–508, 2010.
34. Enjolras O, Mulliken JB: Vascular tumors and vascular malformations. *Adv Dermatol* 13:375–423, 1998.
35. Finn RA: Relationship of vertical maxillary dysplasias, bite force, and integrated EMG. In *Abstracts of Conference on Craniofacial Research*, Ann Arbor, Mich, 1978, University of Michigan Center for Human Growth and Development.
36. Fox RR, Crary DD: Mandibular prognathism in the rabbit. *J Hered* 62:23–27, 1971.
37. Fraser FC: Liability, thresholds, malformations, and syndromes. *Am J Med Genet* 66:75–76, 1996.
38. Freng A, Kvam E: Facial sagittal growth following partial basal resection of the nasal septum: a retrospective study in man. *Eur J Orthod* 1:89–96, 1979.
39. Freng A: Dentofacial development in the long-lasting nasal stenosis. *Scand J Dent Res* 89:260–267, 1979.
40. Garner LD, Butt MH: Malocclusion in black American and Nyeri Kenyans, an epidemiologic study. *Angle Orthod* 55:139–146, 1985.
41. Gass JR, Valiathan M, Tiwari HK, et al: Familial correlations and heritability of maxillary midline diastema. *Am J Orthod Dentofacial Orthop* 123:35–39, 2003.
42. Gilhus-Moe O: *Fractures of the mandibular condyle in the growth period*, Stockholm, 1969, Scandinavian University Books, Universitatsforlaget.
43. Graf H, Grassl J, Aeberhard H: A method for measurement of occlusal force in three directions. *Helv Odont Acta* 18:7–11, 1974.
44. Grewe JM, Cervenka J, Shapiro BL, et al: Prevalence of malocclusion in Chippewa Indian children. *J Dent Res* 47:302–305, 1968.
45. Handelman CS, Osborne G: Growth on nasopharynx and adenoid development from one to eighteen years. *Angle Orthod* 46:243–258, 1976.
46. Hannuksela A: The effects of moderate and severe atopy on the facial skeleton. *Eur J Orthod* 3:187–193, 1981.
47. Harding CO, Green CG, Perloff WH: Respiratory complications in children with spondyloepiphyseal dysplasia congenital. *Pediatr Pulmonol* 9:49–54, 1990.
48. Harris EF, Johnson MG: Heritability of craniometric and occlusal variables: a longitudinal sib analysis. *Am J Orthod Dentofac Orthop* 99:258–268. 1991.
49. Harvold EP: The role of function in the etiology and treatment of malocclusion. *Am J Orthod* 54:883–898, 1968.
50. Harvold EP, Chierici G, Varervik K: Experiments on the development of dental malocclusion. *Am J Orthod* 61:38–44, 1972.
51. Harvold EP, Vargervik K, Chierici G: Primate experiments on oral sensation and dental malocclusion. *Am J Orthod Dentofacial Orthop* 63:494–508, 1973.
52. Harvold EP: Centric relation: a study of pressure and tension systems in bone modeling and mandibular positioning. *Dent Clin North Am* 19:473–484, 1975.
53. Harvold EP, Tomer BS, Varervik K, Chierici G: Primate experiments on oral respiration. *Am J Orthod* 79:359–372, 1981.
54. Harvold EP, Chierici G, Vargervik K, editors: *Treatment of hemifacial microsomia*, New York, 1983, Alan R. Liss.
55. Haryett RD, Hansen FC, Davidson PO: Chronic thumb sucking: the psychological effects and relative effectiveness of various methods of treatment. *Am J Orthod Dentofacial Orthop* 53:569–585, 1967.
56. Holmberg H, Linder-Aronson S: Cephalometric radiographs as a means of evaluating the capacity of the nasal and nasopharyngeal airway. *Am J Orthod* 76:479–490, 1979.
57. Horowitz SL, Osborne RH, Degeorge FV: A cephalometric study of craniofacial variation in adult twins. *Angle Orthod* 30:1–5, 1960.
58. Huang CM, Mi MP, Vogt DW: Mandibular prognathism in the rabbit: discrimination between single-locus and multifactorial models of inheritance. *J Hered* 72:296–298, 1981.
59. Hunt EE: The continuing evolution of modern man. *Cold Spring Harb Symp Quant Biol* 24:245–254, 1959.
60. Iwagake H: Hereditary influence of malocclusion. *Am J Orthod Oral Surg* 24:328–336, 1938.
61. Johnson AL: The constitutional factor in skull form and dental occlusion. *Am J Orthod Oral Surg* 26:627–663, 1940.
62. Kraus BS, Wise WJ, Frie RA: Heredity and the craniofacial complex. *Am J Orthod* 45:172–217, 1959.
63. Kreiborg S, Jensen BL, Møller E, Björk A: Craniofacial growth in a case of congenital muscular dystrophy. *Am J Orthod* 74:207–215, 1978.
64. Krogman WM: The role of urbanization in the dentition of various population groups. *Zf Rassenkunde* 9:41–72, 1938.
65. Lear CSC, Moorrees CFA: Bucco-lingual muscle force and dental arch form. *Am J Orthod Dentofacial Orthop* 56:379, 1969.

66. Lehman H, Harari D, Tarazi E, et al: Orthognathic surgery in primary myopathies: severe case of congenital fiber type disproportion with long-term follow-up and review of the literature. *J Oral Maxillofac Surg* 70:1636–1642, 2012.
67. Lew KK, Foong WC, Loh E: Malocclusion prevalence in an ethnic Chinese population. *Aust Dent J* 38:442–449, 1993.
68. Linder Aronson S, Aschan G: Nasal resistance to breathing and palatal height before and after expansion of the median palatine suture. *Odontol Rev* 14:254–270, 1963.
69. Linder-Aronson S: Adenoids: their effect on mode of breathing and nasal airflow and their relationship to characteristics of the facial skeleton and the dentition: a biometric, rhino-manometric and cephalometro-radiographic study on children with and without adenoids. *Acta Otolaryngol Suppl* 265:1–132, 1970.
70. Linder-Aronson S, Lindgren J: The skeletal and dental effects of rapid maxillary expansion. *Br J Orthod* 6:25–29, 1979.
71. Linderholm H, Wennstrom A: Isometric bite force and its relation to general force and body build. *Acta Odontol Scand* 28:679–689, 1970.
72. Linderholm H, Lindquist B, Ringquist M, Wennstrom A: Isometric bite force in children and its relation to body build and general muscle force. *Acta Odontol Scand* 29:563–568, 1971.
73. Litton SF, Ackerman LV, Issacson RJ, Shapiro BL: A genetic study of Class III malocclusion. *Am J Orthod* 58:565–577, 1970.
74. Lund K: Mandibular growth and remodeling processes after mandibular fractures. *Acta Odontol Scand Suppl* 32:64, 1974.
75. Lundström A: The importance of genetic and non-genetic factors in the facial skeleton studies in 100 pairs of twins. *Trans Eur Orthod Soc* 1954.
76. Lundström A: The significance of genetic and non-genetic factors in the profile of the facial skeleton. *Am J Orthod* 41:910–916, 1955.
77. Lundström A, Woodside DG: Individual variation in growth directions expressed at the chin and the midface. *Eur J Orthod* 2:65–79, 1980.
78. Lundström A: Nature versus nature. *Eur J Orthod* 1986.
79. Mah J: Genetics of the "Habsburg jaw," *Eur Orthod Soc Res* 1:1–8, 2001.
80. Mason RM, Proffit WR: The tongue thrust controversy: background and recommendations. *J Speech Hear Disord* 39:115–132, 1974.
81. McNamara JA, Jr: Neuromuscular and skeletal adaptation to altered function in the orofacial region. *Am J Orthod* 64:578–606, 1973.
82. Mills JRE: Occlusion and malocclusion of the teeth of primates. In Brothwell DR, editor: *Dental anthropology*, New York, 1963, Macmillan, pp 29–51.
83. Mills LF: Epidemiologic studies of occlusion: IV. The prevalence of malocclusion in a population of 1,455 school children. *J Dent Res* 45:332–336, 1966.
84. Miotti A: Rassegna bibliografica sull'eziologia ed epidemiologia del prognatismo mandibolare. *Dent Cadmos* 50:29–32, 1982.
85. Morton NE, Chung CS, Mi MP: *Genetics of interracial crosses in Hawaii*, Basel, 1967, Karger.
86. Mossey PA: The heritability of malocclusion: Part 1–Genetics, principles and terminology. *Br J Orthod* 26:103–113, 1999.
87. Mulliken JB, Glowacki J: Hemangiomas and vascular malformations in infants and children: a classification based on endothelial characteristics. *Plast Reconstr Surg* 69:412–422, 1982.
88. Mulliken JB: Cutaneous vascular anomalies. *Semin Vasc Surg* 6:204–218, 1993.
89. Mulliken JB: Vascular anomalies. In Aston SJ, Beasley RW, Thorne CHM, editors: *Grabb and Smith's plastic surgery*, ed 5, Philadelphia, 1997, Lippincott-Raven, p 191.
90. Nagan P, Fields HW: Open bite: a review of etiology and management. *Pediatr Dent* 19:91–98, 1997.
91. Newman GV: The Eskimo's dentofacial complex; effects of environmental and genetic factors. *U S Armed Forces Med J* 3:1653–1662, 1952.
92. Niswander JD, Sujaku C: Congenital anomalies of teeth in Japanese children. *Am J Phys Anthropol* 21:569–574, 1963.
93. Niswander JD: Further studies on the Xavante Indians: VII. The oral status of the Xavante of Simões Lopes. *Am J Hum Genet* 19:543–553, 1967.
94. Nordstrom SW, Yemm R: The relationship between jaw position and isometric active tension produced by direct stimulation of the rat masseter muscle. *Arch Oral Biol* 19:353–359, 1974.
95. Posnick JC, Cleland HJ, Zuker RM, Mock D: Recurrent giant cell lesion of the nasomaxillary region in a child: excision and immediate reconstruction with a free rectus abdominis muscle transfer. *J Oral Maxillofac Surg* 50:1009–1015, 1992.
96. Posnick JC, Al-Qattan MM, Zuker RM: Large vascular malformation of the face undergoing resection with facial nerve preservation in infancy. *Ann Plast Surg* 30(1):67–70, 1993.
97. Posnick JC, Chen P, Zuker R, et al: Extensive malignant melanoma of the uvea in childhood: resection and immediate reconstruction with microsurgical and craniofacial techniques. *Ann Plast Surg* 31(3):265–270, 1993.
98. Posnick JC, Wells MD, Drake JM, et al: Childhood fibrous dysplasia presenting as blindness: a skull base approach for resection and immediate reconstruction. *Pediatr Neurosurg* 19(5):260–266, 1993.
99. Posnick JC: Pediatric craniomaxillofacial tumors. *Oral Maxillofac Surg Clin North Am* 197–210, 1994.
100. Proffit WR, Gamble JW, Christiansen RL: Generalized muscular weakness with severe anterior open bite. *Am J Orthod* 54:104–110, 1968.
101. Proffit WR, Norton LA: The tongue and oral morphology: influences of tongue activity during speech and swallowing. *J Speech Hear Res* 5:106, 1970.
102. Proffit WR: Muscle pressures and tooth position: North American whites and Australian aborigines. *Angle Orthod* 45:1–11, 1975.
103. Proffit WR, Sellers KT: The effect of intermittent forces on eruption of the rabbit incisor. *J Dent Res* 65:118–122, 1986.
104. Proffit WR, Phillips C: Adaptations in lip posture and pressure following orthognathic surgery. *Am J Orthod* 93:294–304, 1988.
105. Proffit WR, Phillips C, Fields HW, Turvey TA: The effect of orthognathic surgery on occlusal force. *J Oral Maxillofac Surg* 47:457–463, 1989.
106. Samman N, Tong AC, Cheung LK, et al: Analysis of 300 dentofacial deformities in Hong Kong. *Int J Adult Orthodon Orthognath Surg* 7:181–185, 1992.
107. Schudy FF: The rotation of the mandible resulting from growth: its implications in orthodontic treatment. *Angle Orthod* 35:36–50, 1965.
108. Schull WJ, Neel JV: *The effects of inbreeding on Japanese children*, New York, 1965, Harper & Row.
109. Seigel ML: Mechanisms of early maxillary growth: implications for surgery. *J Oral Surg* 34:106–112, 1976.
110. Shprintzen RJ: Pierre Robin, micrognathia, and airway obstruction: the dependency of treatment on accurate diagnosis. *Int Anesthesiol Clin* 26:64–71, 1988.
111. Sillman JH: Dimensional changes in the dental arches: longitudinal study from birth to 25 years. *Am J Orthod Dentofacial Orthop* 50:824, 1964.
112. Singh GD, McNamara JA, Jr, Lozanoff S: Procrustes, Euclidean and cephalometric analysis of the morphology of the mandible in human class III malocclusions. *Arch Oral Biol* 43:535–543, 1998.
113. Singh GD: Morphologic determinants in the etiology of class III malocclusion: a review. *Clin Anat* 12:382–405, 1999.
114. Siriwat PP, Jarabak JR: Malocclusion and facial morphology: is there a relationship? *Angle Orthod* 55:127–138, 1985.
115. Sousa JB, Anselmo-Lima WT, Valera FC, et al: Cephalometric assessment of the mandibular growth pattern in mouth-breathing children. *Int J Pediatr Otorhinolaryngol* 69:311–317, 2005.
116. Stiles KA, Luke JE: The inheritance of malocclusion due to mandibular prognathism. *J Hered* 44:241–245, 1953.
117. Stockard CR: *The genetic and endocrine basis for differences in form and behavior as*

elucidated by studies of contrasted pure-line dog breeds and their hybrids, Philadelphia, 1941, Wistar Institute.
118. Subtelny JD: Oral respiration: facial maldevelopment and corrective dentofacial orthopedics. *Angle Orthod* 50:147–164, 1980.
119. Tenczar P, Bader RS: Maternal effect in dental traits of the house mouse. *Science* 15:1398–1400, 1966.
120. Thompson EM, Winter RM: Another family with the "Habsburg jaw," *J Med Genet* 25:838–842, 1988.
121. Thompson JR, Broadie AG: Factors in the position of the mandible. *J Am Dent Assoc* 29:925–941, 1942.
122. Throckmorton G, Finn R, Bell W: Biomechanics of differences in lower face height. *Am J Orthod* 77:410–420, 1980.
123. Tollaro I, Baccetti T, Franchi L: Class III malocclusion in the deciduous dentition: a morphological and correlation study. *Eur J Orthod* 16:401–408, 1994.
124. Treacher Collins Syndrome Collaborative Group: Positional cloning of a gene involved in pathogenesis of Treacher Collins syndrome. *Nat Genet* 12:130–136, 1996.
125. Turvey TA, Hall DJ, Warren DW: Alterations in nasal airway resistance following superior repositioning of the maxilla. *Am J Orthod* 85:109–115, 1984.
126. Tweed CH: *Clinical orthodontics*, St. Louis, 1966, Mosby.
127. Valera FC, Travitzki LV, Mattar SE, et al: Muscular, functional and orthodontic changes in preschool children with enlarged adenoids and tonsils. *Int J Pediatr Otorhinolaryngol* 67:761–770, 2003.
128. Vargervik K, Miller AJ, Chierici G, et al: Morphologic response to changes in neuromuscular patterns experimentally induced by altered mode of respiration. *Am J Orthod* 85:115–124, 1984.
129. Vargervik K: Relationship between bone and muscles of mastication in hemifacial microsomia [discussion]. *Plast Reconstr Surg* 99:998–999, 1997.
130. Vargervik K, Mill AJ, Chierici G, et al: Morphologic response to changes in neuromuscular patterns experimentally induce by altered mode of respiration. *Am J Orthod* 85:115–124, 1984.
131. Walker RV: Traumatic mandibular condyle fracture dislocations. Effect on growth in the Macaca rhesus monkey. *Am J Surg* 100:850–863, 1960.
132. Walker RV: Condylar abnormalities. In *Transactions of the 2nd International Conference on Oral Surgery*, Copenhagen, 1965, Munksgaard Copenhagen, pp 81–96.
133. Warren DW, Hershey HG, Turvey TA, et al: The nasal airway following maxillary expansion. *Am J Orthod* 91:111–116, 1987.
134. Wenzel A, Henriksen J, Melsen B: Nasal respiratory resistance and head posture: effect of intra-nasal corticosteroid (Budesonide) in children with asthma and perennial rhinitis. *Am J Orthod* 84:422–426, 1983.
135. Wickwire NA, White RP, Proffit WR: The effect of mandibular osteotomy on tongue position. *J Oral Surg* 30:184–190, 1972.
136. Williams MD, Sarver DM, Sadowsky PL, Bradley E: Combined rapid maxillary expansion and protraction facemask in the treatment of Class III malocclusion in growing children: a prospective long-term study. *Semin Orthod* 3:265–274, 1997.
137. Wollf G, Wienker TF, Sander H: On the genetics of mandibular prognathism: analysis of large European noble families. *J Med Genet* 30:112–116, 1993.
138. Wyszynski DF, Beaty TH, Maestri NE: Genetics of nonsyndromic oral clefts revisited. *Cleft Palate Craniofac J* 33:406–417, 1996.
139. Yamaguchi T, Park SB, Narita A, et al: Genome-wide linkage analysis of mandibular prognathism in Korean and Japanese patients. *J Dent Res* 84:255–259, 2005.
140. Yurkstas A, Curby W: Force analysis of prosthetic appliance during function. *J Prosthet Dent* 3:82–87, 1953.

5

Orthodontic Considerations in the Evaluation and Treatment of Dentofacial Deformities

JEFFREY C. POSNICK, DMD, MD

- Orthodontic Treatment Philosophy
- The Biologic Basis for Orthodontic Tooth Movement
- Periodontal Ligament Structure and Function
- Response of the Teeth and the Periodontium to Normal Occlusal Function
- Orthodontic Tooth Movement
- Conclusions

Orthodontic Treatment Philosophy

In current clinical practice, orthodontic therapy is carried out to provide an idealized occlusion, to enhance facial aesthetics, and to contribute to the individual's overall health and self-image. Through manipulation of the periodontal apparatus, orthodontic treatment has the potential to not only improve occlusion and periodontal relationships but to facilitate their maintenance over a lifetime. For those individuals with dentofacial deformities, orthodontic treatment is indispensable to remove dental compensations, thereby allowing for the correction of the jaw relationships while providing optimal occlusion and periodontal health. Orthodontics has come a long way since 1972, when Dr. T.M. Graber stated the following in the standard textbook of the day:

> *"Orthodontic therapy can produce dire consequences that will later require the services of the periodontist or even the prosthodontist. The limitations of orthodontic treatment are stringent. The [useful] role of tooth sacrifice [and/or orthognathic surgery] must be recognized. Traditionally, orthodontists had their patients wear retaining appliances indefinitely after the removal of tooth moving appliances and attributed any change back toward the original malocclusion to 'the wisdom teeth' or 'the lack of patient cooperation wearing the removable retainer as instructed.'"*

Current issues in orthodontic therapy are, for the most part, no longer just a matter of what attachments or mechanical approaches to use.* Contemporary orthodontics focuses on accurate diagnosis and a basic philosophy of treatment to enhance facial aesthetics and to correct dysfunction. The role of orthognathic surgical interventions and coordinated comprehensive dental rehabilitation to achieve the most favorable outcome should always be considered.[110]

Malocclusions result from a combination of environmental influences and the patient's morphogenetic hereditary predisposition (see Chapter 4).[1,11,15,18,43,44] The dental alignment that naturally occurs in each individual is greatly influenced by the baseline jaw relationships as well as by his or her daily postural masticatory and airway function.† In the presence of a dentofacial deformity, the mere orthodontic movement of the teeth into a "better" interdigitation will not establish normal jaw relationships nor will it correct the head and neck dysfunctions that contribute to or result from the malocclusion.[147,157,179-181] The observed crowding of the teeth generally reflects nature's attempt to establish a homeostatic balance among the available space in the alveolus, the size of the teeth and the dental roots, the position and size of the jaws, and the constant molding and balancing effects of the masticatory and facial musculature with regard to daily function.[6,146,147,162-165,182,183]

*References 20, 22, 35, 45, 46, 48, 83, 87, 100, 115, 158, 171, 198, 207, 220-223, 226-228

†References 50, 58, 59, 75, 76, 91, 93-97, 119, 127, 134-136, 141, 150-152, 167, 213, 216, 229

The Biologic Basis for Orthodontic Tooth Movement

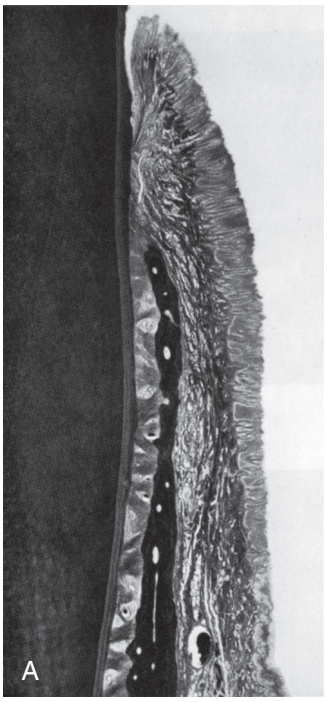
Currently, there are a plethora of appliances than can accomplish almost any desired change in the alignment of the teeth.[69,72] From a purely mechanical perspective, orthodontists are trained in the techniques of tooth movement with the goals of improving dental alignment in each jaw and achieving "ideal" occlusion between the upper and lower arches. The orthodontic movement of the teeth is based on the principle that, if prolonged pressure is applied to a tooth, movement will occur as the bone around the tooth remodels. This is primarily a periodontal ligament phenomenon. In the process, bone is selectively resorbed in some areas and deposited in others. The bone that surrounds the tooth responds to the applied force through intermediate structures (periodontal ligaments), and the objective is for the tooth to move without the loss of crestal height or cortical plates while carrying its attachment apparatus along with it. The whole socket must migrate along with the tooth during this process.

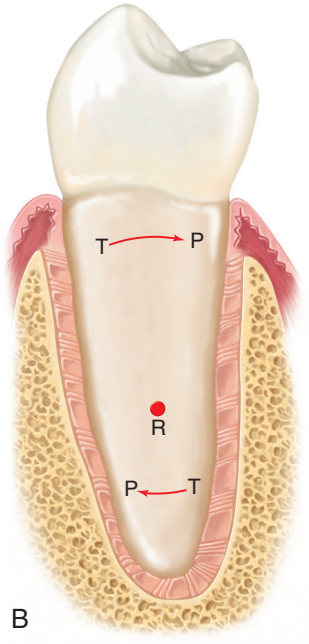
When orthodontic therapy is used to move teeth outside of a biologic envelope, extended treatment time, the potential for periodontal sequelae, and a less-than-ideal long-term occlusion are the likely consequences.[70,86,178] These may occur as a result of the following: (1) attempts to further compensate the position of the teeth toward the skeletal disharmony and outside of the alveolar housing; (2) dental (root) crowding within limited available alveolar bone; or (3) ongoing nasal airway obstruction with an open mouth posture and inadequate lip coverage of the incisors.[4,8,9,32,73,85,193]

Periodontal Ligament Structure and Function

Unless ankylosis is present, each tooth is connected to and separated from the adjacent alveolar bone by a specialized collagen supporting structure known as the *periodontal ligament* (PDL) (Fig. 5-1). The PDL normally occupies a space of approximately 0.5 mm around all parts of the tooth root. The PDL is comprised of a network of parallel collagenous fibers that insert into the cementum (i.e., the covering of the root surface of each tooth) and then into a dense bony plate (radiographically, this is called the *lamina dura*) that is adjacent to each tooth within the alveolar bone. These supporting PDL fibers are angulated between the root of the tooth and adjacent bone; they attach apically on the root and extend occlusally to the adjacent bone. Biomechanically, these angulated PDL fibers act as shock absorbers by helping to resist forces that are applied to the tooth during normal chewing function.

Within the periodontal ligament space, there are cellular elements (i.e., mesenchymal cells, nerve endings, and vessels) and interstitial fluid. The primary *cellular elements* (i.e., mesenchymal cells) are thought to differentiate to form the fibrocytes and the osteocytes. The *collagenous elements* of the PDL are constantly being renewed and altered, depending on the level of functional stress that they are undergoing. The remodeling and recontouring of the alveolar bone is a necessary component of orthodontic tooth movement, and it is dependent on specialized cells (Fig.





• **Figure 5-1** **A,** Principal fiber bundles of the periodontal ligament on the facial surface of a mandibular premolar (silver stain). **B,** Distribution of faciolingual forces *(arrows)* around the axis of rotation *(R)* in a mandibular premolar. The periodontal ligament fibers are compressed in areas of pressure *(P)* and taut in areas of tension *(T)*. *Part A from Glickman I:* Clinical periodontology, *ed 4, Philadelphia, 1972, W.B. Saunders Company, Figures 2-3 and 2-8, pp 34 and 38, respectively.*

5-2). As part of the remodeling process, bone is removed by osteoclasts that are thought to be derived from the *mesenchymal cells*. Cementum is removed by specialized cementoclasts, which are also derived from mesenchymal cells. Nerve endings attach to the PDL and to the blood vessels that surround them. The nerve endings are effective for proprioception and for the perception of pain. The *interstitial fluid* found within the PDL space is derived from the vascular network. It serves as a nutrient source, and it contributes to the shock-absorbing mechanism to better withstand the physiologic forces as well as any pathologic forces that are applied directly to the teeth.

Response of the Teeth and the Periodontium to Normal Occlusal Function

During normal chewing function, the teeth are subjected to intermittent forces that are then transmitted to the periodontal structures.[55,88,89,92,118,192,209] When forces are applied to a tooth, the load is dissipated by the periodontal ligament and the interstitial fluid within the tooth socket. These mechanisms allow a more limited force to then be transmitted to the cortical bone that surrounds the tooth socket (i.e., the lamina dura), which "bends" in response. The term *bending* is used to describe the physiologic response to normal masticatory forces. The bone bending that occurs in response to normal (chewing) function is thought to be an important stimulus to osseous regeneration and repair. This intermittent but repetitive stress loading allows for the modification of the bony architecture as an adaptation to functional demands. If pressure against the tooth is maintained (as opposed to being intermittent, as when chewing), the interstitial fluid within the tooth socket is expressed (i.e., pushed out) and the tooth itself is displaced, thereby compressing the PDL directly against the adjacent bone. This is perceived by the individual as being quite painful, and, if the force is excessive and prolonged, a different physiologic response occurs. This "trauma from occlusion" is a response in which the remodeling of the adjacent compressed bone occurs. A definition of the term *traumatic occlusion* was first provided by Stillman in 1917. He stated that traumatic occlusion is "a condition where injury results to the supporting structures of the teeth by the act of bringing the jaws into closed contacts."[171] The definition given by the American Association of Periodontics in 1986 is "an injury to the attachment apparatus as a result of excessive occlusal force."[171] *Primary occlusal trauma* involves excessive forces on a normal periodontium, and *secondary occlusal trauma* is considered to have occurred when normal forces are placed on a compromised periodontium (see Chapter 6).

The intentional application of these principles is how controlled orthodontic tooth movement occurs. The application of light prolonged vector-controlled forces allows for more or less predictable man-made movements of the teeth. Interestingly, nature's application of these light prolonged forces on the teeth can also occur from maintained posturing of the muscles of the lips, cheeks, and tongue as they press against the teeth, thereby causing dental movement and remodeling of the alveolar ridge. When postural forces from the lip, cheek, or tongue are pathologic, they may contribute to the classic dental compensations that are seen with each specific pattern of developmental dentofacial deformity.* These environmental forces may then be compounded by iatrogenic orthodontic dental compensation maneuvers to further affect periodontal health. For example, consider the retroclination of the lower anterior teeth that is seen with Class III skeletal patterns (Fig. 5-3) (see Chapter 20) or the proclination of the lower incisors that is seen with skeletal Class II cases (Fig. 5-4) (see Chapter 19). In both instances, negative periodontal sequelae may occur.

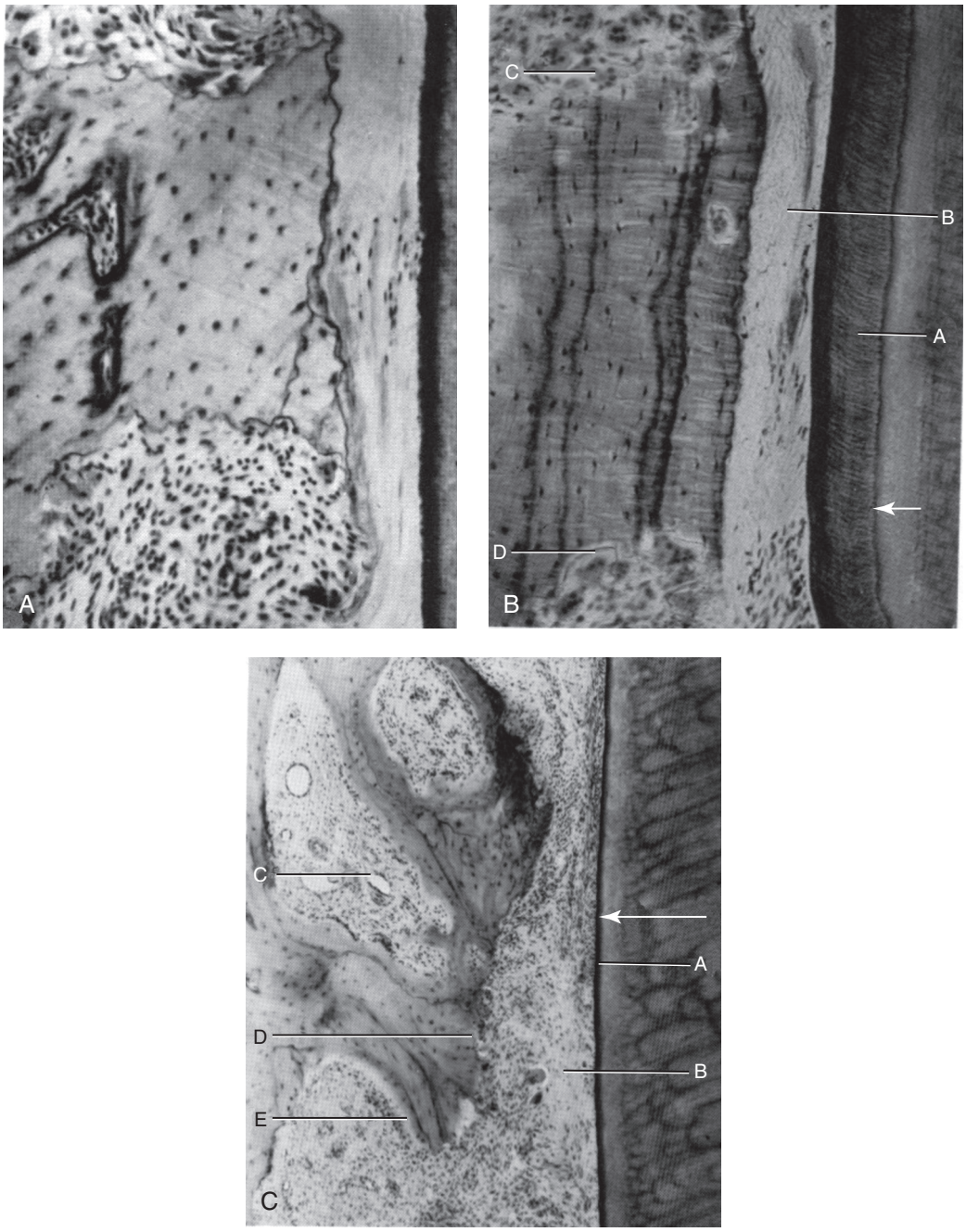
Orthodontic Tooth Movement

The Periodontal Ligament and Bone Response to Sustained Orthodontic Force

The specific anatomic response to orthodontic forces is dependent on the force magnitude. *"Heavy" forces* often result in pain, necrosis of cellular elements within the PDL (i.e., hyalinization), and remodeling with the resorption of the alveolar bone that is being compressed (i.e., the hyalinization zone). It may also result in the resorption of the root apex, remodeling with the loss of crestal bone, and fenestrations or dehiscence through the cortical plates. When *light and constant forces* are applied to the tooth, there is compression without necrosis of the PDL elements and remodeling of the tooth socket without significant irreversible necrosis. As long as the tooth is being moved into adequate alveolar bone and the forces are light, the root of the tooth is less likely to resorb, and the alveolar crest height and cortical plates are for the most part preserved.[30] The objective of the orthodontic movement of a tooth is to produce the desired new tooth position as quickly as can be done, with as little damage as possible to the tooth, the PDL, the alveolar bone, and the adjacent teeth.

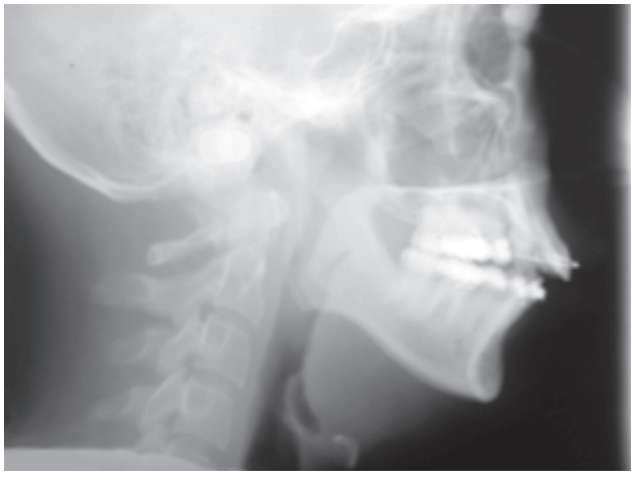
There are two major theories to explain the observed process of orthodontic human tooth movement: the *bioelectric theory* and the *pressure-tension theory.* The bioelectric theory holds that the changes in bone metabolism that are required to effectively move teeth and their sockets are controlled by the electric signals produced when alveolar bone bends and the crystalline structure is distorted. The pressure-tension theory presumes that cellular changes occur when pressure or tension is applied within the PDL or tooth socket, which then produces chemical changes. The pressure and tension that occur within the PDL result in specific blood flow changes and the release of chemicals that

*References 50, 58, 75, 76, 91, 93-97, 119, 127, 134-136, 141, 150-152, 167, 213, 216, 229



• **Figure 5-2** **A,** Undermining resorption in a 12-year-old child. This is a view from a short-rooted premolar tooth. The cementum that covers the root surface has not been broken, although the periodontal membrane is hyalinized at the area of greatest pressure. A spicule of bone is being attacked from above and below, away from the compressed PDM. **B,** Undermining resorption is taking place in a 39-year-old adult. No osteoclastic activity occurs in the compressed PDM, but osteoclasts are at work on bundle bone that has been laid down previously from above and below the area of greatest pressure. *A,* Thick cementum layer. *B,* Hyalinized periodontal ligament. *C* and *D,* Frontal undermining bone resorption on both sides of the cell-free area. **C,** Tooth movement with light forces. This image shows the pressure side during the period shortly after hyalinization. *A,* Root surface. *B,* Remnants of cell-free, formerly hyalinized tissue. *C,* Large marrow space in alveolar bone. *D,* Direct bone resorption. *E,* Compensatory bone formation in the marrow space as a response to bone resorption on the side of the periodontal ligament. **A** *courtesy K. Reitan from Graber TM:* Orthodontics: principles and practice, *ed 3, Philadelphia, 1972, W.B. Saunders Company, Figure 10-5, p 496.* **B** *courtesy K. Reitan from Graber TM:* Orthodontics: principles and practice, *ed 3, Philadelphia, 1972, W.B. Saunders Company, Figure 10-5, p 496.* **C** *courtesy K. Reitan from Graber TM:* Orthodontics: principles and practice, *ed 3, Philadelphia, 1972, W.B. Saunders Company, Figure 10-6, p 497.*

• **Figure 5-3** A 34-year-old man arrived for the evaluation of a long-standing jaw deformity with malocclusion, chronic obstructive nasal breathing, and periodontal issues. Since his early childhood years, he was known to have a maxillary deficiency with a relative mandibular excess growth pattern. The family elected a camouflage orthodontic approach that included four bicuspid extractions and the introduction of dental compensation in an attempt to neutralize the occlusion when he was 10 to 15 years old. He was a forced mouth breather as a result of the associated increased nasal airway resistance. He underwent septoplasty when he was 20 years old, but this did not result in significant improvement. His general dentist confirmed labial bone loss and gingival recession of the mandibular anterior and maxillary posterior dentition. He was referred to this surgeon, and a comprehensive orthognathic and dental rehabilitation approach was recommended. Consultations with a periodontist, an orthodontist, and an ear, nose, and throat specialist were carried out. The patient was confirmed to have residual deviation of the septum and enlarged inferior turbinates, which explained the continued difficulty that he had with breathing through his nose. A degree of root resorption of the anterior dentition was confirmed. The need for gingival grafting to attain improved root coverage and to generate a wider band of attachment was recommended (i.e., teeth nos. 3, 11, 14, 19, 20, 23 through 26, 29, and 30). This will be followed by the orthodontic removal of dental compensation and then orthognathic and intranasal procedures to improve long-term dental health, to enhance facial aesthetics, and to open the upper airway. **A,** Frontal facial and occlusal views before retreatment. **B,** Profile facial view and lateral cephalometric radiograph before retreatment. **C,** Panorex radiograph with retreatment in progress.



• **Figure 5-4** A man in his early twenties with a primary mandibular deficiency growth pattern. During his teenage years, he underwent failed growth modification attempts followed by orthodontic camouflage maneuvers that included four bicuspid extractions in an attempt to neutralize the occlusion. Deterioration of the periodontium with labial bone loss and gingiva recession along the mandibular anterior dentition is evident. The patient was referred to this surgeon, and a comprehensive orthognathic and dental rehabilitation approach was approved. **A,** Frontal facial and occlusal views before retreatment. **B,** Profile facial view and lateral cephalometric radiograph before retreatment.

affect cellular function, thereby causing the observed changes in bone architecture. These two theories of bone metabolism are also discussed and debated within the orthopedic literature, and they are not mutually exclusive. Many scientists and clinicians believe that both biologic mechanisms play a role in the remodeling and repair of bone. The pressure-tension theory represents the classic theory of tooth movement. It proposes that chemical rather than electric signals are the stimuli for the cellular differentiation required to alter the tooth socket and ultimately to move the teeth. In either case, when an alteration in blood flow within the PDL occurs and is sustained through pressure that is applied directly to a tooth, the tooth shifts position within the PDL space, thereby compressing the ligament on one side and stretching it on the other. Blood flow is decreased where the PDL is compressed, although it may be increased or maintained in areas in which the PDL is under light tension. Interestingly, if the PDL is overstretched (i.e., if "heavy" tooth forces are applied), then the blood flow may also be decreased on the tension side. It is easy to understand how these alterations in blood flow can create changes in the chemical environment. Oxygen levels fall in the areas of compression, although they may even increase on the tension side of the tooth socket. The changes in oxygen content cause a series of biologic responses that bring new cells into the area either through migration or differentiation.

The Amount of Orthodontic Force Applied to the Teeth

The *heavier the sustained pressure,* the greater the reduction in blood flow where compression is occurring in the PDL.[25,166,174] When only *light prolonged force* is applied to a tooth, the partially compressed PDL experiences diminished blood flow, and the interstitial fluids are expressed (pushed out) from the PDL space as the tooth moves within the socket.[125] Animal experiments confirm that, within a few hours, there will be increased levels of cyclic adenosine monophosphate (cAMP).[175] cAMP is known for its importance in the alteration of cell functions. Within 4 hours of continued sustained low pressure, mesenchymal cell differentiation occurs. From a clinical perspective, it is known that, if orthodontic appliances are used for less than 4 to 6 hours per day, not enough sustained force will be applied, and little or no tooth movement will occur.[143,148]

For the tooth to actually move further than just within the socket itself, osteoclasts must arrive (i.e., migrate or differentiate from mesenchymal cells) and then remove bone from the area adjacent to the compressed part of the PDL. Osteoblasts are required to form new bone on the tension side. Prostaglandin E (a family of prostaglandins that are often used pharmaceutically in medicine) has been shown to increase its levels during this phase of orthodontic tooth movement. It is known that prostaglandin E stimulates both osteoclastic and osteoblastic activity, thereby leading to the belief that it is an important mediator of tooth movement.

It is known that the osteoclasts attack the compressed area of lamina dura to remove bone.[184] The radiographic appearance of a widened PDL (on the tension side) in the presence of light prolonged orthodontic forces indicates that tooth movement is soon to begin.[66] On the compression side, osteoclasts are removing the lamina dura. On the tension side, remodeling activity involves osteoblasts laying down new bone. The course of events is different if the prolonged force applied to the tooth is too great. If the force transmitted to the periodontal ligament totally occludes the blood vessels, thereby cutting off all blood supply to the area, generalized necrosis occurs within the compressed area. Although some avascular areas in the PDL will occur even with light forces, if the predominant forces are "heavy," then the response is necrosis. Irreversible damage can then occur, and this includes remodeling with the loss of crestal bone, root resorption, and cortical plate dehiscence. The histologic findings within the PDL—even with light forces during the compression process—show the disappearance of cells within the vessels. The histologic appearance is descriptively said to be "hyalinized" because of its visual resemblance to hyaline connective tissue. After this hyalinization process begins, after several days of delay, the remodeling of bone that borders the sporadic necrotic areas of the PDL will occur as osteoclasts (derived from adjacent undamaged areas) arrive and do their work. The process of bone removal is called *undermining resorption.* This term is visually descriptive when it is viewed under the microscope, because the attack by the osteoclasts is from the underside of the lamina dura. After the *hyalinization* (i.e., the compression of blood flow with isolated necrosis) and the undermining resorption (i.e., the osteoclastic activity on the underside of the lamina dura) have "softened" the lamina dura, tooth movement is soon to occur. The chronology of tooth movement with *frontal resorption* is different than that of undermining resorption. With frontal resorption, a steady attack on the outer surface of the lamina dura results in smooth, continuous tooth movement. With undermining resorption, there will be a delay in the removal of the bone adjacent to the tooth. When heavy force (i.e., pressure) is again reinitiated, there will be a similar delay in tooth movement until a second round of undermining resorption can occur.[171]

The orthodontic movement of a tooth is clinically more efficient when there are only minimal areas of PDL necrosis. As a result, only limited levels of pain are experienced by the individual undergoing treatment. Unfortunately, neither aspect is completely avoidable. To date, a completely smooth progression of tooth movement with consistent light force over time has proven to be unattainable within the human biologic system. One important compounding factor is that the individual who is undergoing orthodontic tooth movement must also continue with the everyday functions of mastication, speech, swallowing, and breathing within a

24-hour cycle. These functions also place forces on the teeth, which may either conflict with or expedite the applied orthodontic forces. Another factor is the relative ineffectiveness of the appliances (i.e., the materials) used to apply sustained appropriate levels of force. To review, in clinical orthodontic practice, on one end of the spectrum, too much sustained force is detrimental to the tissues; alternatively, too little force will not produce the tooth movement that is desired.[25]

Control of Orthodontic Tooth Movement

The basic principle of the ideal orthodontic movement of a tooth includes the application of just enough force to stimulate required cellular activity without the complete occlusion of the blood vessels within the PDL. For example, it is estimated that as little as 30 g of force may be required to tip a single rooted tooth. For controlled resorption and remodeling to occur, force is first delivered to the tooth and then to the PDL, which results in compression, tension, or both at the appropriate locations of the lamina dura. Depending on the desired three-dimensional repositioning of the tooth, different amounts of pressure (i.e., the force per unit area) are applied to different locations along the lamina dura.

The different basic forms of tooth movement include tipping, rotation, and translation. *Tipping* results from a single force applied at a distance to the center of resistance. Although the center of resistance of a tooth is generally located about halfway down the root, the center of rotation for tipping movements is apical to that. Here, the crown will move in one direction, and the apex will move in the opposite direction. When the tooth tips in this fashion, the PDL is compressed most strongly near the root apex on the same side as the force being applied as well as at the crest of the alveolar ridge on the opposite side. Progressively less pressure is created as the center of rotation is approached, where there is minimum pressure applied. During *translation* movement of the tooth, the PDL space is loaded uniformly from the alveolar crest to the apex on the side of tooth compression. A pure translational force will allow for the bodily movement of the tooth rather than tipping. The forces that are required for the bodily movement (i.e., translation) of incisors up to multi-rooted teeth is in the range of 70 to 120 g. This represents the maximum orthodontic tooth movement force to be used in clinical practice. The optimal force for the orthodontic *rotation* of a tooth around its long axis (i.e., 35 to 60 g) is less than that required for the bodily movement of a tooth. The optimal force required for the orthodontic *extrusion* of a tooth is in the range of 35 to 60 g. The *intrusion* of teeth requires the carefully controlled application of consistent very light forces that are applied to the tooth in a specific way. It is difficult to apply controlled intrusion forces to a tooth (i.e., 10 to 20 g) without also applying tipping forces. Small implants (i.e., temporary anchorage devices [TADs]) can be used to help this aspect of tooth movement and to provide greater control, as described later in this chapter.

Control of Force Duration and Decay in the Orthodontic Movement of Teeth

A basic principle in the process of orthodontic tooth movement is the application of *sustained force*. The force must be present for a significant percentage of the time during the whole process of tooth movement. Animal experiments suggest that, only after force is maintained for approximately 4 to 6 hours, will levels of cAMP be measurably increased in the PDL. Appropriate levels of cAMP are thought to be necessary for the stimulation of cellular differentiation. Human clinical experience suggests that a force duration of at least 4 to 8 hours (per 24-hour cycle) is required to effect tooth movement. The teeth can be moved more rapidly if force is maintained for longer durations. In fact, the application of continuous light force (24 hours per day) would produce the most efficient tooth movement. Through technical advances in materials and appliances, the ability to apply continuous force and to control the duration of that force to each tooth has greatly improved. For example, the nickel-titanium alloys and the self-ligating brackets that are in use today have advanced the process of the sustained duration of light force to achieve tooth movement over time. Office visits can often be spaced every 6 to 10 weeks. Applied orthodontic forces are classified as either continuous or interrupted (intermittent). In general, the orthodontist would like to apply *continuous force* (or at least some appreciable fraction of the original applied force) from one patient visit to the next. Orthodontists also use *intermittent forces* for specific purposes. The so-called activated appliances (e.g., removable plates, headgear, elastics) may be used intermittently.

Medication Effects on the Rate of Orthodontic Tooth Movement

The bisphosphonates act as inhibitors of osteoclast-mediated bone resorption, which by definition have an effect on bone remodeling.[5,102] There is evidence to suggest that tooth movement in patients who are receiving parenteral bisphosphonate therapy may be retarded, with a subsequent risk for osteonecrosis of the jaws.[113]

Bartzela and colleagues conducted a systematic review of the literature regarding the effects of medications and dietary supplements on the rate of experimental orthodontic tooth movement.[23] The results of their literature search indicated the following:

- The therapeutic administration of eicosanoids results in increased tooth movement.
- Nonsteroidal anti-inflammatory drugs decreased tooth movement; however, non-nonsteroidal anti-inflammatory analgesics (e.g., Paracetamol [acetaminophen]) had no effect.
- Corticosteroid hormones, parathyroid hormone, and thyroxin have all been shown to increase tooth movement.

- Estrogens probably reduce tooth movement, although no direct evidence is available.
- Vitamin D_3 stimulates tooth movement, and dietary calcium seems to reduce it.

The Role of Anchorage in the Orthodontic Movement of Teeth

The term *anchorage* as it applies to orthodontics refers to the nature and degree of resistance to displacement provided by an anatomic unit (e.g., molars and premolars, temporary skeletal anchorage devices, hard palate) when it is used for the purpose of effecting tooth movement.[139]

The principle of anchorage in the orthodontic movement of teeth is an application of Sir Isaac Newton's realization that action and reaction are equal and opposite. Restated, this means that, for every force that is directly applied, there is an equal and opposite (reciprocal) force. Depending on how the orthodontic force is applied to a tooth and given the fact that each tooth has a different resistance value to tooth movement (as a result of the nonuniform nature of the teeth and the periodontium), the orthodontist may choose to use certain teeth or groupings of teeth (e.g., ligating molars together as a unit) for "anchorage" when moving other teeth into a more desirable position. Sources other than teeth may also be used for anchorage, including TADs, headgear, a facemask, the hard palate, or the lingual or buccal alveolar supporting bone of the mandible.

Dental anchorage is most typically used to overcome resistance to the orthodontic movement of teeth. To assess the anchorage requirements of the desired tooth movement, the clinician must first evaluate the part of each tooth that is anchored into the alveolar bone. The overall anchorage requirements primarily include the number of roots and the shape, size, and length of each root of the teeth that require movement. This is primarily the sum total of the surface area of the root portions of the teeth that are enveloped by alveolar bone and that are to be moved. A tooth (root) with a large surface area (enveloped by alveolar bone) is more resistant to displacement than one with a smaller surface area. A multi-rooted tooth is more resistant to displacement than a single-rooted tooth. A long-rooted tooth is more difficult to move than a short-rooted tooth. A triangular-shaped root offers greater resistance to movement than a conical-shaped root. Other factors are also important, including the following: (1) the relationship with the contiguous teeth; (2) the ongoing forces of occlusion; (3) the age of the patient and the soft-tissue forces on the teeth (i.e., cheeks, tongue, and lips); and (4) the individual's unique tissue response. Thus, molars are generally good teeth to use for anchorage, because they have deeper and longer roots, they have a greater root surface area, and they are less susceptible to tipping forces. As stated previously, anchorage may also be increased by ligating multiple teeth together, thereby summing their individual resistance. Anchorage forces may also be provided by the hard palate, the buccal cortical plate of the posterior mandible, the head (headgear), the face (a face mask), or implants into bone (TADs).

Reciprocal Tooth Movement

When planning orthodontic therapy, the clinician must consider not just the tooth movement that is desired but any *reciprocal effects* that may occur throughout the dental arches as well. Reciprocal effects must be controlled to achieve the desired repositioning of all of the teeth and to establish preferred occlusion. In a completely reciprocal situation, Newton's theory is easy to understand. When the forces applied to similar teeth and to similar arch segments are equal, there will be an equal force distribution in each PDL with this same degree of movement. A classic clinical case analysis that is frequently used to understand the "reciprocal/nonreciprocal" concept is to consider the type of tooth movement that occurs when an elastomeric chain is placed across a first premolar extraction site in the maxilla. This pits the central incisor, the lateral incisor, and the canine in the anterior segment in opposition to the second premolar and the first molar in the posterior segment. The same total force would be felt by the three anterior teeth and the two posterior teeth as the action of the chain is equal in each segment. However, equal reciprocal movement would require the same total PDL surface area over which the force is distributed. Because the measured PDL area for the two posterior teeth is larger than the three anterior teeth, the movement will not be reciprocal. In this typical, real-life clinical setting, the anterior teeth move back into the bicuspid extraction sites more than the posterior teeth migrate forward. If it is desired that the anterior teeth move entirely into the extraction space, reinforced anchorage will be required. This may be accomplished by incorporating the second molar into the posterior unit. This changes the root surface area ratios, which results in additional retraction of the anterior teeth. Other methods of reinforcing posterior anchorage include the use of extra oral force, such as headgear, TADs, and palatal support through the use of Nance appliances or a transpalatal bar.[148]

Cortical Walls as Limiting Factors to Orthodontic Tooth Movement

Cortical bone is more resistant to resorption; therefore, tooth movement is slowed whenever the root to be moved contacts it. This can be problematic, for example, when there is a dense cortical layer of bone formed within the alveolar process of a previously (long-ago) extracted tooth. It can be more difficult to close an "old" extraction site, because tooth movement is slowed to a minimum when the root encounters cortical bone along the resorbing side of the alveolar ridge.[118,126] Tooth movement is also greatly slowed and root resorption more likely when a tooth (or tooth root) is forced against a cortical plate (e.g., a labial plate, a lingual plate), such as may occur during attempts to correct incisor inclination.

In theory, the optimal force level for the orthodontic movement of a tooth is the lightest force (and the resulting pressure) needed to produce the near-maximum preferred response.[176] Forces that are more than "optimal"—although effective for the production of tooth movement—are both unnecessary and possibly traumatic to the tooth, the periodontium, the alveolar ridge height, and the cortical plates.

Negative Sequelae of Orthodontic Forces

Mobility and Pain

During the usual orthodontic movement of teeth, the PDL fibers are stretched on the tension side, which is seen radiographically as a widened PDL space. At the same time, the PDL fibers are compressed on the pressure side, which results in a narrowed PDL space. If forces are excessive, greater degrees of tooth mobility can be anticipated, and this will also involve a complaint of pain from the patient. A similar sequence of events can occur with heavy clenching or grinding of the teeth or in the presence of a traumatic occlusion, which results in excessive force to individual teeth. Excessive mobility will generally correct itself if the force is removed quickly enough.

Heavy pressure applied to the tooth will also result in immediate pain transmitted through the nerve fibers in the PDL. Several hours after normal orthodontic forces are first applied, teeth typically have at least some sensitivity to pressure, such as with the biting of hard objects. Within 2 to 4 days, the pain should dissipate considerably. With current therapeutic orthodontic mechanics, the forces applied should result in very little immediate discomfort.

Effects on the Pulp

The normal therapeutic level of sustained light orthodontic forces on the crowns of the teeth should have little effect on the pulp. The loss of tooth vitality during orthodontic treatment has occasionally been reported in previously traumatized teeth. This should be a rare consequence of orthodontic therapy when light continuous forces are being applied.

Effects on Root Structure

Recent research indicates that, when therapeutic orthodontic forces are applied, there is a degree of injury to the cementum. In current clinical practice, with the use of continuous light forces, this occurrence is less frequent. The repair of any limited cemental injury generally occurs. Alternatively, heavy orthodontic forces—whether continuous or intermittent—may lead to root resorption, because the cementoclasts remove not only cementum but also dentin of the root (Fig. 5-5). The permanent loss of root structure only occurs when an island of cementum and dentin becomes completely separated from the rest of the root surface. When this happens, resorption without reincorporation occurs.[34,37,84,103,121] The permanent loss of root structure related to orthodontic treatment is generally seen at the root apex.

The resorption of the tooth roots during orthodontic treatment has been documented to occur in three distinct forms: (1) moderate generalized resorption; (2) severe generalized resorption (see Figs. 5-5 and 5-6); and (3) severe localized resorption. The shortening of the root length of the maxillary incisors (i.e., severe localized resorption) is seen more often than this condition appears to occur in the other teeth (Fig. 5-7). Although some studies have shown that measurable root resorption can be identified in 90% of maxillary incisors and in more than half of all other teeth during a typical fixed orthodontic appliance treatment, the shortening is almost imperceptible, and it is clinically insignificant for most. Nevertheless, occasionally the loss of one third or one half or more of the root structure is observed in patients who underwent what was considered to be routine orthodontic treatment. When this does occur, it is either severe generalized resorption or severe localized resorption. Fortunately, severe root resorption of all orthodontically treated teeth is not common. When severe generalized resorption does occur, it is often seen without any orthodontic or other forms of active dental treatment, and it may be of an uncertain etiology. If there is evidence of root resorption before orthodontic treatment, the patient should be considered at high risk for further resorption during orthodontic treatment. Radiographs should be taken during treatment to watch for this occurrence. If resorption becomes apparent, the patient is informed, and the treatment plan altered. In contrast with severe generalized resorption, severe localized resorption is more frequently associated with specific forms of orthodontic treatment. Risk factors are likely to include heavy continuous orthodontic forces being place on the teeth and a prolonged duration of treatment. Localized resorption is more frequently seen in the maxillary incisors, when the root apices are forced against the facial cortical plate during treatment. For example, in the clinician's attempts to compensate for a long face Class II skeletal pattern with an anterior open bite, the maxillary incisor crowns may be detorqued (resulting in a loss of proper buccolingual inclination) and vertically lengthened as the bicuspid extraction sites and the open bite are closed. This may cause the incisor roots to come into heavy contact with the labial cortical plate and thus result in so-called "round tripping." Forces must then be reapplied to the incisors to establish adequate palatal root torque.

Effects on the Alveolar Ridge Height, the Cortical Plates, and the Gingiva

When teeth naturally erupt, they bring alveolar bone with them. It is also known that teeth may overerupt and cause excess alveolar bone height. For example, in the presence of a constant open mouth posture during growth, the hypereruption of the maxillary molars will occur (see Chapter 4). This defines the long face growth pattern (see Chapter 21).[92-97] One argument for functional appliance use is to limit this molar hypereruption. The reverse

Text continued on p. 156

• **Figure 5-5** A 14-year-old girl was referred by her orthodontist for surgical evaluation. She presented with a jaw deformity and associated malocclusion characterized by maxillary deficiency in combination with asymmetrical mandibular excess. She is congenitally missing teeth (all of the third molars, the maxillary right second molar, the maxillary left second molar, the mandibular left second bicuspid, and the mandibular right second bicuspid), and she has short dental roots. At the time of arrival, she had already undergone attempts at growth modification, including a face mask followed by rapid palatal expansion. At presentation, there was an asymmetrical Angle Class III negative overjet with a bilateral posterior crossbite malocclusion. Current maxillofacial dysmorphology and dental anomalies have negative consequences on the patient's speech, swallowing, breathing, chewing, and lip closure/posture. At this time, the patient agreed to a comprehensive facial and dental rehabilitation approach with a desire to achieve improvements in breathing, lip closure/posture, occlusion, long-term dental health, and enhanced aesthetics. After periodontal and restorative dental evaluation, limited orthodontics and then jaw and intranasal procedures were carried out. The procedures included a Le Fort I osteotomy, sagittal ramus osteotomies of the mandible, an osseous genioplasty, a septoplasty, and inferior turbinate reduction. With finishing orthodontics, restorative dentistry is planned. **A,** Frontal views in repose before and after surgery. **B,** Frontal views with smile before and after surgery.

Continued

• **Figure 5-5, cont'd C,** Oblique facial views before and after surgery. **D,** Profile views before and after surgery.

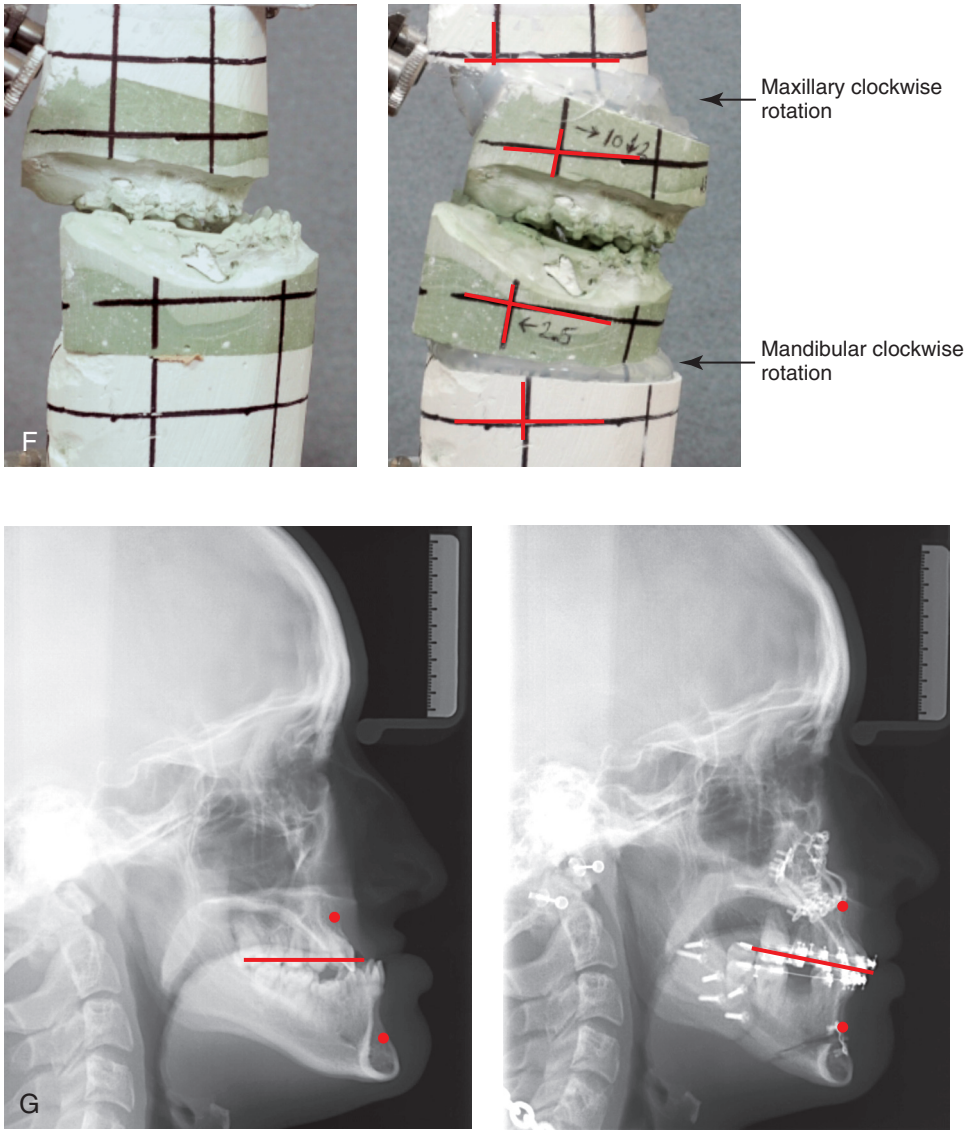
Prior to retreatment

Pre surgery

After surgery and prior to further restorative dental procedures.

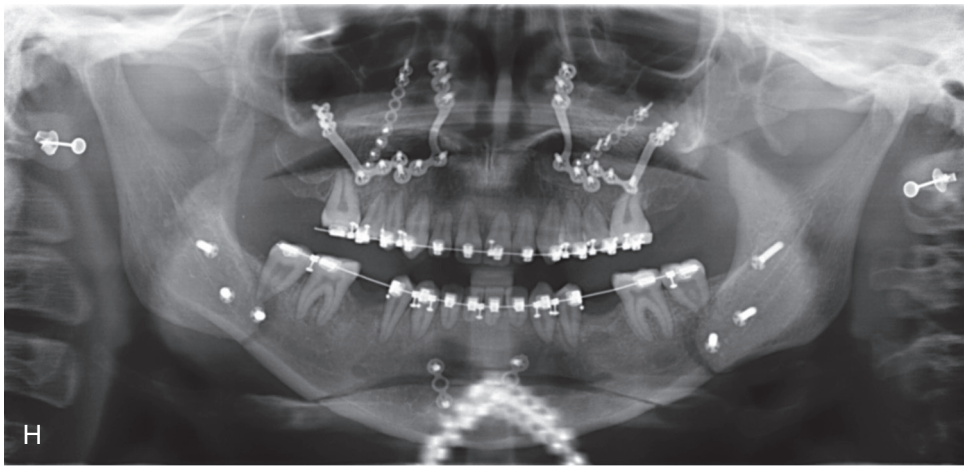
• **Figure 5-5, cont'd E,** Occlusal views before retreatment, with orthodontics in progress, and then after surgery but before final restorative work.

Continued



• **Figure 5-5, cont'd F,** Articulated dental casts that indicate analytic model planning. **G,** Lateral cephalometric radiographs before and after surgery.

Before treatment



After surgery

• **Figure 5-5, cont'd H,** Panoramic radiographs before retreatment and after surgery.

• **Figure 5-6** A 20-year-old woman was referred by her orthodontist for surgical evaluation. She presented with a narrow maxilla, a significant curve of Spee, and dental crowding. The mandibular arch also had dental crowding, tipping, and rotations. Both jaws lacked horizontal projection, and there was a severe anterior open bite malocclusion. The patient was a forced mouth breather with increased nasal airway resistance as a result of septal deviation, enlarged inferior turbinates, and a high nasal floor. The right maxillary lateral incisor was congenitally absent, and the left maxillary lateral incisor was hypoplastic and rudimentary. The shortness of the roots throughout the maxillary and mandibular dentition is presumed to be congenital. The patient agreed to a comprehensive facial and dental rehabilitation approach with a desire to achieve improvements in breathing, lip closure/posture, occlusion, long-term dental health, and enhanced aesthetics. She underwent periodontal treatment that included the extraction of tooth no. 10, grafting to the labial aspects of the mandibular incisors, and maxillary gingivectomy. This was followed by limited orthodontic manipulation and then jaw and intranasal surgery. The procedures included a maxillary Le Fort I osteotomy in three segments (horizontal advancement, vertical shortening, clockwise rotation, arch expansion, and the correction of the curve of Spee); bilateral sagittal split osteotomies of the mandible (horizontal advancement and counterclockwise rotation); osseous genioplasty (horizontal advancement and vertical shortening); and septoplasty/inferior turbinate reduction. **A,** Frontal views in repose before and after treatment. **B,** Frontal views with smile before and after treatment.

• **Figure 5-6, cont'd C,** Oblique facial views before and after treatment. **D,** Profile views before and after treatment.

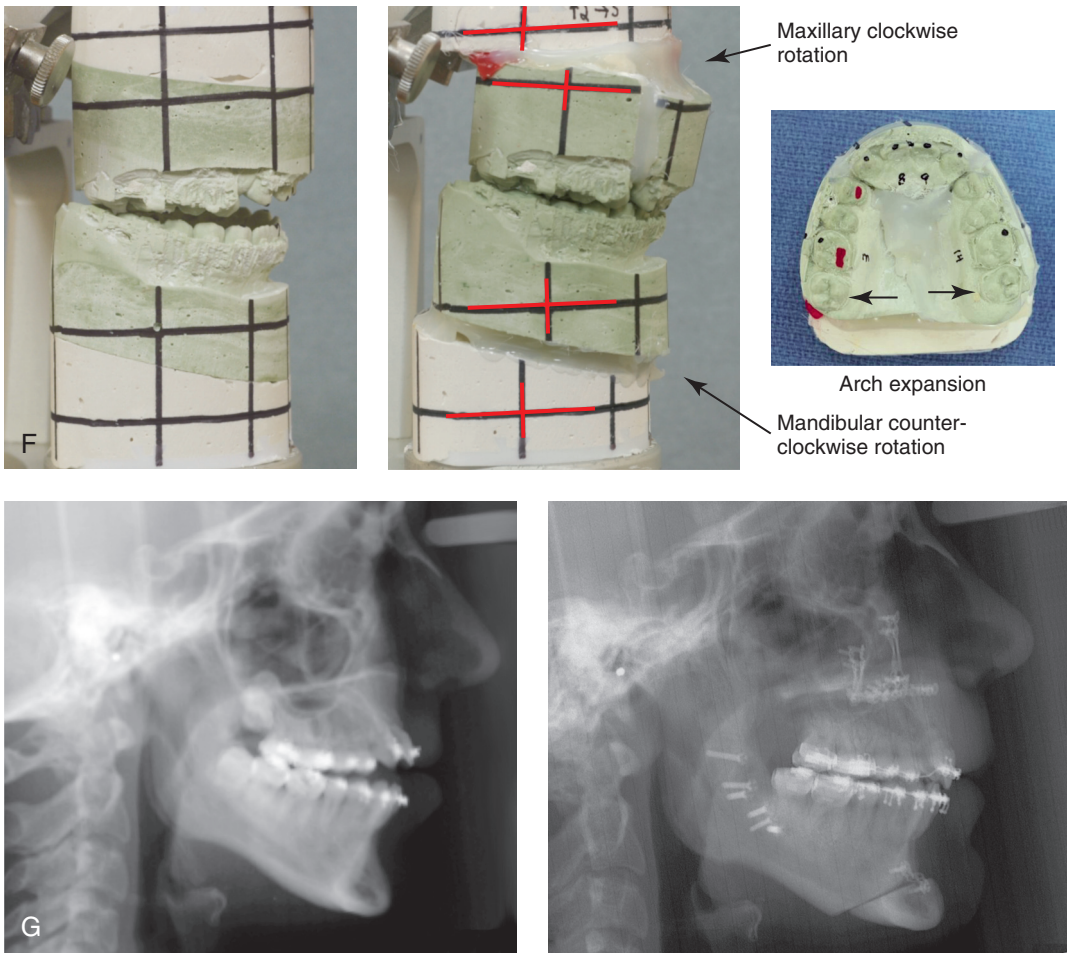
Continued

Prior to retreatment

Pre surgery

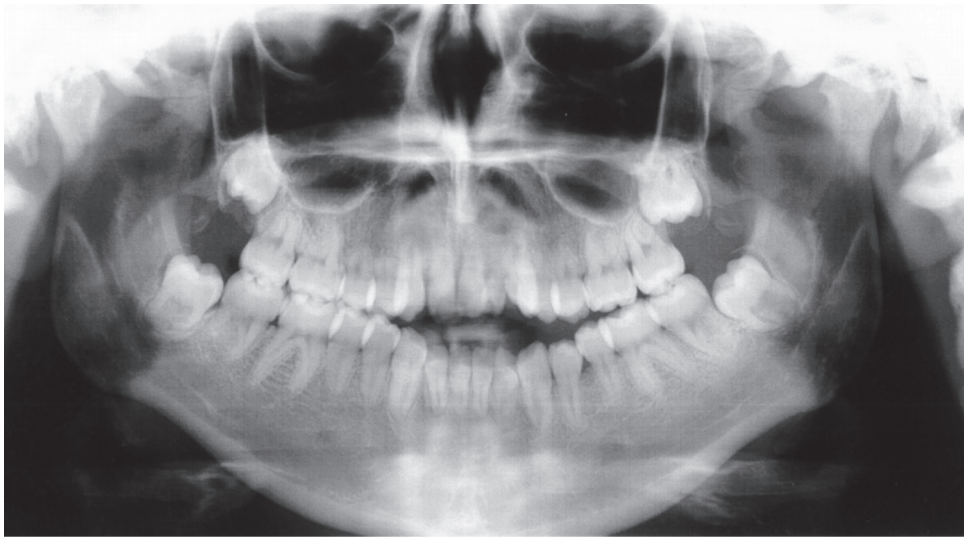
After treatment

• **Figure 5-6, cont'd E,** Occlusal views before treatment, with orthodontics in progress, and after treatment.



• **Figure 5-6, cont'd F,** Articulated dental casts that indicate analytic model planning. **G,** Lateral cephalometric radiographs before and after surgery.

Continued



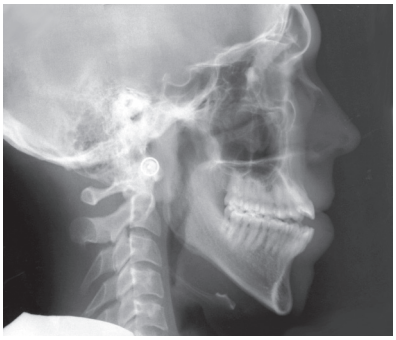
Prior to retreatment

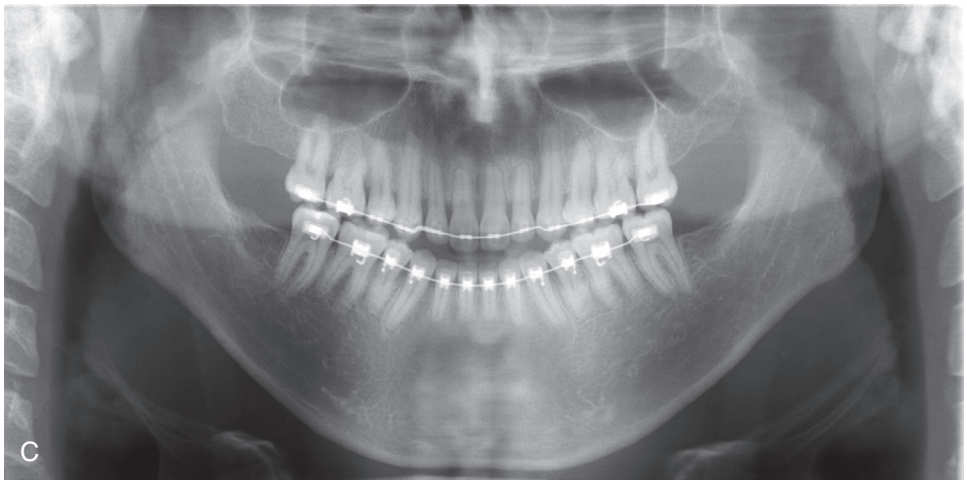
After surgery

• **Figure 5-6, cont'd** **H,** Sequential panoramic radiographs before retreatment and then after surgery.

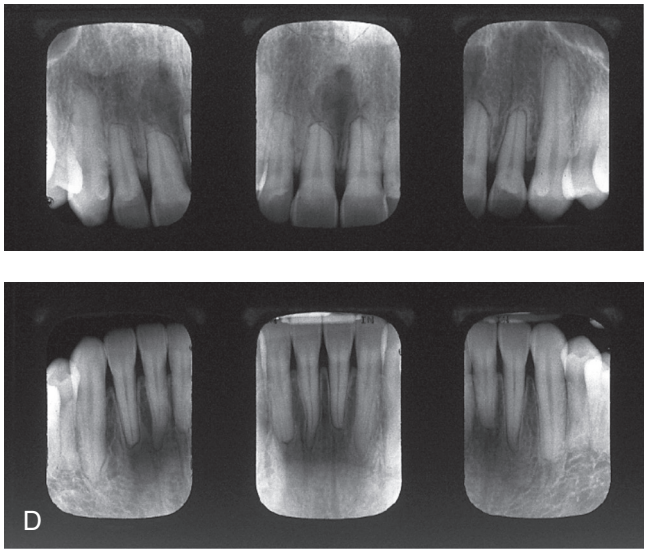
• **Figure 5-7** A 25-year-old man with a long face growth pattern. He underwent a first-stage surgically assisted rapid palatal expansion (SARPE) procedure to expand the maxillary arch width at another institution. There was a need for definitive orthognathic correction, which was carried out by this surgeon in conjunction with continued orthodontic treatment. **A,** Frontal facial view and occlusal views after SARPE procedure and before definitive orthognathic correction.

©Elsevier Inc.
Orthognathic Surgery; J.C. Posnick





Prior to definitive treatment



Prior to retreatment

• **Figure 5-7, cont'd** **B,** Profile facial and cephalometric views after SARPE procedure and before further treatment. **C,** Panorex radiograph after SARPE. **D,** Periapical radiographs of anterior dentition before further treatment. A degree of root resorption of the anterior dentition had occurred after the SARPE procedure and before definitive orthognathic correction.

effect on the alveolar process is observed at the edentulous space of a congenitally absent tooth (e.g., a retained primary molar with the congenital absence of a mandibular permanent bicuspid). The associated alveolar bone will be concave with limited vertical height (Fig. 5-8). The alveolar ridge volume deficiency at the edentulous site will also have a ripple effect, thereby preventing the full eruption of the adjacent teeth. Another example in which ridge height and volume is affected is when a maxillary canine erupts into a successfully bone-grafted alveolar cleft. Typically, the canine eruption followed by the orthodontic closure of the cleft dental gap will achieve normal vertical height and an improved arch form (see Chapters 32 and 33; Fig. 5-9).

It is also known that, if a tooth is orthodontically intruded, alveolar height will be lost, thereby leaving the same amount of root embedded in the bone as before (i.e., the reverse effect of hypereruption). In this clinical scenario, the movement of the intruded tooth's gingival margin relative to the surrounding teeth will also be observed. TADs and other orthodontic means are often used in an attempt to achieve this objective. Unfortunately, the relapse tendencies after these orthodontic maneuvers remain a concern.

Teeth can generally be orthodontically moved through the alveolus without significant damage being done to the bone.[71,155] The essential point is to move the tooth through the alveolus rather than outside of it. An exception to this is seen in the presence of active periodontal disease or when the orthodontic forces applied are excessive.[128] In this scenario, remodeling with a loss of crestal height and cortical wall dehiscence are likely negative effects to the alveolar bone. If the periodontal disease is first controlled, a normal bone response to therapeutic orthodontic mechanics should be expected (see Chapter 6).[26]

Before orthodontic treatment is initiated, the individual's biotype (i.e., the biologic predisposition of the periodontium) should be considered.[3,87] This includes an assessment of the adequacy of the gingiva and the alveolus. A thick attachment apparatus and a full-volume alveolus are desired. In an adult (even more so than in a teenager), the movement of teeth outside of the alveolar bone housing or without adequate attached gingiva is poorly tolerated (see Figs. 5-3 and 5-4). In addition, the extrusion of the incisors, the buccal tipping of the maxillary molars, and the labial version of the mandibular incisors should be limited. The adult dentition may also be compromised by wear abrasion facets, the presence of poorly designed and poorly fitted restorations, and the pretreatment loss of the periodontium. In addition to standard orthodontics, ideal treatment may require the following: (1) periodontal evaluation and management; (2) the completion of functional restorations; (3) the surgical correction of any existing jaw discrepancy; and (4) the management of edentulous space. Clarifying the willingness of the patient to maintain a hygiene program before the initiation of treatment is also a benchmark requirement to ensure success.

Mechanical Techniques Used in Orthodontic Force Control

Orthodontic tooth movement is best produced by *light continuous force.* Orthodontic appliances in clinical practice strive to use light continuous forces that are neither too great nor too variable over time.[156] The use of sound mechanical principles; an understanding of the available options with regard to materials, appliance design, and anchorage control; and the prevention of a rapid decrease in the orthodontic forces are more or less achievable.

The potential response of each tooth to the mechanical forces that can be orthodontically applied (e.g., elastic modules, wires, springs) must be considered by the clinician when he or she is designing appliances (e.g., fixed, removable) and then orchestrating anticipated dental changes. The orthodontist delivers mechanical therapy with the placement of brackets and bands on the teeth; the use of wires that connect teeth together; the use of springs, coils, and elastics; and with dental and non-dental anchorage control. However, this is always done with an understanding of the overall clinical objectives and pitfalls of treatment.

At times, it may be tempting to pursue an improved occlusion by moving teeth outside of the alveolar housing rather than coordinating treatment with either needed orthognathic procedures or with selective extractions to create necessary space[40,74,98,124,137,219,235] (see Figs. 5-3 and 5-4). These two biologic treatment pitfalls contribute to many of the everyday frustrations that are experienced in clinical practice.[31,41,47,60-62,67,132,133,149,168,195,212,214] The orthodontist should remain vigilant when assessing the degree of dental compensation introduced while attempting to correct the occlusion.[2,24,42,65,82,169,170,194,196,206]

Fixed Appliances Used in Orthodontic Treatment

Edward Angle is considered the father of modern orthodontics (see Chapter 2). His treatment was based on the development of appliances from which all contemporary orthodontics stem.[15,16,138,228] Current fixed appliances in orthodontics are all variations of Angle's edgewise appliance system.[36,112,233] By the late 1800s, orthodontic fixed appliances depended on a rigid framework that was positioned on the outside circumference of the teeth.[214] The teeth were then tied to the rigid framework to move them out to the desired arch form (i.e., expansion) as dictated by the appliance. Angle's *"E" arch* was an innovation on earlier designs. Bands were placed on the molar teeth, and a heavy *U-shaped arch wire* was attached to the banded molars posteriorly. The arch wire was positioned around the labial side of the individual teeth, which were then ligated to it. The wire was gradually unscrewed at the molar location, which resulted in expansion as the arch perimeter increased. The teeth were forced to expand with the labial wire. Unfortunately, the "E" arch was only capable of tipping the teeth into the new

• **Figure 5-8** This 22-year-old man is congenitally missing the mandibular first and second bicuspids and the right maxillary first bicuspid. He has a maxillary deficiency in combination with relative mandibular excess. He underwent earlier compensating orthodontic treatment, but an Angle Class III, negative overjet, posterior crossbite malocclusion remains. With the congenital absence of the permanent lower bicuspid teeth, there is hypoplasia of the associated alveolar ridge. The congenital absence of the bicuspids causes a ripple effect on the vertical alveolar height of the adjacent teeth. **A,** Frontal facial view and occlusal views before definitive orthognathic correction and dental rehabilitation. **B,** Profile facial and cephalometric views before further treatment. **C,** Panorex radiograph before further treatment.

• **Figure 5-9** A child who was born with van der Woude syndrome, including bilateral cleft lip and palate and lower lip pits, was followed since birth by this surgeon. The child underwent lip and palate repair, including lower lip pit excisions, during childhood. He underwent successful mixed dentition grafting and fistula closure before the eruption of the canines. As a teenager, he demonstrated maxillary hypoplasia with secondary deformities of the mandible. He then underwent a comprehensive orthodontic and orthognathic/intranasal surgical approach that included a standard Le Fort I osteotomy (vertical lengthening, horizontal advancement, and clockwise rotation) with interpositional grafting; bilateral sagittal split ramus osteotomies (clockwise rotation); osseous genioplasty (horizontal advancement); and septoplasty/inferior turbinate reduction/nasal floor recontouring (see Fig 33-3). **A,** Frontal views with smile before and after reconstruction. **B,** Profile views before and after reconstruction.

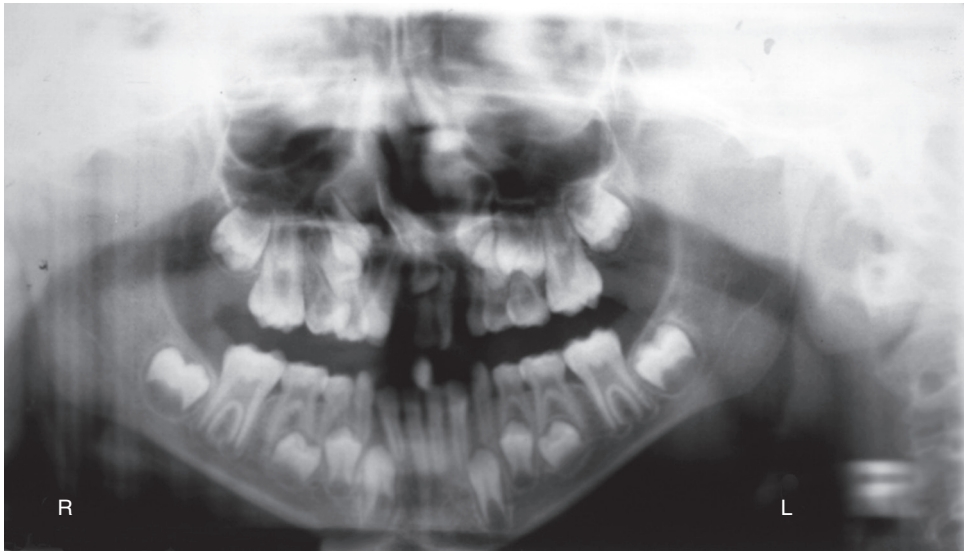
Mixed dention prior to bone grafting

Pre orthognathic surgery after successful bone grafting and orthodontic closure of cleft dental gaps

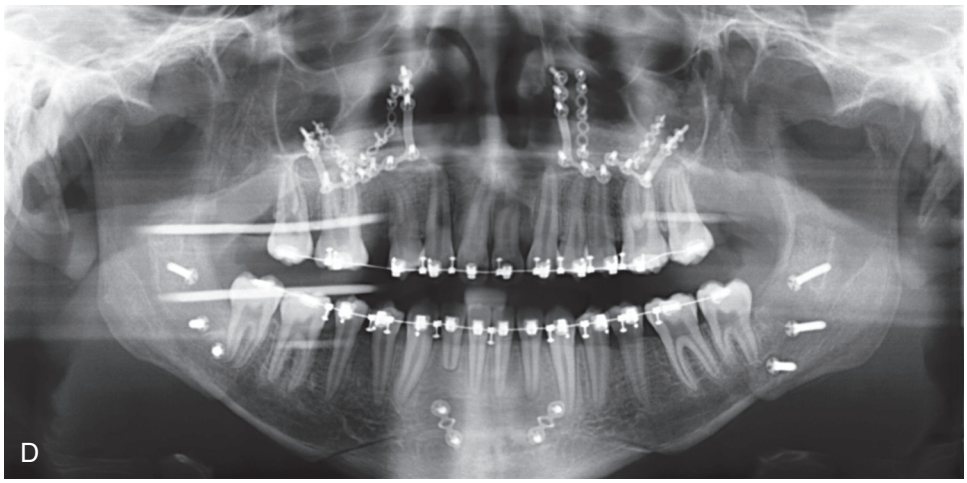
After treatment

• **Figure 5-9, cont'd C,** Occlusal views before mixed dentition bone grafting and fistula closure, with orthodontics in progress before orthognathic surgery, and then after the completion of reconstruction. The placement of a dental implant for the congenitally missing right maxillary bicuspid is planned for when the patient reaches 18 years of age.

Continued



Mixed dentition — prior to grafting



After orthognathic surgery

• **Figure 5-9, cont'd D,** Panorex radiographs of the mixed dentition before cleft bone grafting and then after orthognathic reconstruction. With effective bone grafting of the clefts in the mixed dentition followed by orthodontic closure of the cleft dental gaps, the maxillary canines and central incisors achieved a normal alveolar ridge and periodontium.

position. In addition, it could not precisely differentially position individual teeth over the dental arch. Angle then began to band each individual tooth (not just the molars), and he refined the connection of each banded tooth to the labial arch wire. This allowed him to move each tooth toward the wire at different rates to round out the arch. This technique required ingenuity and a sophisticated mechanical understanding of the process. Accomplishing these maneuvers had a steep learning curve, and it was not easily grasped by clinicians in practice. Angle's next appliance innovation modified the tube (band) that was placed around individual teeth to include a vertically positioned rectangular slot. The *ribbon arch* was made of gold wire, and it remained on the labial side of the arch.[63] The gold wire was inserted into the slot (bracket) on each banded tooth and held in place with a pin. This improved the efficiency of the alignment of malpositioned teeth. Unfortunately, these appliances still did not achieve consistent bodily movement of the teeth. Tipping of the teeth continued to be problematic. To overcome the deficiencies of the ribbon arch, Angle reoriented the slot (i.e., the shape and location of the bracket on the labial side of each banded tooth) from vertical to horizontal and then inserted a rectangular labial arch wire. He also coined the term *edgewise.* The edgewise appliance allowed for the improved control of crown and root positioning in all three planes of space. By the early 1930s, Angle's *edgewise appliance* became the mainstay of fixed orthodontic appliance therapy.[222] The *rectangular arch wire* was tied into a *rectangular slot* on the labial side of each tooth with additional wire ligatures; pins were no longer used. This further improved the control of the root position (i.e., it limited tipping) during the movement of the teeth.

Eyelets were added to the corners of the bands on each tooth, which could be tied into the labial arch wire as needed for rotation control. Angle was the first clinician to place attachments on each individual tooth to achieve the precise repositioning of each tooth toward a labial arch wire.

Interestingly, Angle's edgewise appliance system was also able to provide the level of control of root position that was necessary during the process of tooth movement to close an extraction site. If Angle had known that his appliance would be used in this way, he likely would have prevented its development. Charles Tweed, who was initially an Angle disciple, adapted the edgewise appliance for this purpose.[214] He did so after he observed significant relapse in many of his own non-extraction (i.e., arch expansion) cases. After he made a decision in favor of extraction, he was able to move the teeth bodily into the bicuspid extraction site with molar anchorage control. The teeth were then able to be retracted into the extraction sites with *closing loops,* one at a time (canines first, incisors second). In Australia, Begg adopted the *ribbon arch appliance* (rather than the rectangular wire) to close extraction sites.[223] He felt that it provided better control of the root position. He first tipped the anterior teeth with light continuous forces and then used torquing and uprighting auxiliaries to complete tooth alignment. When this was coupled with the detorquing of the incisors, root resorption sometimes occurred.

Orthodontic appliances were further modified to allow for rotational control without the need for additional ligatures.[11-14,54,107,108,111,190,191] This was accomplished by using either *twin brackets* or *extension wings with single brackets* that could contact the underside of the arch wire. Further modifications included the alteration of the bracket slot dimension, *self-ligating brackets,* and the addition of *lingual appliances.* With improvements in dental materials, *bonding attachments* (i.e., brackets) that were applied directly to the tooth surface (rather than the banding of teeth) became popular. Bonded attachments have no interproximal component and therefore require no separation of teeth, and they are less painful for the patient. They are more efficient for the clinician to put on and to remove, and they are more aesthetic because they are less visible to the casual observer at conversational distance. Clear and tooth-colored appliances have also been developed. An additional advantage to direct bonded brackets and tubes is improved access for oral hygiene and periodontal maintenance.

Contemporary edgewise appliances still remain an integral component of orthodontic treatment.[99] Brackets or tubes are customized for each individual tooth, thereby allowing for the minimum number of bends in the arch wire that are necessary to produce an ideal arrangement of the teeth. This *"straight wire"* approach requires some bending of the wire to allow for a passive fit of the arch wire when the teeth are ideally aligned.

Auxiliary attachments are components of the edgewise appliance system and include the following: (1) headgear tubes; (2) auxiliary edgewise tubes; (3) auxiliary labial hooks; and (4) lingual arch attachments. Headgear tubes can accept heavy auxiliary labial arch wires. Auxiliary edgewise tubes allow for the application of segmental arch mechanics. They are generally used for the intrusion of teeth, and they are placed gingival to the plane of the main arch wire. Auxiliary labial hooks can be used for intra-arch elastics, but they require meticulous oral hygiene. Lingual arch attachments can also be used for adjunct anchorage control.

Bracket Materials and Design

Orthodontic brackets are generally made from various classes of stainless steel.[99,112,140] More often than not, they are made by a metal injection molding process rather than milling. Clear brackets that are made of reinforced resin composite or alumina have become popular; this aesthetic option is often requested by the patient. Friction forces created between metal arch wires and ceramic brackets have largely been overcome by designs that incorporate a metal slot in the bracket or by reducing surface roughness within the slot. *Self-ligating brackets* are classified as either passive or active, depending on how the wire is held in the bracket slot. Both types of self-ligating brackets can offer low friction with a reduction in the anchorage requirements and less chair time without a loss in the quality of the results. The term *prescription* is used in orthodontics to describe the angulation, inclination, and offset values used in the bracket system to treat a particular patient's malocclusion. Traditional prescriptions have been based on the systems of either Roth or Andrews, although many variations exist.[12-14,190,191] Current prescriptions often use newer arch wire and bracket technology and involve claims of improved treatment efficiency. For example, *SureSmile* uses a variety of prescription brackets in conjunction with computer-generated, pre-bent prescription wires for faster and more accurate detailing and finishing. The *Insignia system* also uses a computer to mill special brackets for each tooth of each patient. The brackets are then bonded indirectly to each tooth with the use of transfer jigs. The *Incognito system* creates custom-made gold brackets for lingual orthodontics. This method of tooth movement has gained popularity in Europe, and it is also used in the United States. Problems with all of these prescription techniques may arise if they are improperly used by a clinician for an individual with a jaw deformity. If attempts are made to correct the malocclusion by orthodontic mechanics alone, the preset wires will result in excessive dental compensation with injury to the dentition and the surrounding periodontium as the teeth are compressed against the cortical plates and pulled out of the alveolar housing. However, when properly incorporated into a comprehensive orthodontic and orthognathic treatment plan, these newer techniques may improve results and involve reduced treatment times.

The banding of individual teeth has been used in orthodontics for more than 100 years as a method to gain attachment to the teeth. With the introduction of the acid-etch technique, the anterior teeth, as part of orthodontic therapy,

are commonly bonded rather than banded. The bonding of molar teeth has also become more common in clinical practice, which is due at least in part to improvements in light-cured adhesive technology and bracket design (i.e., a contoured bracket design and a micromesh base). In addition, the cost of bonded molar tubes has come down, and the avoidance of molar banding means that the separation of the teeth for the placement of the bands is no longer required.

Cements and Adhesives

The spectrum of cements and adhesives used in orthodontic practice includes self-etching primers, wet bonding, fluoride release to prevent demineralization, light-cured band cements, colored cements, and curing lights.* *Self-etching primers* are now available for orthodontics. They are relatively moisture insensitive, but they may have reduced bond strength as compared with the conventional acid-etch technique. The *wet bonding products* (i.e., modified resin-filled glass ionomer adhesives) that are currently available show lower bond strengths with a higher failure rate than conventional composite, but they are generally acceptable for accomplishing needed objectives. Fixed-appliance orthodontics can lead to problems with oral hygiene, increased plaque accumulation, and altered plaque ecology, which may lead to enamel demineralization. Fluoride in the oral environment has been shown to lead to altered bacterial function, reduced enamel dissolution, and increased remineralization. *Fluoride-releasing bonding cements* have been shown to reduce the depth and extent of demineralization microscopically. Unfortunately, long-term success with regard to the limiting of demineralization has not been significantly demonstrated. *Conventional glass ionomers* are still the preferred material for use beneath molar bands to limit demineralization as compared with cements that do not release fluoride. *Polyacid-modified composite cements* are now available to secure bands, and they offer a light-cured snap set. These new cements appear to clinically perform as well as conventional glass ionomer cement, but, because they do not bond to enamel, they may result in greater demineralization in some patients. High-performance *halogen curing lights* are now available; these contain bulbs that do not degrade over time, and they have curing times of 5 to 8 seconds. *Plasma arc lights* are also available; these can cure adhesive in 3 to 5 seconds, but they have the disadvantage of generating much heat during the process. Currently available *light-emitting diode curing lights* are gaining popularity; they generate low levels of heat, and they are cordless.

Arch Wire (Materials and Design)

Before the 1950s, precious metal alloys were the commonly used materials for orthodontic arch wires. Stainless steel alloys surpassed gold as the preferred arch wire materials during the 1940s to reduce costs, to provide higher-yield strength and stiffness, and to result in higher elastic modulus and flexibility with improved properties to orthodontically close dental space. These alloys generally included platinum and palladium, which were often mixed with gold and copper. During the 1970s, NASA developed a nickel-titanium alloy that was easily deformed at low temperatures but that was able to memorize its original shape with warming (i.e., the thermal dynamic shape memory effect). The term *active* has been coined to describe this phenomenon. Nitinol was then developed as a cheaper, stabilizing "passive" version of this material, and it has been found to be useful as an orthodontic wire, because it still possessed low stiffness and good recovery when it was used as an aligning or intermediate arch wire. Currently, two active nickel-titanium alloy forms of this material are used in orthodontic practice, because they both possess excellent elastic properties. This includes the *strength-induced superelastic wires* and the *thermally induced superelastic wires*. The term *superelastic* refers to a material that can undergo martensite-austenite phase transformations, which are generated by either mechanical stress or heat. The crystal structure of the wire changes with mechanical deformation, which results in a unique non-linear stress–strain curve in which stress is independent of applied strain. In theory, the force levels that are used to deform the wire to ligate it into the bracket are less than those created by the wire as it attempts to return to its original shape. This offers the advantage of low continuous forces applied to the teeth through the entire reforming process, despite the wire being tied into teeth that are severely displaced. The thermally induced superelastic wire undergoes phase change as the temperature changes from the ambient temperature to an individual's body temperature. The temperature at which the transformation takes place can be specifically controlled during the manufacturing process to match the intraoral temperature requirements. When using thermally induced superelastic wires, the wire is ligated in place during its low-stiffness phase; then, with the heat of the oral cavity, transformation is induced, thereby returning it to its predetermined austenitic form. The theoretical advantage of both of these superelastic wire types is that they are able to provide a physiologic low and continuous force that can be used for leveling and aligning the teeth. Although individual practitioners are advocates of these wires as compared with the more traditional nickel titanium or multi-strand stainless steel wires, significant differences in the speed of alignment between the wire types has yet to be proven.[42]

Intra-Oral Auxiliaries to Brackets and Arch Wires

Intra-oral auxiliaries to the brackets and arch wires include closing and opening coils, torquing and uprighting auxiliaries, elastomeric chains, other non-compliance devices, and temporary skeletal implants. *Elastomeric modules* are commonly used to secure the arch wire into the slot of a non–self-ligating bracket.

*References 10, 38, 51, 56, 105, 120, 144, 145, 153, 154, 172, 173, 177, 202-204, 232, 236

The definition of friction is the resistive force between surfaces that oppose motion. In orthodontic tooth movement, friction results from the interaction of an arch wire with the walls of an orthodontic bracket or a ligature. It is now known that friction is only a small part of the resistance to movement as a bracket slides along in arch wire.[19,39,64,122,208] In orthodontics, tooth and bracket movements have major interactions that involve the teeth, the PDLs, the alveolar bone, and the forces of mastication. For a bracket that is attached to a tooth to move relative to a wire in the bracket slot, the tooth must move. The tooth's resistance to being moved contributes to its resistance to sliding. This is particularly true when bodily movement rather than tipping is desired. Binding and notching will occur in the clinical setting but not in laboratory simulations, where only brackets and wires are involved. In review, clinical studies and the analysis of laboratory findings support the view that, in orthodontic practice, resistance to sliding has little to do with friction and instead is largely a binding-and-release phenomenon. Interestingly, resistance to sliding is about the same with both conventional and self-ligating brackets.[39]

Space closure with Angle and Tweed edgewise mechanics requires closing loops that involve the use of rectangular steel arch wires. Many clinicians prefer to use edgewise mechanics, because they provide for friction-free tooth movement. Significant disadvantages of this approach include (1) the time required to bend the wires; (2) the need to apply heavy forces across the extraction spaces; and (3) the fact that only a short time range of activation is possible, thus requiring frequent patient appointments. Since the 1990s, sliding mechanics have been used as part of various attempts to decrease the amount of force that is required to close space. The sliding technique for space closure generally uses either elastomeric chains or modules in coil springs. The elastomeric chain was introduced during the 1960s. It has the advantage of being inexpensive, but it suffers from rapid force decay of between 50% and 70% during the first 24 hours. The introduction of space-closing coil springs, which were originally made of stainless steel and cobalt–chromium–nickel alloys and more recently replaced by nickel–titanium springs, have less force decay as compared with elastomeric springs (8% to 20% versus 50% to 70%). Advocates for space closure with both techniques can be found. Inexpensive elastomeric products are still often preferred, because they are generally effective.

In an ideal world, orthodontic treatment would require no compliance from the patient. Unfortunately, removable anchorage devices (e.g., cervical and reverse pull headgear, intermaxillary elastics) are still required with various orthodontic maneuvers. The clinical use of intermaxillary traction with nickel titanium coil spring-and-piston cylinders that are attached to a fixed appliance and used to hold the mandible in a postured position are theoretically interesting, but they involve frequent breakage problems. The use of TADs (e.g., non-osseointegrated titanium micro-screws or titanium plates held in place with screws) that are placed directly into alveolar bone are now used for anchorage; these often circumvent the need for devices that require patient compliance. There is more information about TADs later in this chapter.

Removable Appliances Used in Orthodontic Treatment

The use of removable appliances to guide tooth movement has a long history and persuasive clinical advocates. There have been periods of intense use followed by waning interest. During the late 19th and early 20th centuries, the use of fixed appliances was limited by available materials, the need for labor-intensive "chair time," and an incomplete mechanical understanding of their potential by most clinicians. Removable orthodontic appliances have several immediate advantages, including that they can be fabricated in a laboratory (thereby reducing "chair time" for both the patient and the dentist) and that they can be removed at socially sensitive times or to make oral hygiene easier. Removable appliances that are used for the movement of teeth have disadvantages, including their dependence on consistent patient compliance. They are difficult to construct, thus making precise complex tooth movements problematic. Even with excellent patient compliance, carrying out complex tooth movement remains an issue.

During the early 1900s, removable upper and lower arch appliances used to produce the desired tooth movement were often made of heavy gold wires as a framework from which lighter gold finger springs were placed. The first molars were banded, and wire ligatures were used to tie in heavy labial and lingual arch wires. Interestingly, the use of removable appliances for the movement of teeth was favored in Europe during the first half of the 20th century. This was primarily for economic reasons, and there was a lack of a strong clinical advocate for fixed appliances on that side of the Atlantic. During the same time frame, Edward Angle's profound influence on clinicians in the United States encouraged the preferred use of the edgewise fixed appliance system.

Functional appliances represent a second category of removable design (although they may also be fixed). They are constructed primarily with the hope of changing the position of the mandible (i.e., to hold the lower jaw open and to posture it forward) as well as to limit or enhance the eruption of maxillary molars. The history of functional appliances can be traced back to the mid 1800s, when Norman Kingsley introduced the "bite-jump" appliance (1879).[114] The "monobloc," which was developed by Robin in the United States during the early 1900s, was followed by the "activator," which was developed in Norway by Andresen during the 1920s; both of these devices became widely accepted. Harvold reintroduced removable functional appliances to North American orthodontists during the 1960s, around the same time that the use of fixed appliances finally began to catch on throughout Europe. Harvold's animal model studies confirmed that mandibular posture did in fact affect maxillomandibular growth[92-97] (see

Chapter 4). This led to a belief that functional appliances could be used to "alter back" abnormal jaw growth tendencies. During the 1970s, the Frankel regulator by Rolf Frankel of East Germany popularized the concept of a tissue-borne functional appliance. By the 1980s, William Clark of Scotland introduced the Twin Block, which is a two-piece, tooth-borne appliance. The Twin Block was easier to wear than the Frankel regulator, and it was designed to accomplish the same objectives. Pancherz then popularized the Herbst fixed functional appliance during the 1980s, because it circumvented the problem of patient compliance. This appliance was originally developed by Emil Herbst at the beginning of the 20th century. Proffit has stated that, despite continued strong advocates of functional appliances, *"the results of human clinical trials and retrospective analysis have not confirmed sufficient success with the use of functional appliances to make them mainstay treatment."*[210] Although there is limited evidence-based research to support functional appliances, their continued use in everyday orthodontic practice remains a reality.

Siara-Olds and colleagues recently completed a prospective, randomized, controlled clinical study to assess the early and long-term treatment outcomes of tooth-borne functional appliances.[201] They tested the use of four different functional appliances in a treatment group, and they also followed a group of untreated control subjects with Class II malocclusions. The treatment group (n = 80) was subdivided evenly into four subgroups in accordance with the type of functional appliance being used: the Bionator (n = 20); the Herbst fixed functional appliance (n = 20); the Twin Block (n = 20); and the mandibular anterior repositioning appliance (n = 20). The functional appliance treatment was carried out on individuals with Class II malocclusions who were between the ages of 8 and 12 years. Each randomized Class II malocclusion subject received comprehensive orthodontic treatment with bands and brackets to complete their treatment after the use of the specific functional appliance. The investigators compared lateral cephalometric radiographs that were taken at standard intervals (i.e., before treatment, after functional appliance therapy, and after the completion of comprehensive orthodontics). The four subgroups were also compared with an untreated control group who had similar Class II malocclusions who were not treated with functional appliances, bands, or brackets. *The results of the study confirmed that the use of the functional appliances did not alter long-term mandibular growth patterns as compared with the results seen among the control subjects with Class II malocclusions.* Interestingly, the use of a functional appliance in combination with bands and brackets did demonstrate effects, including the following: (1) the restriction of maxillary horizontal growth; (2) the creation of a steeper occlusal plane (with the Herbst and mandibular anterior repositioning appliances); and (3) the steepening of the mandibular plane angle (i.e., clockwise rotation) with the labial version of the mandibular incisors with the Twin Block appliance.[201] In review, the study by Siara-Olds, et al. confirms that functional appliances for the purpose of correcting micrognathia have negligible impact on the growth potential of the mandible long term.

A third category of removable appliances are used for the retention of the achieved orthodontic results. A spectrum of *retention appliance* designs are used in everyday orthodontic practice. With regard to the issue of retention, Melsen stated the following: "We [orthodontists] deliver the product, but the final result, as when you buy a car, depends on life-long retention and life-long maintenance. Other than diamonds, everything has to be maintained; true stability only occurs post mortem."[142]

We can also add *Invisalign,* which is a product that involves a series of removable clear aligners, as a fourth category to the list of removable orthodontic appliances. It is a method that is used for the purpose of orthodontic tooth movement. Its current level of popularity is at least partially the result of an effective worldwide marketing campaign. Clinical judgment is required to determine when this option is an appropriate form of treatment for the correction of specific—but limited—patterns of malocclusion. Nevertheless, as of 2013, it is estimated that approximately 2 million individuals have completed or are currently receiving Invisalign treatment.

Accelerated Osteogenic Orthodontics

Finding a technique that enhances the speed of orthodontic tooth movement without causing damage has been an objective of clinicians for more than 100 years. Attempts to modify the balance between resorption and deposition and to bypass the waiting time for the alveolar cortex to resorb and thus move the teeth faster without causing irreversible damage to the periodontium has been the focus of much research over the years.[28,29,49,78,79,81,90,101,200,217,218,234] Corticotomy, which is the intentional injury of the cortical bone, was described in the mid 1800s as a surgical approach to correct malocclusion. Osteotomies into the cortical alveolar bone were made, and then the teeth were splinted into new positions. This concept received renewed attention when Kole published a series of clinical articles describing a different approach to the treatment of orthodontic patients.[116,117] In 1975, Duker used a dog model to demonstrate rapid segmental alveolar movement after corticotomy.[68] In 1986, Anholm and colleagues also described corticotomy-facilitated orthodontics in clinical practice.[17] William and Thomas Wilcko further modified the protocol for corticotomy-facilitated orthodontics; they trademarked their technique in 1998.[230,231] That same year, the Wilcko brothers received an endorsement for their technique from the American Academy of Periodontics. Their technique involves elevating a full-thickness mucosal flap off of the alveolar plate (either labial or lingual/palatal) directly over the teeth to be moved. They then place corticotomies (perforations) through the alveolar bone adjacent to the teeth

to be moved with the use of a burr on a handpiece. The Wilckos also recommend placing bone graft material over the surgical perforations in the cortical plate to improve alveolar volume. They then replace the mucosal flap into its anatomic location with sutures. They further claim that the technique enhances difficult tooth movement (i.e., molar intrusion) and that it can provide relative anchorage (i.e., a corticotomy limited to the anterior region will provide relative anchorage from the posterior teeth). The theory behind this technique is that injury to the bone (i.e., the cortical perforations) creates a "regional acceleratory phenomenon."[230] As a result, the body rapidly sends cells to the site to heal the injured bone. This surge in cellular healing activity is thought to promote faster tooth movement. The Wilckos called their technique *accelerated osteogenic orthodontics.*[230]

The animal research by Lino and colleagues[130] and the experimental study completed by Sebaoun and colleagues[197] describe a plausible biologic explanation for the greater speed of tooth movement that is seen with these techniques. **Lino's group** used adult beagle dogs (n = 12). The mandibular right and left first premolars were to be moved mesially with the use of a calibrated spring that attached to the mandibular canines. On one side of each mandible, a flap was elevated, and holes were placed in the alveolar bone (corticotomies) to create a regional acceleratory phenomenon. No corticotomy was performed on the opposite (control) side. The PDL on the mesial (compression) side of each premolar was evaluated histologically at weeks 1, 2, 4, and 8. In addition, the amount of mesial bicuspid tooth movement on each side (i.e., the experimental side versus the control side) was compared at the 8-week interval. Histologic comparisons of the two sides showed very distinct differences. At 1 week, bicuspid tooth compression produced a typical hyalinization of the PDL on both sides. "Hyaline formation" within the PDL, which is referred to as a "sterile necrosis," occurs in response to compressive forces. This is the expected response when significant compressive forces are produced in the PDL. It is known that this sterile necrosis interferes with bone resorption; thus, at the 1-week interval, no tooth movement was experienced on either side. At 2 weeks, hyaline was still present on the control side, so no bone resorption of the tooth socket wall had yet occurred; however, root resorption was evident. At 2 weeks, on the experimental (corticotomy) side, the hyaline had already been removed from the PDL. Presumably, macrophages (the presence of which had been stimulated by the regional acceleratory phenomenon) were in place very early to perform this removal and thus, in the process, no root resorption occurred. At 4 and 8 weeks after the initiation of treatment on the control (non-corticotomy) side, the hyaline had finally been removed and the alveolar bone resorption had begun, thereby allowing mesial bicuspid tooth movement to proceed. During this waiting period, extensive root resorption had occurred on the control side. Interestingly, at the 4- and 8-week post-treatment intervals on the experimental (corticotomy) side, there was no hyaline present in the periodontium, the socket wall had appropriately resorbed, and the bicuspid tooth was able to move mesially without root resorption.

In the rat model, **Sebaoun and colleagues** studied the alveolar response to corticotomy as a function of time and proximity to the surgical injury.[197] Maxillary buccal and palatal cortical plates were injured (corticotomy) in healthy adult rats (n = 36) adjacent to the upper left first molars. Twenty-four of the animals were euthanized at 3, 7, and 11 weeks after injury.

Histomorphometric analysis was performed to study alveolar spongiosa and periodontal ligament dynamics. Both catabolic activity and anabolic actions were also measured. A separate group of animals (n = 12) were fed with calcein fluorescent stain. After the same surgical and orthodontic maneuvers, they were processed for non-decalcified fluorescent stain histology. The results of the study demonstrated that, at 3 weeks, the surgical (corticotomy) group had significantly less calcified spongiosa bone surface, greater periodontal ligament surface, higher osteoclast number, and greater lamina dura apposition width. The catabolic activity (i.e., the osteoclast count) and the anabolic activity (i.e., the apposition rate) increased by threefold, the calcified spongiosa bone surface decreased by twofold, and the PDL surface increased by twofold in the corticotomy group. Interestingly, the surgical injury to the alveolus (corticotomy) that induced a significant increase in tissue turnover by week 3 dissipated to a steady state by postoperative week 11. In addition, the impact of the injury (rapid tooth movement) was localized to the area immediately adjacent to the decortication injury. The authors concluded that selective alveolar decortication induced increased turnover of alveolar spongiosa and that the activity was localized. The escalation of demineralization–mineralization dynamics is the likely biologic mechanism that explains the rapid tooth movement that occurs after selective alveolar decortication.

In review, it appears that the completion of a corticotomy of the alveolar process immediately brings macrophages into the field of bony injury.[116] The macrophages are then able to remove the hyaline (necrotic material) from the periodontium more quickly than occurs in a non-corticotomy setting. The early presence of macrophages can explain the rapid degree of bone resorption and minimal root resorption during orthodontic tooth movement that was documented by Lino and colleagues. When Lino's group compared the amount of tooth movement in their experimental and control groups, the teeth on the corticotomy side moved twice as fast as the teeth on the control side. The enhanced tooth movement after corticotomy should continue as long as the surgical site healing remains in progress (only approximately 2 to 3 months). The potential for morbidity to the teeth and periodontium that is associated with the surgical procedure, must be considered in light of any advantage of reduced time to reach treatment objectives.

Temporary Skeletal Anchorage in Orthodontics

TADs that are made of titanium and temporarily fixed into alveolar bone have been shown to provide effective supplemental or "maximum" anchorage for the movement of teeth while avoiding unwanted negative reciprocal responses in the dentition.* Retromolar titanium (dental) implants simply for the purpose of temporary skeletal anchorage to facilitate orthodontic movement of the teeth were first placed by Roberts and colleagues in 1989.[185-187] Wehrbein and Merz placed titanium implants directly into the hard palate and then tied the molar teeth to them.[224,225] The purpose of the temporary hard-palate implants was to prevent the movement of the molar teeth that were to be used as direct anchorage for the distal movement of anterior teeth into bicuspid extraction sites. Standard titanium bone fixation plates (i.e., mini-plates) were next used as TADs. The mini-plates were fixed to the zygomatic buttress with titanium screws. The end of the titanium plate then perforated through the oral mucosa to provide a point of attachment for orthodontic wires, springs, coils, and elastics.

Mini-plates and mini-screws made of titanium alloy are becoming a frequent temporary source of skeletal anchorage (i.e., TADs) for orthodontic tooth movement. Advantages of these TADs include being relatively simple to place, providing proximity for orthodontic attachments, allowing for immediate loading, requiring no laboratory work, not being socially embarrassing for the patient to use, and being relatively low in cost. Alternatively, TAD use may be complicated by early "loosening" or injury to the teeth and the periodontium. Depending on their location of placement, TADs may also provide vector challenges for accomplishing the task at hand. Although the clinical role of TADs in everyday orthodontic practice has not yet been fully clarified, there are potential applications for a wide spectrum of purposes, including the following:

- The retraction and realignment of the anterior teeth without the need for posterior support (anchorage)
- The closing of edentulous spaces after extraction
- The correction of the midline in patients with missing posterior teeth
- The reestablishment of proper molar positions (i.e., uprighting tipped teeth) for use as abutments
- The intrusion and extrusion of teeth (e.g., to intrude maxillary molars in an attempt to close anterior open bites)
- The protraction and retraction of teeth
- The stabilization of teeth that have reduced alveolar bone support
- The application of orthopedic traction to the maxilla or mandible in the growing individual in an attempts to alter jaw growth

*References 7, 21, 27, 33, 52, 53, 57, 62, 77, 80, 104, 106, 109, 123, 129, 131, 142, 159-161, 188, 189, 199, 205, 211, 215

Conclusions

Current problems in clinical orthodontics are no longer a matter of which attachments or mechanical approaches to use. Of greater importance is the basic treatment philosophy related to combined clinical efforts for the achievement of long-term dental health, enhanced facial aesthetics, and an improved airway. The occlusion observed in each individual is greatly influenced by baseline jaw relationships as well as by daily masticatory and airway function. For these reasons, the physiologic orthodontic movement of teeth has limits and should remain within a biologic envelope. With treatment now being rendered to children, adolescents, and adults of all ages, a coordinated comprehensive interdisciplinary approach to care is more important than ever.

References

1. Ackerman H, Proffit WR: The characteristics of malocclusion: a modern approach to classification and diagnosis. *Am J Orthod* 56:443–454, 1969.
2. Ackerman JL, Proffit WR: Communication in orthodontic treatment planning: bioethical and informed consent issues. *Angle Orthod* 65:253, 1995.
3. Ackerman JL, Proffit WR: Soft tissue limitations in orthodontic treatment. *Angle Orthod* 67:327–336, 1997.
4. Ackerman M: Evidence-based orthodontics for the 21st century. *J Am Dent Assoc* 135:162–167, 2004.
5. Adachi H, Igarashi K, Mitani H, Shinoda H: Effects of topical administration of a bisphosphonate (risedronate) on orthodontic tooth movements in rats. *J Dent Res* 73:1478–1484, 1994.
6. Adamides IP, Spyropoulos MN: The effects of lymphadenoid hypertrophy on the position of the tongue, the mandible and the hyoid bone. *Eur J Orthod* 5:287–294, 1983.
7. Adell R, Hansson BO, Brånemark PI, Breine U: Intra-osseous anchorage of dental prostheses. II. Review of clinical approaches. *Scand J Plast Reconstr Surg* 4:19–34, 1970.
8. Al Yami EA, Kuijpers-Jagtman AM, Van't Hof MA: Stability of orthodontic treatment outcome: Follow-up until 10 years post-retention. *Am J Orthod Dentofacial Orthop* 115:300–304, 1999.
9. Albino JE, Lewis EA, Slakter MJ: Examiner reliability for two methods of assessing malocclusion. *Angle Orthod* 48:297–302, 1978.
10. Al-Munajed MK, Gordon PH, McCabe JF: The use of a cyanoacrylate adhesive for bonding orthodontic brackets: an ex-vivo study. *J Orthod* 27:255–260, 2000.
11. Andrews LF: The six keys to normal occlusion. *Am J Orthod* 63:296–309, 1972.
12. Andrews LF: The straight-wire appliance, origin, controversy, commentary. *J Clin Orthod* 10:99–114, 1976.
13. Andrews LF: The straight-wire appliance: extraction brackets and classification of treatment. *J Clin Orthod* 10:360–379, 1976.
14. Andrews LF: *Straight wire: the concept and appliance,* San Diego, LA Wells Co, 2003.
15. Angle EH: *Treatment of malocclusion of the teeth*, ed 7, Philadelphia, 1907, SS White.
16. Angle EH: The latest and best in orthodontic mechanism. *Dent Cosmos* 70:1143–1158, 1928.

17. Anholm JM, Crites DA, Hoff R, Rathbun WE: Corticotomy-facilitated orthodontics. *CDA J* 14:7–11, 1986.
18. Aronowitz HI: Orthodontics and occlusion: a historical perspective. *J Calif Dent Assoc* 24:16–18, 1996.
19. Articolo LC, Kusy RP: Influence of angulation on the resistance to sliding in fixed appliances. *Am J Orthod Dentofacial Orthop* 115:39–51, 1999.
20. Asbell MB: A brief history of orthodontics. *Am J Orthod Dentofacial Orthop* 98:176–183, 1990.
21. Bae SM, Park HS, Kyung HM, et al: Clinical application of micro-implant anchorage. *J Clin Orthod* 36:298–302, 2002.
22. Baker CR: Orthodontic profiles. *Am J Orthod* 43:210–218, 1957.
23. Bartzela T, Turp J, Motschall E, Maltha J: Medication effect on the rate of orthodontic tooth movement: a systematic literature. *Am J Orthod Dentofacial Orthop* 135:16–26, 2009.
24. Baumrind S, Korn EL, Boyd RL, Maxwell R: The decision to extract: Part 1–Interclinican agreement. *Am J Orthod Dentofacial Orthop* 109:297–309, 1996.
25. Begg PR, Kesling PC: The differential force method of orthodontic treatment. *Am J Orthod* 71:1–39, 1977.
26. Bibb CA: Occlusal evaluation and therapy in the management of periodontal disease. In Newman MG, Takei HH, Carranza FA, editors: *Carranza's clinical periodontology*, ed 9, Philadelphia, 2002, W.B. Saunders Company, pp 698–701.
27. Block MS, Hoffman DR: A new device for absolute anchorage for orthodontics. *Am J Orthod Dentofacial Orthop* 107:251–258, 1995.
28. Bogoch E, Gschwend N, Rahn B, et al: Healing of cancellous bone osteotomy in rabbits–Part I: Regulation of bone volume and the regional acceleratory phenomenon in normal bone. *J Orthop Res* 11:285–291, 1993.
29. Bogoch E, Gschwend N, Rahn B, et al: Healing of cancellous bone osteotomy in rabbits–Part II: Local reversal of arthritis-induced osteopenia after osteotomy. *J Orthop Res* 11:292–298, 1993.
30. Bohl MV, Maltha JC, Von den Hoff JW, Kuijpers-Jagtman AM: Focal hyalinization during experimental tooth movement in beagle dogs. *Am J Orthod Dentofacial Orthop* 125:615–623, 2004.
31. Bolton WA: Disharmony in tooth size and its relation to the analysis and treatment of malocclusion. *Angle Orthod* 28:113–130, 1958.
32. Bowman J: One-stage versus two-stage treatment: are two really necessary? *Am J Orthod Dentofacial Orthop* 113:111–116, 1998.
33. Brånemark PI, Adell R, Breine U, et al: Intra-osseous anchorage of dental prostheses. I. Experimental studies. *Scand J Plast Reconstr Surg* 3:81–100, 1969.
34. Brezniak N, Wasserstein A: Root resorption after orthodontic treatment: Part 2. Literature review. *Am J Orthod Dentofac Orthop* 103:138–146, 1993.
35. Brodie AG: Orthodontic history and what it teaches. *Angle Orthod* 4:85–97, 1934.
36. Brodie AG: Orthodontic concepts prior to the death of Edward H Angle. *Angle Orthod* 26:144–154, 1956.
37. Brudvik P, Rygh P: The initial phase of orthodontic root resorption incident to local compression of periodontal ligament. *Eur J Orthod* 15:249–263, 1993.
38. Buonocore MG: A simple method of increasing the adhesion of acrylic filling materials to enamel surfaces. *J Dent Res* 34:849–853, 1955.
39. Burrow JS: Friction and resistance to sliding in orthodontics: a critical review. *Am J Orthod Dentofacial Orthop* 135:442–447, 2009.
40. Cain KK, Rugh JD, Hatch JP, et al: Readiness for orthognathic surgery: a survey of practitioner opinion. *Int J Adult Orthodon Orthognath Surg* 17:7–11, 2002.
41. Carey CW: Lower arch dimension and tooth size. *Am J Orthod* 35:762–775, 1949.
42. Carranza FA: Bone loss and patterns of bone destructions. In Newman MG, Takei HH, Carranza FA, editors: *Carranza's clinical periodontology*, ed 9, Philadelphia, 2002, W.B. Saunders Company, pp 362.
43. Case CS: Principles of occlusion and dentofacial relations. *Dent Items Int* 27:489, 1905.
44. Case CS: *A practical treatise on the techniques and principles of dental orthopedia and prosthetic correction of cleft palate*, ed 2, Chicago, 1922, CS Case.
45. Casto FM: The trend of orthodontic treatment. *Int J Orthod Oral Surg Rad* 16:1078–1092, 1930.
46. Casto FM: A historical sketch of orthodontia. *Dent Cosmos* 76:111–135, 1934.
47. Cetlin NM, Ten Hoeve AJ: Nonextraction treatment. *J Clin Orthod* 17:396–413, 1983.
48. Chapman H: Orthodontics: fifty years in retrospect. *Am J Orthod* 41:421–442, 1955.
49. Chung KR, Oh MY, Ko SJ: Corticotomy-assisted orthodontics. *J Clin Orthod* 35:331–339, 2001.
50. Cleall JF: Deglutition: a study of form and function. *Am J Orthod* 51:161–182, 1965.
51. Cooper RB, Gross M, Hamula W: Direct bonding with light cured adhesive precoated brackets. *J Clin Orthod* 26:477–479, 1992.
52. Costa A, Raffaini M, Melsen B: Microscrews as orthodontic anchorage: a preliminary report. *Int J Adult Orthodon Orthognath Surg* 13:201–209, 1998.
53. Creekmore TD, Ecklund MK: The possibilities of skeletal anchorage. *J Clin Orthod* 17:266–269, 1983.
54. Creekmore TD, Kunik RL: Straight wire: the next generation. *Am J Orthod Dentofacial Orthop* 104:8–20, 1993.
55. Cryer M: Typical and atypical occlusion of teeth. *Dent Cosmos* 46:713, 1904.
56. Cueto HI: A little bit of history: the first direct bonding in orthodontia. *Am J Orthod Dentofacial Orthop* 98:276–277, 1990.
57. Deguchi T, Takano-Yamamoto T, Kanomi R, et al: The use of small titanium screws for orthodontic anchorage. *J Dent Res* 82:377–381, 2003.
58. Delaire J: Considerations on facial growth (particularly of the maxilla): therapeutic deductions. *Rev Stomatol Chir Maxillofac* 72:57–76, 1971.
59. Delaire J: La croissance maxillaire: deductions therapeutiques. *Trans Eur Orthod Soc* 81–102, 1971.
60. Dewel BF: Second premolar extraction in orthodontics: principles, procedures, and case analysis. *Am J Orthod* 441:107–120, 1955.
61. Dewel BF: The Case-Dewey-Cryer extraction debate. *Am J Orthod* 50:862–865, 1964.
62. Dewel BF: Editorial: Orthodontosie, orthodontics, or dentofacial orthopedics. *Am J Orthod* 70:328, 1976.
63. Dewel BF: The ribbon arch: its influence in the development of orthodontic appliances. *Angle Orthod* 51:263–268, 1981.
64. DiBiase AT, Nasr IH, Scott P, Cobourne MT: Duration of treatment and occlusal outcome using Damon3 self-ligated and conventional orthodontic bracket systems in extraction patients: a prospective randomized clinical trial. *Am J Orthod Dentofacial Orthop* 139:e111–e116, 2011.
65. Dowling P, Espeland L, Krogstad O, et al: Duration of orthodontic treatment involving orthognathic surgery. *Int J Adult Orthodon Orthognath Surg* 14:146–152, 1999.
66. Driel WD, Leeuwen EJ, Hoff JW, et al: Time-dependent mechanical behavior of the periodontal ligament. *Proc Inst Mech Eng H* 214:497–504, 2000.
67. Drobocky OB, Smith RJ: Changes in facial profile during orthodontic treatment with extraction of four first premolars. *Am J Orthod Dentofacial Orthop* 95(5):220–230, 1989.
68. Düker J: Experimental animal research into segmental alveolar movement after corticotomy. *J Maxillofac Surg* 3:81–84, 1975.
69. Eby JD: Clinical approach to orthodontic therapy. *Am J Orthod* 35:407–424, 1949.
70. Edler RJ: Problems in the orthodontic management of orthognathic cases. *Eur J Orthod* 12:420–437, 1990.
71. Engström C, Granström G, Thilander B: Effect of orthodontic force on periodontal tissue metabolism: a histologic and biochemical study in normal and hypocalcemic young rats. *Am J Orthod Dentofacial Orthop* 93:486–495, 1988.
72. Fastlicht S: Evolution of the universal appliance. *Am J Orthod* 36:433–444, 1950.

73. Ferguson J: Comment on two-phase treatment. *Am J Orthod Dentofacial Orthop* 110:14–15, 1996.
74. Fink DF, Smith RJ: The duration of orthodontic treatment. *Am J Orthod Dentofacial Orthop* 102:45–51, 1992.
75. Freng A, Kvam E: Facial sagittal growth following partial basal resection of the nasal septum: a retrospective study in man. *Eur J Orthod* 1:89–96, 1979.
76. Freng A: Dentofacial development in the long-lasting nasal stenosis. *Scand J Dent Res* 87:260–267, 1979.
77. Freudenthaler JW, Bantleon H-P, Haas R: Bicortical titanium screws for critical orthodontic anchorage in the mandible: a preliminary report on clinical applications. *Clin Oral Implants Res* 12:358–363, 2001.
78. Frost HM: The biology of fracture healing: an overview for clinicians. Part I. *Clin Orthop Related Res* 248:283–293, 1989.
79. Frost HM: The biology of fracture healing: an overview for clinicians. Part II. *Clin Orthop Relat Res* 248:294–309, 1989.
80. Gainsforth BL, Higley LB: A study of orthodontic anchorage possibilities in basal bone. *Am J Orthod Oral Surg* 31:406–417, 1945.
81. Gantes B, Rathbun E, Anholm M: Effects on the periodontium following corticotomy-facilitated orthodontics: case reports. *J Periodontol* 61:234–238, 1990.
82. Generson RM, Porter JM, Zell A, Stratigos GT: Combined surgical and orthodontic management of anterior open bite using corticotomy. *J Oral Surg* 36:216–219, 1978.
83. Gianelly A: One-phase versus two-phase treatment. *Am J Orthod Dentofacial Orthop* 108:556–559, 1995.
84. Goldie RS, King GJ: Root resorption and tooth movement in orthodontically treated, calcium-deficient, and lactating rats. *Am J Orthod* 85:424–430, 1984.
85. Goldstein A: The clinical testing of orthodontic results. *Am J Orthod* 51:723–755, 1965.
86. Gottlieb EL: Orthodontics in the year 2000. *J Clin Orthod* 34:9–10, 2000.
87. Graber TM, Vanarsdall RL: *Orthodontics: current principles and techniques*. St. Louis, 2000, Mosby.
88. Graf H, Grassl J, Aeberhard H: A method for measurement of occlusal force in three directions. *Helv Odontol Acta* 18:7–11, 1974.
89. Guilford SH: *Orthodontia: malposition of human teeth*. Philadelphia, 1889, Spangler & Davis.
90. Hajji SS: *The influence of accelerated osteogenic response on mandibular decrowding (thesis)*. St. Louis, 2000, St. Louis University.
91. Hannuksela A: The effect of moderate and severe atopy on the facial skeleton. *Eur J Orthod* 3:187–193, 1981.
92. Harvold EP: The role of function in the etiology and treatment of malocclusion. *Am J Orthod* 54:883–898, 1968.
93. Harvold EP, Chierici G, Vargervik K: Experiments on the development of dental malocclusions. *Am J Orthod* 61:38–44, 1972.
94. Harvold EP, Vargervik K, Chierici G: Primate experiments on oral sensation and dental malocclusion. *Am J Orthod Dentofacial Orthop* 63:494–508, 1973.
95. Harvold EP: Centric relation: a study of pressure and tension systems in bone modeling and mandibular positioning. *Dent Clin North Am* 19:473–484, 1975.
96. Harvold EP, Chierici G, Vargervik K: Primate experiments on oral sensation and dental malocclusions. *Am J Orthod* 79:359–372, 1981.
97. Harvold EP, Chierici G, Vargervik K, editors: *Treatment of hemifacial microsomia*, New York, 1983, Alan R. Liss.
98. Hernández-Alfaro F, Guijarro-Martínez R, Molina-Coral A, Badía-Escriche C: "Surgery first" in bimaxillary orthognathic surgery. *J Oral Maxillofac Surg* 69:e201–e207, 2011.
99. Holdaway RA: Bracket angulation as applied to the edgewise appliance. *Angle Orthod* 22:227–236, 1952.
100. Hsieh TJ, Pinskaya Y, Roberts WE: Assessment of orthodontic treatment outcomes: early treatment versus late treatment. *Angle Orthod* 75:162–170, 2005.
101. Hwang HS, Lee KH: Intrusion of overerupted molars by corticotomy and magnets. *Am J Orthod Dentofacial Orthop* 120:209–215, 2001.
102. Igarashi K, Mitani H, Adachi H, Shinoda H: Anchorage and retentive effects of a bisphosphate (AHBuBP) on tooth movements in rats. *Am J Orthod Dentofacial Orthop* 106:279–289, 1994.
103. Igarashi K, Adachi H, Mitani H, Shinoda H: Inhibitory effect of the topical administration on root resorption incident to orthodontic tooth movement in rats. *J Dent Res* 75:1644–1649, 1996.
104. Ismail SFH, Johal AS: The role of implants in orthodontics. *J Orthod* 29:239–245, 2002.
105. Itoh T, Matsuo N, Fukushima T, et al: Effect of contamination and etching on enamel bond strength of a new light-cured glass ionomer cement. *Angle Orthod* 69:450–456, 1999.
106. Janssens F, Swennen G, Dujardin T, et al: Use of an onplant as orthodontic anchorage. *Am J Orthod Dentofacial Orthop* 122:566–570, 2002.
107. Jarabak JR, Fizzell JA: *Technique and treatment with the light-wire appliances: light, differential forces in orthodontics*. St. Louis, 1963, Mosby.
108. Johnson JE: A new orthodontic mechanism: the twinwire automatic appliance. *J Am Dent Assoc* 19:997–1011, 1932.
109. Kanomi R: Mini-implant for orthodontic anchorage. *J Clin Orthod* 31:763–767, 1997.
110. Kapila S, Conley RS, Harrell WE, Jr: The current status of cone beam computed tomography imaging in orthodontics. *Dentomaxillofac Radiol* 40:24–34, 2011.
111. Kesling PC: Dynamics of the tip-edge bracket. *Am J Orthod Dentofacial Orthop* 96:16–25, 1989.
112. Kesling PC: The edgewise slot: Angle's orthodontic albatross. *World J Orthod* 1:142–151, 2000.
113. Kim TW, Yoshida Y, Yokoya K, Sasaki T: An ultrastructural study of the effects of bisphosphonate administration on osteoclastic bone resorption during relapse of experimentally moved rat molars. *Am J Orthod Dentofacial Orthop* 115:645–653, 1999.
114. Kingsley NW: *A treatise on oral deformities (as a branch of mechanical surgery)*. New York, 1880, Appleton & Company.
115. Kloehn SJ: Mixed dentition treatment. *Angle Orthod* 20:75–96, 1950.
116. Kole H: Surgical operations on the alveolar ridge to correct occlusal abnormalities. *Oral Surg Oral Med Oral Pathol* 12:515–529, 1959.
117. Korkhaus G: *Handbuch de Zahnheilkunde*. Munchen, 1939, J.F. Bergmann.
118. Kraus BS, Jordan RE, Abrams L: *Dental anatomy and occlusion*. Baltimore, 1969, Williams & Wilkins Company.
119. Kreiborg S, Jensen BL, Møller E, Björk A: Craniofacial growth in a case of congenital muscular dystrophy. *Am J Orthod* 74:207–215, 1978.
120. Krishnan PH, Gupta DS, Sharma VP: Application of cyanoacrylate in orthodontics. *J Polymer Materials* 11:299–300, 1994.
121. Kurol J, Owman-Moll P: Hyalinization and root resorption during early orthodontic tooth movement in adolescents. *Angle Orthod* 68:161–165, 1998
122. Kusy RP, Whitley JQ: Influence of archwire and bracket dimensions on sliding mechanics: derivations and determinations of the critical contact angles for binding. *Eur J Orthod* 21:199–208, 1999
123. Lee JS, Park HS, Kyung HM: Micro-implant anchorage for lingual treatment of a skeletal class II malocclusion. *J Clin Orthod* 35:643–647, 2001.
124. Lee RT: The benefits of post-surgical orthodontic treatment. *Br J Orthod* 21:265–274, 1994.
125. Leeuwen EJ, Maltha JC, Kuijpers-Jagtman AM: Tooth movement with light continuous and discontinuous forces in beagle dogs. *Eur J Oral Sci* 107:468–474, 1999.
126. Lewis PD: Space closure in extraction cases. *Am J Orthod* 36:172–191, 1950.
127. Linder-Aronson S: Adenoids. Their effect on mode of breathing and nasal airflow and their relationship to characteristics of the facial skeleton and the dentition. A biometric, rhino-manometric and cephalometro-radiography study on children with and without adenoids. *Acta Otolaryngol Suppl* 265:1–132, 1970.
128. Lindskog S, Lilja E: Scanning electron microscopic study of orthodontically induced injuries to the periodontal membrane. *Scand J Dent Res* 92:334–343, 1984.
129. Linkow LI: The endosseous blade implant and its use in orthodontics. *Int J Orthod* 18:149–154, 1969.

130. Lino S, Sakoda S, Ito G, et al: Acceleration of orthodontic tooth movement by alveolar corticotomy in the dog. *Am J Orthod Dentofacial Orthop* 131:448.e1–448.e8, 2007.
131. Liou EJW, Pai BCJ, Lin JCY: Do miniscrews remain stationary under orthodontic forces? *Am J Orthod Dentofacial Orthop* 126:42–47, 2004.
132. Livieratos FA, Johnston LE, Jr: A comparison of one-stage and two-stage nonextraction alternatives in matched Class II samples. *Am J Orthod Dentofacial Orthop* 108:118–131, 1995.
133. Logan LR: Second premolar extraction in Class I and Class II. *Am J Orthod* 63:115–147, 1973.
134. Lundström A: The importance of genetic and non-genetic factors in the facial skeleton studied in 100 pairs of twins. In *The Hague: European Orthodontic Society*, 1954, pp 92–102.
135. Lundström A, Woodside DG: Individual variation in growth directions expressed at the chin and the midface. *Eur J Orthod* 2:65–79, 1980.
136. Lundström A: Nature versus nurture in dento-facial variation. *Eur J Orthod* 6:77–91, 1984.
137. Luther F, Morris DO, Hart C: Orthodontic preparation for orthognathic surgery: how long does it take and why? A retrospective study. *Br J Oral Maxillofac Surg* 41:401–406, 2003.
138. Matasa CG, Graber TM: Angle, the innovator, mechanical genius, and clinician. *Am J Orthod Dentofacial Orthop* 117:444–452, 2000.
139. McLaughlin RP, Bennett JC: Anchorage leveling and aligning with a preadjusted appliance system. *J Clin Orthod* 25:687–696, 1991.
140. McManaman JE, Woodside DG: Evolution of orthodontic bracket design. *Alpha Omegan* 93:55–65, 2000.
141. Meikle MC: Remodeling the dentofacial skeleton: the biological basis of orthodontics and dentofacial orthopedics. *J Dent Res* 86:12–24, 2007.
142. Melsen B, Petersen JK, Costa A: Zygoma ligatures: an alternative form of maxillary anchorage. *J Clin Orthod* 32:154–158, 1998.
143. Midgett RJ, Shaye R, Fruge JF, Jr: The effect of altered bone metabolism on orthodontic tooth movement. *Am J Orthod* 80:256–262, 1981.
144. Mitchell DL: The first direct bonding in orthodontia, revisited [viewpoint]. *Am J Orthod Dentofacial Orthop* 101:187–189, 1992.
145. Miura F, Nakagawa K, Masuhara E: New direct bonding system for plastic brackets. *Am J Orthod* 59:350–361, 1971.
146. Monson GS: Occlusion as applied to crown and bridge work. *J Am Dent Assoc* 7:399–413, 1920.
147. Moyers R: *Handbook of orthodontics*, ed 4, Chicago, 1988, Year Book.
148. Nance HN: The limitations of orthodontic treatment. *Am J Orthod* 33:177–223, 1947.
149. Nance HN: The removal of second premolars in orthodontic treatment. *Am J Orthod* 35:685–695, 1949.
150. Nanda R: Protraction of maxilla in rhesus monkeys by controlled extraoral forces. *Am J Orthod* 74:121–141, 1978.
151. Nanda R: Biomechanical and clinical considerations of a modified protraction headgear. *Am J Orthod* 78:125–139, 1980.
152. Nanda R, Goldin B: Biomechanical approaches to the study of alterations of facial morphology. *Am J Orthod* 78:213–226, 1980.
153. Newman GV: First direct bonding in orthodontics. *Am J Orthod Dentofacial Orthop* 101:190–191, 1992.
154. Newman GV, Newman RA, Sun BI, et al: Adhesion promoters, their effect on the bond strength of metal brackets. *Am J Orthod Dentofacial Orthop* 108:237–241, 1995.
155. Noxon SJ, King GJ, Gu G, Huang G: Osteoclast clearance from periodontal tissues during orthodontic tooth movement. *Am J Orthod Dentofacial Orthop* 120:466–476, 2001.
156. Nyman S, Karring T, Bergenholtz G: Bone regeneration in alveolar bone dehiscences produced by jiggling forces. *J Periodontal Res* 17:316–322, 1982.
157. Otuyemi OD, Jones SP: Long-term evaluation of treated Class II division 1 malocclusions utilizing the PAR index. *Br J Orthod* 22:171–178, 1995.
158. Paladin P, editor: *Orthodontic history: braced for a century*, St. Louis, 2005, American Association of Orthodontics.
159. Park HS: The skeletal cortical anchorage using titanium microscrew implants. *Korea J Orthod* 29:699–706, 1999.
160. Park HS, Bae SM, Kyung HM, Sung JH: Micro-implant anchorage for treatment of skeletal Class I bialveolar protrusion. *J Clin Orthod* 35:417–422, 2001.
161. Park HS, Kyung HM, Sung JH: Simple method of molar uprighting with micro-implant anchorage. *J Clin Orthod* 36:592–596, 2002.
162. Parker WS: Review of the D'Amico concept of cuspid function and its relationship to orthodontic treatment. *Bull Pac Coast Soc Orthod* 42:11–14, 1967.
163. Pascal M, Gallucci G, Belser U: Anatomic crown/width ratios of unworn and worn maxillary teeth in white subjects. *J Prosthet Dent* 89:453–461, 2003.
164. Peck H, Peck S: An index for assessing tooth-shape deviations as applied to the mandibular incisors. *Am J Orthod* 61:384–401, 1972.
165. Peterson HW: Some observations on the progress of orthodontics during the past fifty years. *Am J Orthod* 39:289–290, 1953.
166. Pilion JJGM, Kuijpers-Jagtman AM, Maltha JC: Magnitude of orthodontic forces and rate of bodily tooth movement: an experimental study in beagle dogs. *Am J Orthod Dentofacial Orthop* 110:16–23, 1996.
167. Proffit WR, Gamble JW, Christiansen RL: Generalized muscular weakness with severe anterior open bite. *Am J Orthod* 54:104–110, 1968.
168. Proffit WR: Forty-year review of extraction frequencies at a university orthodontic clinic. *Angle Orthod* 64:407–414, 1994.
169. Proffit WR, Miguel JA: The duration and sequencing of surgical orthodontic treatment. *Int J Adult Orthod Orthognath Surg* 10:35–42, 1995.
170. Proffit WR, White RP, Sarver DM: *Contemporary treatment of dentofacial deformity*. St. Louis, 2003, Mosby.
171. Proffit WR, Fields HW, Sarver DM, editors: *Contemporary orthodontics*, ed 5, St. Louis, 2012, Mosby/Elsevier.
172. Pus MD, Way DC: Enamel loss due to orthodontic bonding with filled and unfilled resins using various clean-up techniques. *Am J Orthod* 77:269–283, 1980.
173. Redd TB, Shivapuja PK: Debonding ceramic brackets: effects on enamel. *J Clin Orthod* 25:475–481, 1991.
174. Reitan K: Continuous bodily tooth movement and its histological significance. *Acta Odontol Scand* 6:115–144, 1947.
175. Ren Y, Maltha JC, Bohl MV, et al: Cytokine levels in crevicular fluid are less responsive to orthodontic force in adults than in juveniles. *J Clin Periodontol* 29:757–762, 2002.
176. Ren Y, Maltha JC, Von den Hoff JW, Kuijpers-Jagtman AM: Age effect on orthodontic tooth movement in rats. *J Dent Res* 82:38–42, 2003.
177. Retief DH, Dreyer CJ, Gavron G: The direct bonding of orthodontic attachments to teeth by means of an epoxy resin adhesive. *Am J Orthod* 58:21–40, 1970.
178. Richmond S, Shaw WC, Roberts CT, Andrews M: The PAR index (Peer Assessment Rating): methods to determine outcome of orthodontic treatment in terms of improvement and standards. *Eur J Orthod* 14:180–187, 1992.
179. Ricketts RM: Keystone triad: Part 2. *Am J Orthod* 50:728–750, 1964.
180. Ricketts RM: A detailed consideration of the line of occlusion. *Angle Orthod* 48:274–282, 1978.
181. Ricketts RM: The wisdom of the bioprogressive philosophy. *Semin Orthod* 4:201–209, 1998.
182. Rinchuse DJ, Kandasamy S: Articulators in orthodontics: an evidence-based perspective. *Am J Orthod Dentofacial Orthop* 129:299–308, 2006.
183. Rinchuse DJ, Kandasamy S: Centric relation: a historical and contemporary orthodontic perspective. *J Am Dent Assoc* 137:494–501, 2006.
184. Roberts WE, Goodwin WC, Jr, Heiner SR: Cellular response to orthodontic force. *Dent Clin North Am* 25:3–17, 1981.
185. Roberts WE, Smith RK, Zilberman Y, et al: Osseous adaptation to continuous loading of

rigid endosseous implants. *Am J Orthod* 86:95–111, 1984.
186. Roberts WE, Helm FR, Marshal KJ, Gongloff RK: Rigid endosseous implants for orthodontic and orthopaedic anchorage. *Angle Orthod* 59:247–256, 1989.
187. Roberts WE, Marshall KJ, Mozsary PG: Rigid endosseous implant utilized as anchorage to protract molars and close an atrophic extraction site. *Angle Orthod* 60:135–151, 1990.
188. Roberts WE, Arbuckle GR, Analoui M: Rate of mesial translation of mandibular molars using implant anchored mechanics. *Angle Orthod* 66:331–338, 1996.
189. Roberts WE, Huja S, Roberts JA: Bone modeling: biomechanics, molecular mechanisms, and clinical perspectives. *Semin Orthod* 10:123–161, 2004.
190. Roth RH: Five-year clinical evaluation of the Andrews straightwire appliance. *J Clin Orthod* 10:836–850, 1976.
191. Roth RH: The straight-wire appliance 17 years later. *J Clin Orthod* 21:632–642, 1987.
192. Roth RH: Gnathological considerations for orthodontic therapy. In McNeill C, editor: *Science and practice of occlusion*, Chicago, 1997, Quintessence, pp 502–514.
193. Sabri R: Orthodontic objectives in orthognathic surgery: state of the art today. *World J Orthod* 7:177–191, 2006.
194. Sato S, Kaneko M, Sasaguri K, et al: Different mechanics for orthodontic correction of Class II openbite and Class III openbite malocclusions. *Bulletin of Kanagawa Dental College* 35(1):65–77, 2007.
195. Schoppe RJ: An analysis of second premolar extraction procedures. *Angle Orthod* 34:292–302, 1964.
196. Schubert P, Bailey LJ, White RP, Proffit WR: Long-term cephalometric changes in untreated adults compared to those treated with orthognathic surgery. *Int J Adult Orthodon Orthognath Surg* 14:91–99, 1999.
197. Sebaoun JD, Kantarci A, Turner JW, et al: Modeling of trabecular bone and lamina dura following selective alveolar decortication in rats. *J Periodontol* 79(9):1679–1688, 2008.
198. Shankland WM: *The American Association of Orthodontists: the biography of a specialty organization*. St. Louis, 1971, The American Association of Orthodontists.
199. Shapiro PA, Kokich VG: Uses of implants in orthodontics. *Dent Clin North Am* 32:539–540, 1988.
200. Shih MS, Norrdin RW: Regional acceleration of remodeling during healing of bone defects in beagles of various ages. *Bone* 5:377–379, 1985.
201. Siara-Olds NJ, Pangrazio-Kulbersh V, Berger J, Bayirli B: Long-term dentoskeletal changes with the Bionator, Herbst, Twin Block, and MARA functional appliances. *Angle Orthod* 80:18–29, 2010.
202. Silverman E, Cohen M, Gianelly AA, Dietz VS: A universal direct bonding system for both metal and plastic brackets. *Am J Orthod* 62:236–244, 1972.
203. Siomka LV, Powers JM: In vitro bond strength of treated direct-bonding metal bases. *Am J Orthod* 88:133–136, 1985.
204. Sirirungrojying S, Saito K, Hayakawa T, Kasai K: Efficacy of using self-etching primer with a 4-META/MMA-TBB resin cement in bonding orthodontic brackets to human enamel and effect of saliva contamination on shear bond strength. *Angle Orthod* 4:251–258, 2004.
205. Southard TE, Buckley MJ, Spivey JD, et al: Intrusion anchorage potential of teeth versus rigid endosseous implants: a clinical and radiographic evaluation. *Am J Orthod Dentofacial Orthop* 107:115–120, 1995.
206. Squire D, Best AM, Lindauer SJ, Laskin DM: Determining the limits of orthodontic treatment of overbite, overjet, and transverse discrepancy: a pilot study. *Am J Orthod Dentofacial Orthop* 129:804–808, 2006.
207. Strang RH: Highlights of sixty-four years in orthodontics. *Angle Orthod* 44:101–112, 1974.
208. Swartz LS: Fact or fiction: the clinical relevance of in vitro steady state friction studies. *J Clin Orthod* 8:427–432, 2007.
209. Thompson JR, Craddock FW: Functional analysis of occlusion. *J Am Dent Assoc* 33:404–406, 1949.
210. Tulloch C, Phillips C, Proffit W: Benefit of early Class II treatment: progress report of a two-phase randomized clinical trial. *Am J Orthod Dentofacial Orthop* 113:62–71, 1998.
211. Turley PK, Kean C, Schur J, et al: Orthodontic force application to titanium endosseous implants. *Angle Orthod* 58:151–162, 1988.
212. Tweed CH: Indications for extraction of teeth in orthodontic procedure. *Am J Orthod Oral Surg* 30:405–428, 1944.
213. Tweed CH: Pre-orthodontic guidance procedure: classification of facial growth trends; treatment and timing. In Kraus BS, Riedel RA, editors: *Vistas in orthodontics*, Philadelphia, 1962, Lea & Febiger, pp 359–389.
214. Tweed CH: *Clinical orthodontics*. St. Louis, 1966, Mosby.
215. Umemori M, Sugawara J, Mitani H, et al: Skeletal anchorage system for open-bite correction. *Am J Orthod Dentofacial Orthop* 115:166–174, 1999.
216. Vargervik K, Miller AJ, Chierici G, et al: Morphologic response to changes in neuromuscular patterns experimentally induced by altered mode of respiration. *Am J Orthod* 85:115–124, 1984.
217. Verna C, Dalstra M, Melsen B: The rate and the type of orthodontic tooth movement is influenced by bone turnover in a rat model. *Eur J Orthod* 22:343–352, 2000.
218. Verna C, Melsen B: Tissue reaction to orthodontic tooth movement in different bone turnover conditions. *Orthod Craniofac Res* 6:155–163, 2003.
219. Vig PS, Weintraub JA, Brown C, Kowalski CJ: The duration of orthodontic treatment with and without extractions: a pilot study of five selected practices. *Am J Orthod Dentofacial Orthop* 97:45–51, 1990.
220. Wahl N: Orthodontics in 3 millennia. Chapter 1: Antiquity to the mid-19th century. *Am J Orthod Dentofacial Orthop* 127:255–259, 2005.
221. Wahl N: Orthodontics in 3 millennia. Chapter 2: Entering the modern era. *Am J Orthod Dentofacial Orthop* 127:510–515, 2005.
222. Wahl N: Orthodontics in 3 millennia. Chapter 5: The American Board of Orthodontics, Albert Ketcham, and early 20th-century appliances. *Am J Orthod Dentofacial Orthop* 128:535–540, 2005.
223. Wahl N: Orthodontics in 3 millennia. Chapter 6: More early 20th-century appliances and the extraction controversy. *Am J Orthod Dentofacial Orthop* 128:795–800, 2005.
224. Wehrbein H, Merz BR, Diedrich P, Glatzmaier J: The use of palatal implants for orthodontic anchorage. *Clin Oral Implants Res* 7:410–417, 1996.
225. Wehrbein H, Merz BR: Aspects of the use of endosseous palatal implants in orthodontic therapy. *J Esthet Dent* 10:315–324, 1998.
226. Weinberger BW: *Orthodontics: a historical review of its origin and evolution, including an extensive bibliography of orthodontic literature up to the time of specialization*. St. Louis, 1926, The CV Mosby Company.
227. Weinberger BW: Historical résumé of the evolution and growth of orthodontia. *J Am Dent Assoc* 21:2001–2021, 1934.
228. Weinberger BW: Dr Edward Hartley Angle: his influence on orthodontics. *Am J Orthod* 36:596–607, 1950.
229. Wenzel A, Henriksen J, Melsen B: Nasal respiratory resistance and head posture: effect of intra-nasal corticosteroid (Budesonide) in children with asthma and perennial rhinitis. *Am J Orthod* 84:422–426, 1983.
230. Wilcko WM, Wilcko MT, Bouquot JE, Ferguson DJ: Rapid orthodontics with alveolar reshaping: two case reports of decrowding. *Int J Periodontics Restorative Dent* 21:9–19, 2001.
231. Wilcko WM, Ferguson DJ, Bouguot JE, Wilcko MT: Rapid orthodontic decrowding with alveolar augmentation: case report. *World J Orthod* 4:197–205, 2003.
232. Wilson AD, Kent BE: A new translucent cement for dentistry. The glass ionomer cement. *Br Dent J* 132:133–135, 1972.
233. Wright CF: Status of orthodontic education prior to 1930. *Angle Orthod* 17:92–96, 1947.
234. Yaffe A, Fine N, Binderman I: Regional accelerated phenomenon in the mandible following mucoperiosteal flap surgery. *J Periodontol* 65:79–83, 1994.
235. Yaqoob O, Patel A, O'Neill J: Active postsurgical orthodontic treatment: how long does it take? *Br Orthod Soc Clin Effect Bull* 19:12, 2006.
236. Zachrisson BU, Årtun J: Enamel surface after various debonding techniques. *Am J Orthod* 75:121–127, 1979.

6

Periodontal Considerations in the Evaluation and Treatment of Dentofacial Deformities

JEFFREY C. POSNICK, DMD, MD

- Teeth
- The Tissues of the Periodontium
- Aging and the Periodontium
- Etiology of Periodontal Disease
- Assessing the Individual's Biologic and Anatomic Risk Factors
- Potential Detrimental Effects of Orthodontic "Camouflage" Maneuvers on the Periodontium
- Establishing a Healthy Periodontium and Periodontal Therapeutics
- Adult Orthodontic Treatment
- Conclusions

Degeneration of the periodontium is likely to accelerate in the presence of specific intrinsic biologic factors (i.e., the individual's biotype or predisposition of the periodontium), including the following: (1) a baseline jaw discrepancy with malocclusion; (2) the crowding of the teeth (i.e., the dental roots) within limited alveolar bone; (3) a forced mouth-breathing pattern in combination with a limited ability to close the lips together (i.e., inadequate incisor coverage) caused by anterior skeletal vertical (height) excess; and (4) an inadequate amount of attached gingiva at the clinical crown interface of the teeth. When attempting to achieve a favorable alignment of the teeth in each arch as well as improved occlusion through orthodontic maneuvers, these factors should be considered. Additional compounding factors that may also negatively affect the periodontium include the following: (1) active infection or inflammation of the periodontium; (2) non-biologic restorations at the dental–gingival interface; (3) para-occlusal habits (e.g., bruxism, clenching); (4) the use of medications that effect the saliva (e.g., antipsychotics); (5) local toxins (e.g., nicotine); (6) poor oral hygiene; and (7) the effects of age.

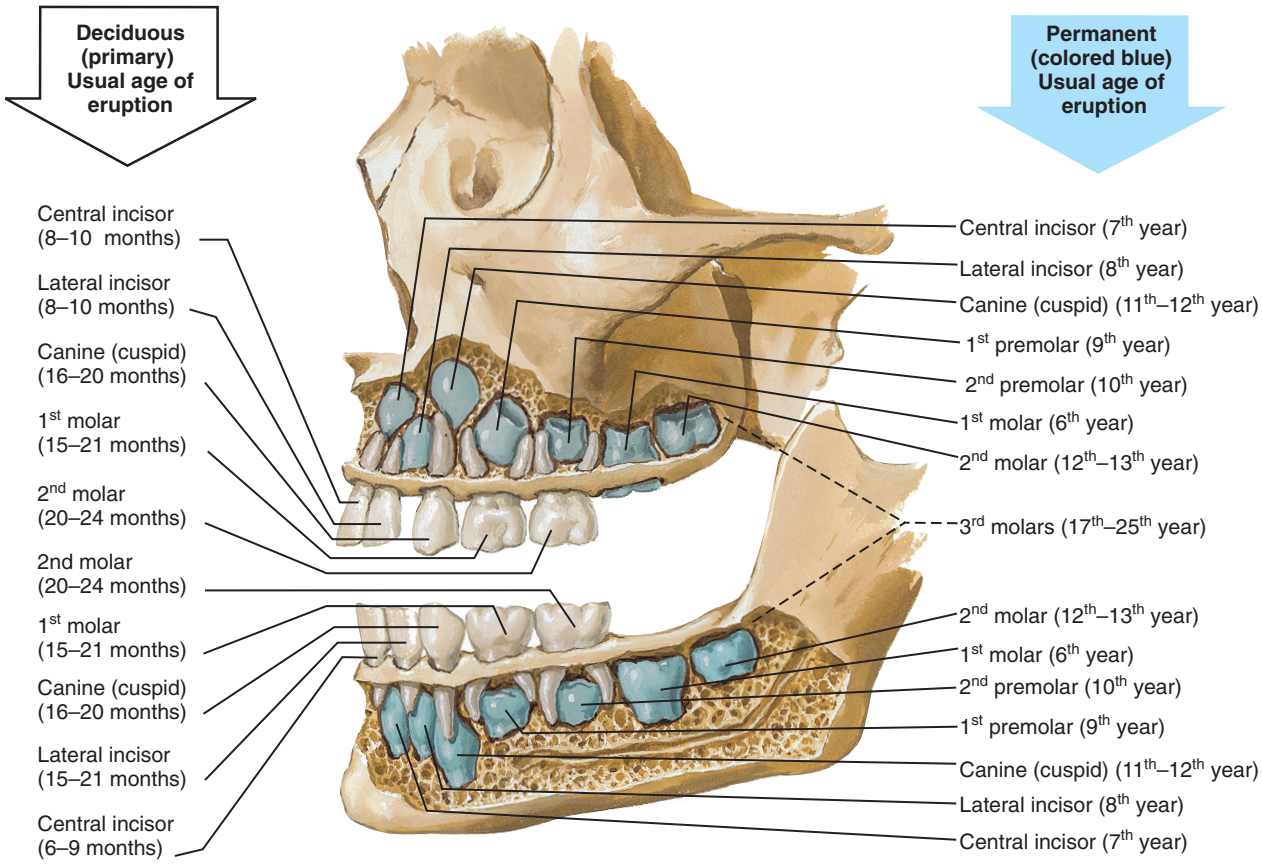
Teeth

General Information (Fig. 6-1)

Teeth are hard structures that are made up of an enamel-covered surface called the *crown* and a cementum-covered surface called the *root.* The root of the tooth is attached to the alveolar bone (i.e., the jaw bone) via the periodontal ligament (PDL). Teeth are primarily used for chewing, speech, and swallowing. The maxillary and mandibular arches both contain teeth. Humans have two sets of teeth during their lifetimes: the deciduous teeth, which are also known as the *primary dentition,* and the permanent teeth, which are known as the *secondary dentition.* Between the ages of 6 and 12 years, there is a mixed dentition during which both the primary and permanent teeth are present in the oral cavity at the same time.

There are 20 total deciduous teeth, which are composed of two incisors, one canine, and two molars in each of the four quadrants of the oral cavity. Under normal circumstances, no deciduous teeth are present at birth. Generally, by the third year of life, all 20 deciduous teeth have erupted.

There are 32 total permanent teeth, which are composed of two incisors, one canine, two premolars, and three molars in each of the four quadrants of the oral cavity. Usually the first permanent tooth to erupt into the oral cavity is the mandibular first molar. The first molar typically erupts at approximately 6 years of age. The molar erupts distal to the primary dentition and behind the second primary molar. The primary teeth eventually are replaced by the permanent teeth. If a permanent tooth is congenitally absent, then the primary tooth typically remains in the arch for an extended period of time.



• **Figure 6-1** Illustration of a sagittal view of a maxilla and mandible with the outer cortex of bone removed to demonstrate a fully erupted deciduous (primary) dentition and the location of the developing permanent tooth buds. Also indicated on the illustration are the usual ages of dental eruption for each tooth of both the deciduous and the permanent dentitions. *From* www.netterimages.com. *© Elsevier Inc. All rights reserved.*

Descriptions of the Surfaces of the Teeth (Fig. 6-2)

Labial: The surface of the anterior teeth that is closest to the lip
Buccal: The surface of the posterior teeth that is closest to the cheek
Facial: A synonym for *labial* or *buccal*
Lingual: Opposite the tongue in the mandibular arch and opposite the hard palate in the maxillary arch
Mesial: Closest to the midline of the dental arch
Distal: Farthest from the midline of the dental arch
Incisal: The cutting edge of the anterior teeth
Occlusal: Used for chewing on the posterior teeth

Basic Anatomy of a Tooth (Fig. 6-3)

Crown

- Anatomic crown: The portion of the tooth that has a surface of enamel.
- Clinical crown: The portion of the tooth that is exposed within the oral cavity.

Root

- Anatomic root: The portion of the tooth that has a surface of cementum.
- Clinical root: The portion of the tooth that is housed within the maxilla or mandible and that is not exposed to the oral cavity.

Apex of the Root

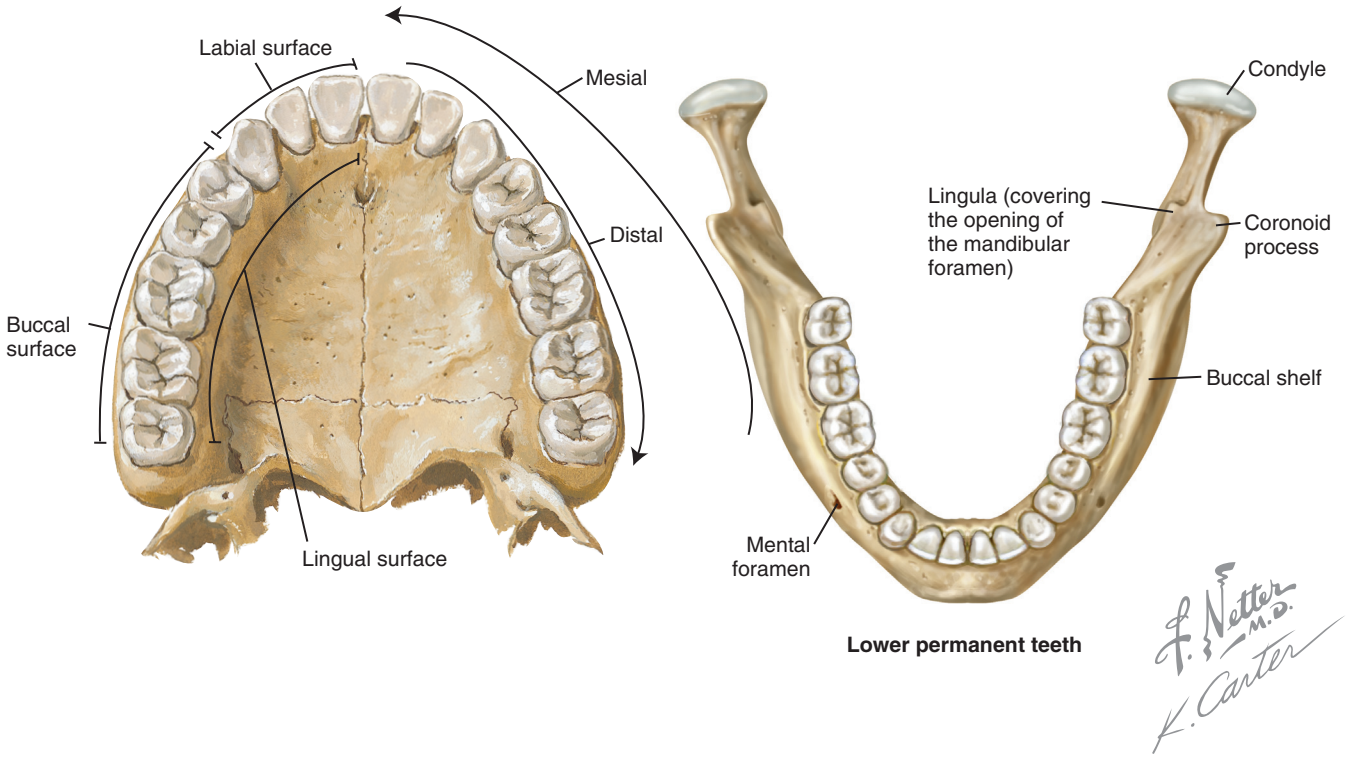
- The apex of the root is the end tip of the root, which is also the location of a small aperture at the point of each root. The aperture provides an entrance for the neurovascular connective tissue into the pulp cavity.

Cervical Line

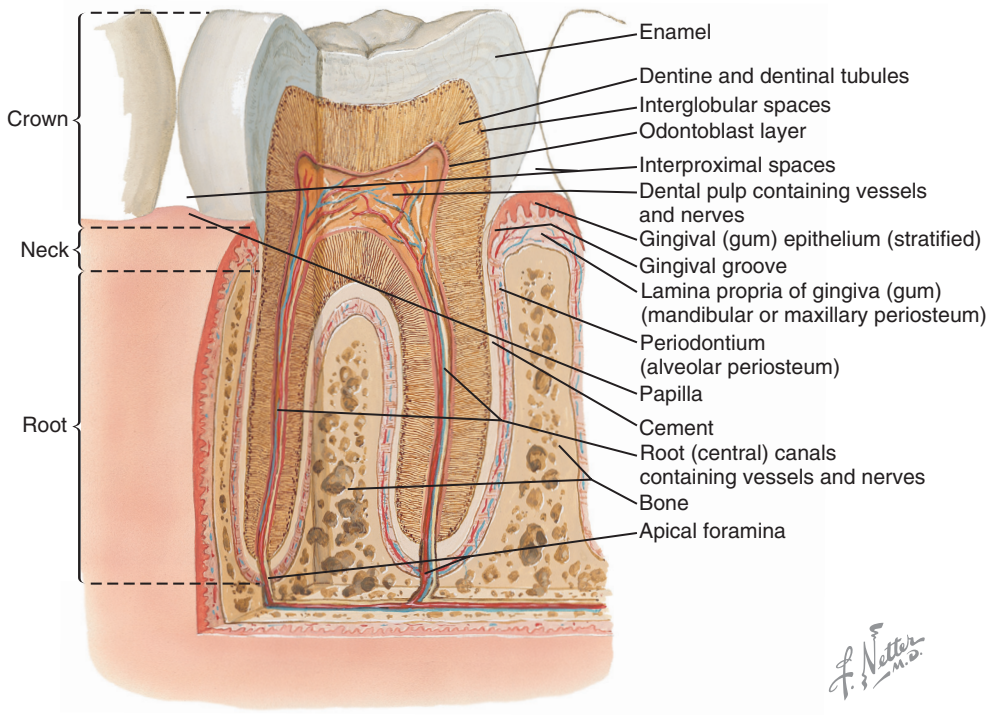
- The cervical line is the anatomic demarcation between the crown and the root. It is often called the *cementoenamel junction* (CEJ).

Enamel

- The enamel is the hard, shiny surface of the anatomic crown as well as the hardest portion of the tooth. It is



• **Figure 6-2** Illustrations of the fully erupted permanent dentition in the maxilla and mandible. The palate and the lingual view illustration are used to define terminology for the surfaces of a tooth. *From* www.netterimages.com. *© Elsevier Inc. All rights reserved.*



• **Figure 6-3** Illustration of a cross-sectional view through a permanent molar and the associated alveolus. Pertinent anatomic terminology is labeled. *From* www.netterimages.com. *© Elsevier Inc. All rights reserved.*

made of small hexagonal rods called *enamel prisms* that are parallel to one another.
- Enamel is 96% inorganic minerals by weight and 4% organic matter and water by weight.

Cementum

- The cementum is a thin, dull layer on the surface of the anatomic root. The cementum is similar in structure and chemical composition to bone. With age, cementum increases in thickness.
- Cementum is 45% to 50% inorganic material by weight and 50% to 55% organic matter and water by weight.

Dentin

- The dentin is the hard tissue that underlies both the enamel and the cementum and that constitutes the major portion of the tooth. Dentin is a modification of osseous tissue. It is composed of a number of dental tubules (small, wavy, and branching tubes) that are located in a dense matrix.
- Dentin is 70% inorganic material by weight, 20% organic material, and 10% water.

Cusp

- A cusp is an elevation on the occlusal surface of the molars and the premolars that makes up a divisional part of the tooth. The incisal edge of the canines is referred to as the cusp, and it is used for prehension (i.e., the grasping and tearing) of food.

Pulp Cavity

- The pulp cavity contains the dental pulp (neurovascular connective tissue). The pulp is separated into the pulp chamber, which is located in the coronal portion of the tooth, and the pulp canal, which is located in the root portion of the tooth.

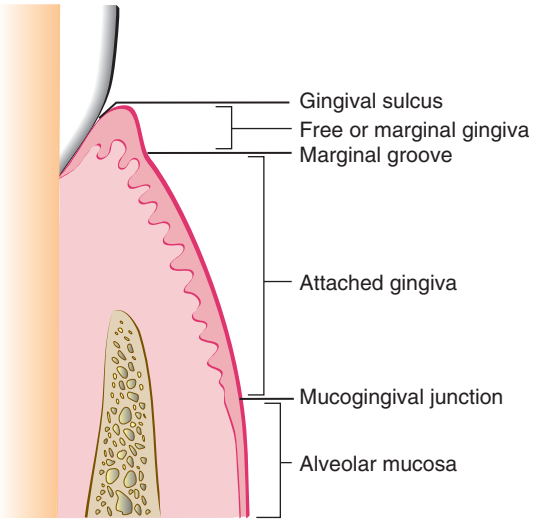
Cingulum

- The cingulum is a convex elevation that is located on the lingual surface of the crowns of anterior teeth just incisal to the CEJ.

The Tissues of the Periodontium

The periodontium includes the investing and supporting tissues of the teeth. It consists of two parts: the *attachment apparatus* (the cementum, the alveolar bone, and the intervening PDL) and the *dentogingival unit* (the gingival connective tissue that inserts into the supracrestal cementum and the sulcular and junctional epithelium as well as the latter's attachment to enamel) (Fig. 6-4). The cementum covers the root surfaces. It serves as the attachment for the fibers of the PDL to the tooth as does the (cortical) alveolar bone of the alveolar socket. The periodontium is affected by the individual's unique maxillofacial skeletal anatomy, by functional (occlusal) forces, and by degenerative cellular changes that occur with age. The greatest modifier of the periodontium is inflammatory disease.

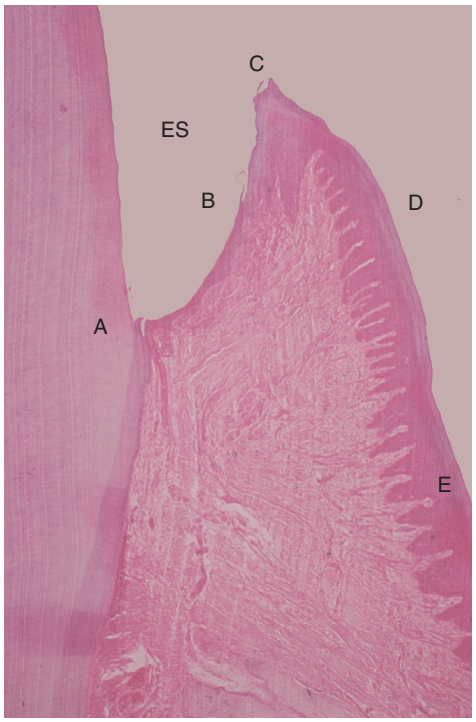
• **Figure 6-4** Intraoral view demonstrating the normal width of attached gingiva in the human permanent dentition. *From Newman MG, Takei HH, Klokkevold PR, Carranza FA:* Carranza's clinical periodontology, *ed 11, St. Louis, 2012, W.B. Saunders Company, Figure 2-3.*



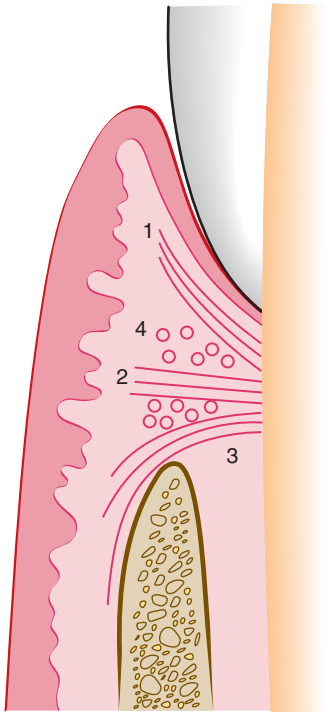
• **Figure 6-5** Diagram showing the anatomic landmarks of the gingiva. *From Newman MG, Takei HH, Klokkevold PR, Carranza FA:* Carranza's clinical periodontology, *ed 11, St. Louis, 2012, W.B. Saunders Company, Figure 2-2.*

The oral epithelium consists of three zones (Figs. 6-5 and 6-6):

1. *Masticatory mucosa (keratinized tissue):* This includes the gingiva, the hard palate, and the dorsal surface of the tongue. The gingiva is a fibrous investing tissue that immediately surrounds and is contiguous with its PDL and with the mucosal tissues of the mouth. Keratinized epithelium also covers the hard palate.
   - *Attached gingiva*: This is the portion of the gingiva that is firm, dense, stippled, and tightly bound to the underlying periosteum, tooth, and bone.
   - *Free gingiva*: This part of the gingiva surrounds the tooth and is not directly attached to the tooth surface.



• **Figure 6-6** Histologic appearance of healthy gingiva. A photomicrograph of a demineralized tooth with the gingival tissues in situ (hematoxylin and eosin stain, low magnification). The amelocemental junction *(A)* and the enamel space *(ES)* are shown. Gingival health is characterized by the organization of the epithelium into distinct zones: the junctional epithelium *(A* to *B)*, the sulcular epithelium *(B* to *C)*, the free gingiva *(C* to *D)*, and the attached gingiva *(D* to *E)*. The gingival connective tissue is composed of densely packed, organized, and interlacing collagen bundles. There are a few scattered inflammatory cells, but there is no significant inflammatory cell infiltrate. *From Newman MG, Takei HH, Klokkevold PR, Carranza FA:* Carranza's clinical periodontology, *ed 11, St. Louis, 2012, W.B. Saunders Company, Figure 21-1.*



• **Figure 6-7** Diagram of the gingival dental fibers that extend from the cementum *(1)* to the crest of the gingiva, *(2)* to the outer surface, and *(3)* external to the periosteum of the labial plate. Circular fibers *(4)* are shown in cross-section. *From Newman MG, Takei HH, Klokkevold PR, Carranza FA:* Carranza's clinical periodontology, *ed 11, St. Louis, 2012, W.B. Saunders Company, Figure 2-20.*

- *Marginal gingiva*: This is most coronal portion of the gingiva. This term is often used to refer to the free gingiva that forms the wall of the gingival crevice in a normal, healthy mouth.
- *Crevicular (sulcular) epithelium*: This is the nonkeratinized epithelium of the gingival crevice.
- *Junctional epithelium*: This involves a single layer or multiple layers of non-keratinized cells that adhere to the tooth surface at the base of the gingival crevice. This was formerly called the *epithelial attachment.*

2. *Lining mucosa (non-keratinized tissue):* This epithelium makes up the majority of the mucosa of the mouth and is the primary lining of the oral mucosa.
3. *Specialized mucosa:* This is found specifically in the regions of the taste buds on the dorsum of the tongue.

The *gingiva* is the part of the oral mucous membrane that covers the occlusal aspect of the alveolar processes of the jaws and that surrounds the necks of the teeth. The *junctional epithelium* and the *connective tissue attachment* have a characteristic dimension of 2 to 4 mm; this zone of tissue is called the *biologic width.* In general, an individual can only maintain health when the sulcus depth is no more than 3 to 4 mm. Greater depth provides a safe haven for bacteria that cannot be easily removed through normal oral hygiene maneuvers. A lower-than-average alveolar crest level is acceptable as long as it is stable (i.e., not progressive) and the periodontium is free of active disease. On the palatal surface of the teeth, the *attached gingiva* blends with the equally firm and highly keratinized *palatal (masticatory) mucosa.* The *interdental gingiva* occupies the gingival embrasure (i.e., the interproximal space beneath the area of tooth contact). It consists of the facial papilla, the lingual papilla, and the valley-like depression that connects the two in the interproximal contact area, which is called the *col.* In the absence of proximal tooth contact, the gingiva is firmly bound over the interdental bone; it forms a smooth, round surface without a triangular interdental papilla. The connective tissue of the gingiva is densely collagenous, and it contains collagen fiber bundles called *gingival fibers* (Fig. 6-7). When these fiber bundles insert into the tooth's cementum, they are called *Sharpey's fibers,* and they result in the mechanical attachment of the sulcular and supracrestal (i.e., crestal to the alveolar process) gingiva. These fibers are able to withstand masticatory forces without deflection or detachment from the tooth surface. The connective tissue attachments extend from just apical to the *junctional*

epithelium of the *sulcular (crevicular) gingiva* to the supracrestal cementum of the root.

The junctional epithelium is connected to the tooth surface via a hemidesmosomal adhesion. These two ectodermal tissues and their interface act to block the flow of toxic chemicals and organisms from the oral cavity into the body.

The *gingival sulcus* contains a fluid that is derived from the gingival connective tissue and that flows through the thin epithelium of the sulcular wall. The gingival fluid is cleansing, and it contains sticky plasma proteins to increase the adhesion of the epithelial attachment to the tooth. The fluid possesses antimicrobial properties, and it is active in its defense of the gingiva. It may also serve as a medium for bacterial growth, which contributes to the formation of dental plaque and calculus. The amount of gingival fluid (exudate) increases with inflammation. The composition of gingival fluid has many similarities to that of blood serum.

The *attached gingiva* is continuous with the *marginal gingiva,* and it consists of stratified squamous epithelium and an underlying connective tissue stroma. The superficial layer is keratinized, parakeratinized, or both. The connective tissue of the gingiva is known as the *lamina propria.* It is densely collagenous, with few elastic fibers, and it consists of two layers: a papillary layer next to the epithelium and a reticular layer next to the periosteum of the alveolar process.

Each *interdental papilla* consists of a central core of densely collagenous connective tissue covered by stratified squamous epithelium. The facial and lingual papillae are joined with the connective tissue of the col and the stratified squamous epithelium from the adjacent interdental papillae. The epithelium of the col is not keratinized and therefore more susceptible to inflammation. The shape of the interdental gingival papillae correlates with the shape of the teeth and the embrasures. The interdental papillae may be broad or narrow, depending on dental positioning and points of connection.

In a normal physiologic state, the gingiva is firm and resilient. With the exception of the movable free margins (i.e., the marginal gingiva), it is tightly bound to the underlying bone. This is the result of the collagenous nature of the lamina propria and its integration with the periosteum of the alveolar process. The visual stippling that is seen when viewing the gingival surface represents the pulling of the superficial gingival layer by the tight connective tissue attachments to the underlying alveolar process.

The Periodontal Ligament

The *PDL* is the connective tissue structure that surrounds the root and that connects the root with the bone in the tooth socket. It is continuous with the connective tissue of the gingiva, and it communicates with the bone through vascular channels. The principal fibers of the PDL are collagenous organized bundles that insert into the cementum on one side and into the bone on the other side; they are called *Sharpey's fibers.* Cellular elements of the PDL include fibroblasts, endothelial cells, cementoblasts, osteoblasts, osteoclasts, tissue macrophages, and stratified epithelial cells. The PDL has physical, formative, nutritional, and sensory functions. These include the physical functions of the transmission of occlusal forces to the bone, the attachment of the teeth to the bone, the maintenance of the gingival tissues in relationship to the teeth, the resistance to the impact of occlusal forces, and the protection of the vessels and nerves from injury by mechanical forces. Destruction of the PDL and the alveolar bone by disease or injury disrupts the balance between the periodontium and the occlusal forces. The PDL supplies nutrients to the cementum, the bone, and the gingiva through the blood vessels; it also provides lymphatic drainage to the same structures. The innervation of the PDL provides proprioception and tactile sensitivity. The PDL includes transseptal, alveolar–crestal, oblique, and apical oriented fibers.

Cementum

Cementum is the calcified mesenchymal tissue that forms the outer covering of the tooth root. There are two types of cementum: acellular and cellular. Both consist of a calcified interfibrillar matrix and collagen fibrils. The cellular type contains cementocytes in individual spaces (lacunae) that allow for communication with each other through a system of canaliculi. There are two types of collagen fibers. The first type is Sharpey's fibers, which are the principal fibers of the PDL and which are formed by fibroblasts. The second group of fibers is thought to be produced by the cementoblasts and to form an interfibrillar substance. The distribution of acellular and cellular cementum varies. The coronal half of the root is usually covered by the acellular type of cementum, whereas the cellular cementum is more common in the apical half of the root. The inorganic content of cementum includes hydroxyapatite, a carbohydrate–protein complex, and acid mucopolysaccharides.

The relationship of the interface between the cementum and the enamel at the crown–root interface varies. Cementum overlaps the enamel in about two thirds of cases. In the other third, there is an edge-to-edge arrangement in which a small percentage of the cementum and the enamel fail to meet.

When cementum resorption occurs, it is seen microscopically as concavities on the root surface. Multinucleated giant cells and large mononuclear macrophages are generally found adjacent to the cementum that is undergoing active resorption. The resorptive process may extend into the underlying dentin and even into the pulp. In a physiologic setting, embedded fibers of the PDL reestablish a functional relationship in the new cementum. Cementum repair requires the presence of viable connective tissue. If epithelium proliferates into an area of cementum, then resorption rather than repair will likely take place. Fusion of the cementum and the alveolar bone with obliteration of

the PDL is termed *ankylosis.* When ankylosis does occur, it generally happens after chronic periapical inflammation, tooth replantation, or significant occlusal trauma. It may also represent a congenital failure of eruption.

The *anatomic root* is the portion of the tooth that is normally covered by cementum. The anatomic crown is the portion of the tooth that is covered by enamel. The *clinical crown* is the part of the tooth that includes the anatomic part of the crown and the part of the root that has been denuded of periodontium and that is visible in the oral cavity. The clinical root is that portion of the tooth that remains covered by periodontal tissues (i.e., the PDL, the cementum, and the gingiva). Exposure of the root via the apical migration of the gingiva margin is called *gingival recession.* Exposure of the root to the oral cavity via the apical migration of the junctional epithelium without apical migration of the gingival margin results in pocketing. In either case, periodontal degenerative changes result in the permanent exposure of connective tissue (i.e., the dentin and the cementum) to the external environment.

The Alveolar Bone

All parts of the alveolar process serve to support the teeth. Occlusal forces are transmitted through the PDL to the inner wall of the alveolus, which is then supported by the cancellous trabeculae. The cancellous trabeculae in turn are buttressed by the labial and lingual cortical plates. The alveolar process is the bone that supports the tooth socket, and it consists of the following:

- The inner socket wall of thin compact bone (the alveolar bone proper);
- The supporting alveolar medullary bone (the cancellous trabeculae); and
- The facial and lingual plates (compact bone).

The alveolar process consists of calcified matrix with osteocytes that are enclosed within spaces called *lacunae.* The *canaliculi* form an anastomosing system through the intercellular matrix of the bone, which brings oxygen and nutrients to the osteocytes and which removes metabolic waste products. In the cancellous trabeculae, the matrix is arranged in lamellae that are demarcated from each other by prominent cement linings. The *compact alveolar bone* (i.e., the bony lining of the alveolus) consists of closely arranged lamellae and Haversian systems. The principal fibers of the PDL that anchor the tooth in the socket are embedded into the alveolar bone and are referred to as *Sharpey's fibers.* The *socket wall* consists of dense laminated bone. The cancellous portion of the alveolar bone consists of trabeculae, which enclose irregularly shaped marrow spaces that are lined with a layer of thin, flattened ostial cells.

The bony wall of the tooth socket appears radiographically as a thin radiopaque line; it is called the *lamina dura*. The alveolar bone is perforated by numerous channels that contain blood vessels, lymph vessels, and nerves, which link the PDL with the cancellous portion of the alveolar bone. The vascular supply of the bone is derived from blood vessels in the PDL and in the marrow spaces and from small branches of peripheral vessels that penetrate the cortical plates. The interdental septum consists of cancellous bone that is bordered by the socket walls of approximating teeth and the facial and lingual cortical plates.

The mesiodistal angulation of the crest of the interdental septum parallels a line drawn between the CEJs of the approximating teeth. The average distance between the crest of the alveolar bone and the CEJ in the mandibular anterior region in an individual who is periodontally healthy is approximately 1 mm. With disease progression, the distance between the bone ridge and the CEJ increases throughout the mouth.

The alveolar bone contour normally conforms to the prominence of the roots of the teeth, with regions of mild depression in between. The height and thickness of the facial and lingual bony plates are affected by the alignment of the teeth and the angulation of the roots in the bone. For example, in the patient with mandibular deficiency and excessive incisor procumbency, the labial bone may be thin, and apical migration may be seen along the tooth surface. When the mandibular incisor teeth are lingually positioned (i.e., skeletal Class III), the facial bony plate is generally thicker than normal. Another example is seen when the maxillary molar roots are at acute angles to the palatal bone. The effects of the root location and angulation result in recession of the marginal bone.

An isolated area in which a tooth root is denuded of labial cortical bone, the root surface is covered only by periosteum and overlying gingiva, and the marginal bone remains intact is termed *fenestration*. An area of the tooth root in which there is denuded bone and where the root surface is covered only by periosteum where the root denudation extends to the margin is called *dehiscence*. Dehiscences are more common on the labial surface of the lower anterior teeth and on the molars and premolars of the maxilla. Congenital or developmental jaw hypoplasia with dental crowding and trauma from occlusal treatments or orthodontics are common etiologic factors for fenestrations and dehiscences.

Alveolar bone is a structure that is constantly in a state of flux. It is maintained by a delicate balance between bone formation and bone resorption, and it is controlled by local and systemic influences. Bone is resorbed in areas of pressure and deposited in areas of tension. With age and normal occlusal forces, there is a tendency for the mesial migration of the teeth. As a result, the associated alveolar bone is remodeled via resorption and deposition.

The purpose of the alveolar process is to support the teeth both while they are at rest and while they are functioning. Its structure is dependent on the stimulation that it receives from masticatory function, because it undergoes

constant remodeling in response to occlusal forces. When force is applied to a tooth, that force is displaced against the resilient PDL, thereby creating areas of tension and compression. The facial and lingual walls of the tooth socket bend in the direction of the force. When the force is released, the tooth, the PDL, and the bone spring back toward their original positions. In response to these forces, osteoblasts and newly formed osteoid will line the socket in areas of tension, whereas osteoclasts position themselves and resorption occurs in areas of pressure. The bone trabeculae are aligned in the path of the tension and compressive stresses to provide maximum resistance to the occlusal force with the minimum of bone substance. Forces that exceed the adaptive capacity of the bone produce injury. When occlusal forces are increased, the cancellous trabeculae also increase in number and thickness to provide necessary support.

Aging and the Periodontium

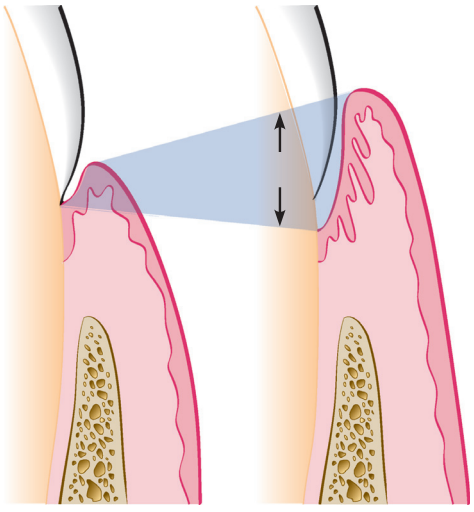
The prevalence of periodontal disease with tissue destruction and the loss of teeth and tooth structure tends to increase with age.[106,155,160,171] Another consequence of age is reduced tissue elasticity through the degeneration of the elastic fibers. Hormonal changes also occur, and these change the local tissue environment. Traditional thinking is that the degenerative gingival changes associated with aging may include recession, diminished keratinization, reduced stippling, decreased connective tissue cellularity, increased intercellular substances, and reduced oxygen consumption. In the PDL, degenerative aging effects are thought to include a decrease in elastic fibers and a decrease in vascularity and mitotic activity. If these effects occur, the ability of the alveolar bone to withstand occlusal forces is diminished. Frequent changes in tooth structure with age are seen, including occlusal wear with a loss of enamel substance that reduces cusp height and inclination. The degree of attrition is influenced by the masticatory musculature, the consistency of the food eaten, and occlusal factors and habits such as clenching and bruxism. A degree of continued tooth eruption usually occurs as teeth wear. As a result, the clinical crown may become longer, which creates further leverage on the bone with masticatory forces. An opposing factor is the fact that the clinical crowns are simultaneously reduced through attrition, often with an equilibrium or balance being present between the teeth and their bony support. The wear of the teeth along the proximal surfaces may also occur, which results in mesial migration. On average, proximal wear reduces the anteroposterior length of the dental arch by approximately 5 mm by the age of 40 years and by twice that by life's end. When chronic periodontal disease is added to physiologic degenerative aging, the destructive response of the periodontium is exacerbated. There may be continued gingival recession, attrition, and the reduction of alveolar bone height as a result of a combination of these factors.[31]

Etiology of Periodontal Disease

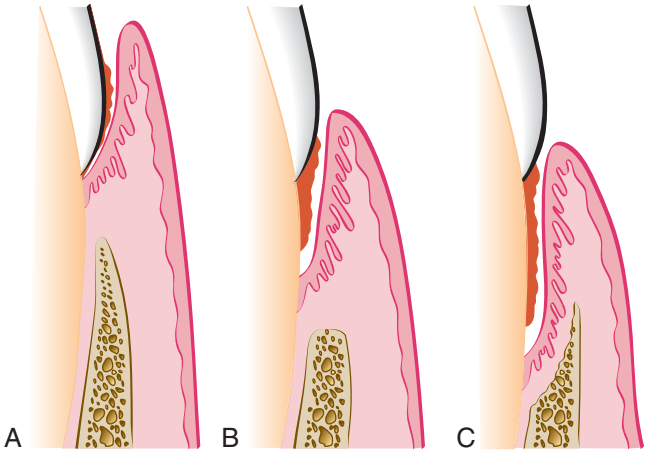
Gingivitis and Periodontitis

Inflammation of the gingiva, which is also known as *gingivitis,* is the most common form of gingival disease (Figs. 6-8 through 6-12).[1,19,177,183,216] This occurs as a result of local irritants, such as the toxins released by the microorganisms related to dental plaque and calculus. Foreign bodies (e.g., orthodontic appliances, irregular dental restorations) may also serve as local irritants as well as plaque traps. The

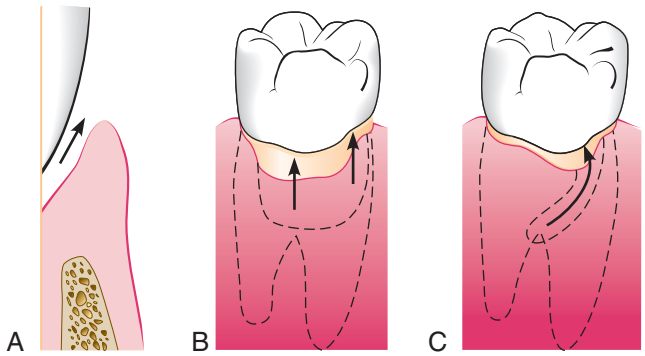
• **Figure 6-8** Photograph of the maxillary anterior dentoalveolar region. Dehiscence of the labial cortical plate of the canine and fenestration of the labial cortical plate of the first premolar are demonstrated. *From Newman MG, Takei HH, Klokkevold PR, Carranza FA:* Carranza's clinical periodontology, *ed 11, St. Louis, 2012, W.B. Saunders Company, Figure 2-60.*



• **Figure 6-9** Illustration of pocket formation that indicates expansion in two directions *(arrows)* from the normal gingival sulcus *(left)* to the periodontal pocket *(right). From Newman MG, Takei HH, Klokkevold PR, Carranza FA:* Carranza's clinical periodontology, *ed 11, St. Louis, 2012, W.B. Saunders Company, Figure 13-1.*

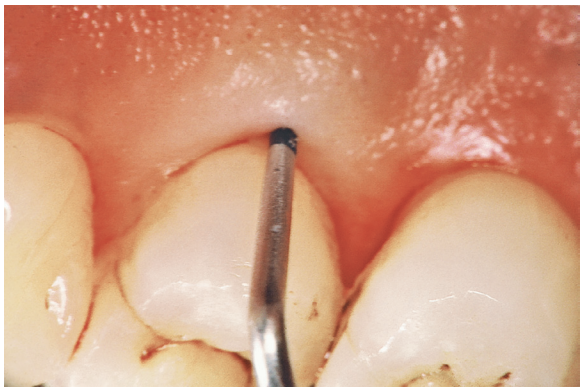


• **Figure 6-10** Different types of periodontal pockets. With a gingival pocket *(A)*, there is no destruction of the supporting periodontal tissues. With a suprabony pocket *(B)*, the base of the pocket is coronal to the level of the underlying bone, and bone loss is horizontal. With an intrabony pocket *(C)*, the base of the pocket is apical to the level of the adjacent bone, and bone loss is vertical. *From Newman MG, Takei HH, Klokkevold PR, Carranza FA:* Carranza's clinical periodontology, *ed 11, St. Louis, 2012, W.B. Saunders Company, Figure 13-2.*



• **Figure 6-11** Classification of pockets according to involved tooth surfaces. **A,** Simple pocket. **B,** Compound pocket. **C,** Complex pocket. *From Newman MG, Takei HH, Klokkevold PR, Carranza FA:* Carranza's clinical periodontology, *ed 11, St. Louis, 2012, W.B. Saunders Company, Figure 13-3.*

inflammation caused by local irritants can result in ulcerative, necrotic, and proliferative changes in the gingival tissues.[29,30,34,99,165,170,221] When there is deepening of the gingival sulcus, there can be injury to the supporting periodontal tissues; this may become an irreversible process. With continued inflammation, hyperplastic changes of the gingiva occur, and the crest of the gingival margin extends toward the crown. Inflammation causes a proliferation of and a change in the quality of the sulcus and the junctional epithelium such that their normal protective nature becomes dysfunctional. The sulcus becomes a pocket; ulceration through the epithelial barrier with exposure of the underlying connective tissue to the oral cavity is a frequent occurrence. The organisms and their toxins are then able to access the exposed connective tissue, which undergoes further pathologic changes.[93,133,135,154,193] As the process continues,



• **Figure 6-12** Probing of a deep periodontal pocket. The entire length of the periodontal probe has been inserted into the base of a pocket in the palatal surface of the first premolar. *From Newman MG, Takei HH, Klokkevold PR, Carranza FA:* Carranza's clinical periodontology, *ed 11, St. Louis, 2012, W.B. Saunders Company, Figure 13-4.*

the epithelial junction may separate from the root, and the pocket will migrate downward. The epithelium of the lateral wall of the pocket proliferates with inflammatory tissue, which results in varying degrees of degeneration and necrosis. Intrabony periodontal pockets are said to be present when the base is apical to the level of the alveolar bone. The extension of inflammation from the margin of the gingiva into the supporting periodontal tissues marks the transition from gingivitis to periodontitis.[6,40,41,107,117,175,200] The essential problem of periodontal disease is the destruction of alveolar bone with a loss of crestal height and PDL destruction. If periodontal disease is left untreated, it will lead to the loosening and loss of the teeth.

Trauma from Occlusion

Occlusal forces affect the condition and structure of the periodontium.[53,54,59,104,129,134,160,181,191,204] To remain structurally and metabolically sound, the PDLs and the alveolar bone require the mechanical stimulation of occlusal forces. When occlusal forces exceed the adaptive capacity of the tissue, injury occurs. The injury that occurs to the periodontium is called *trauma from occlusion,* and it can be classified as either primary or secondary occlusal trauma. *Primary occlusal trauma* occurs when greater-than-normal occlusal forces are placed on teeth with a normal periodontal attachment apparatus (i.e., those that are periodontally stable). *Secondary occlusal trauma* occurs when normal occlusal forces are placed on teeth with compromised periodontal attachment (i.e., those with periodontal disease).

The periodontium attempts to accommodate the functional demands that are placed on it by the masticatory system. The adaptive capacity of the periodontium varies from person to person. The periodontium is influenced by the severity, direction, frequency, and duration of the force that is put on it and the patient's specific anatomy. The principal fibers of the PDL are arranged so that they can best accommodate occlusal forces in the long axis of the

tooth. Forces that are not aligned well with the tooth's load-bearing capacity place increased compression on specific locations of the PDL. This may result in resorption of bone, depending on the extent and duration of the occlusal force. If the trauma is excessive, a destructive change in the periodontium will occur. If the injurious force is relieved rapidly enough, repair rather than destruction will occur. When trauma is combined with active infection, rapid irreversible destruction of the periodontium is more likely. The degree to which these factors interact and cause destruction remains controversial. There is a need for ongoing clinical research to clarify these issues.

Traditional thinking is that trauma from occlusion is caused by alterations in the occlusal forces, the reduced capacity of the periodontium to withstand what would otherwise be considered normal, or both. Malocclusion is a risk factor for occlusal trauma, but damage may also occur in the presence of a "normal" occlusion. *Trauma from occlusion* refers to the tissue injury rather than the specific occlusion. Increased masticatory forces are not traumatic if the periodontium can accommodate them. Trauma from occlusion and inflammation from an infectious or foreign-body process are different pathologic processes that can occur either in isolation or together. Inflammation typically starts in the gingiva and then spreads into the supporting periodontal tissues. Trauma from occlusion causes pathologic change of the attachment apparatus. When this occurs in the presence of inflammatory processes, these two pathogenic factors may act synergistically to cause greater damage to the periodontium than the sum of each factor's singular effect. These combined components are called *co-destructive factors.*

The histopathologic lesion of occlusal trauma is called the *lesion of the attachment apparatus,* and it is characterized by the following:

- Hyaline degeneration of the PDL
- Vascular thrombosis in the PDL
- Necrosis in the PDL
- Frontal and rear "undermining" resorption of the alveolar bone
- Cementum resorption

The lesion of the attachment apparatus can occur and progress rapidly. It is believed that, when the lesion interacts with plaque-induced inflammatory responses, then the loss of the periodontium is likely to be accelerated.

Effects of Orthodontic Appliances and Tooth Movement Forces

Tooth movement during orthodontic therapy is the result of controlled forces placed on the teeth and then transmitted to the PDL. Strong or heavy forces (i.e., forces that far exceed capillary blood pressure) result in the crushing of the PDL on the compression side of the tooth, with local ischemia and degeneration (i.e., hyalinization). Moderate forces that exceed capillary blood pressure result in the compression of the PDL with a delay in bone resorption and the movement of the tooth.[89] Light continuous forces that are less than the capillary blood pressure result in only limited ischemia to the PDL, with gradual bone resorption on the compression side.[174] The patient's age is not a contraindication to orthodontic treatment per se. Interestingly, in the adult, the hyalinized (necrotic) zones are formed more readily on the pressure side of the orthodontically moving tooth; this will temporarily slow tooth movement.[62] The hyalinized zone is soon eliminated with the reorganization of the tissues, first through the resorption of the marrow spaces (thus undermining resorption) and then through the repair of the PDL and finally of the alveolar bone.[172] The anticipated regeneration of the PDL on the compression side and the formation of new bone on the tension side will likely be hampered by the presence of active inflammation in the periodontal tissues (i.e., periodontitis).[169] This pathologic response is dependent on how long the PDL remains compromised. This is the reason why inflammation should be controlled through effective periodontal treatment before orthodontic tooth movement.*

Until the mid 1980s, heavy intermittent orthodontic forces were routinely used, and this required patient visits every 3 to 4 weeks.[176] This allowed the hyalinized fibers to recover before another heavy orthodontic force was applied. Contemporary orthodontics involves the use of light, continuous force. This moves the teeth with less discomfort and more rapidly, and it also allows visits to be spaced at longer intervals.

In a patient with a periodontally compromised dentition and with a baseline loss of alveolar bone, the center of resistance of the involved teeth moves apically.[141,157] The net effect is that the involved teeth are more prone to tipping rather than to bodily movement when orthodontic forces are applied. To achieve improvement in the periodontium, orthodontic treatment requires a combination of light controlled forces as well as the movement of teeth more completely into the alveolar housing. In the presence of active disease, orthodontic therapy should be postponed until effective periodontal treatment is accomplished. This approach to orthodontic tooth movement has been shown to improve any preexisting compromised periodontium.

Interestingly, the orthodontic movement of endodontically treated teeth is not a risk factor to the periodontium, because the response of the PDL is not affected by the pulp. Some studies do indicate that endodontically treated teeth are slightly more prone to root resorption during orthodontic treatment as compared with teeth with normal vitality.

Teeth that are already tipped outside of the cortical plate (e.g., proclined mandibular incisors in the individual with Class II malocclusion) and that are orthodontically

*References 20, 21, 24, 25, 36, 43, 47, 49, 56, 66, 100, 111, 115, 116, 121, 122, 142, 144, 151, 156, 173, 182, 192, 197, 198, 202, 218-220

uprighted into sound alveolar housing are likely to improve in overall periodontal health, even when the gingiva levels remain borderline. Animal studies indicate that, without the presence of plaque, orthodontic forces on the teeth do not in themselves induce gingivitis.[67] In the presence of plaque, however, similar forces can cause angular bone defects and, with tipping or torquing movements, gingival attachment loss (i.e., recession) can occur.[68-70] Clinical studies have demonstrated that, with adequate plaque control, even teeth with longstanding reduced periodontal support can undergo successful tooth movement without further compromise.[63] In patients with no active periodontal disease and with good oral hygiene—and even in adults with reduced but healthy residual periodontium—physiologic orthodontic treatment causes no significant detrimental long-term effects on the periodontal attachment, including the bone levels. Physiologic tooth movement involves light forces and the movement of teeth into (not outside of) alveolar bone.[37-39]

In a cross-sectional study, radiographic crestal bone levels in adults (N = 104) who completed orthodontic treatment at least 10 years previously were shown to be no different than those of matched control subjects (N = 76).[161] In a 2-year post orthodontic study, Trosello and colleagues compared adult women who had multi-banded orthodontic therapy (N = 30) with age-matched (non-orthodontically treated) controls (N = 30).[203] It was found that the orthodontically treated patients had a higher prevalence of root resorption (17% versus 2%), although there was a lower prevalence of mucogingival defects (5% versus 12%). The root resorption differences were most common in the maxillary incisors, followed by the mandibular incisors. It appears that, in adults, when biologically sound orthodontic maneuvers are carried out, minimal detrimental effects on the health of the periodontium occur. In the short term, gingivitis and gingival hyperplasia may occur, but there is no attachment loss or irreversible effects. In the long term, when the teeth are moved into (not out of) the alveolar bone, mild root resorption (i.e., 1.0 to 1.5 mm) may be documented, but attachment loss (i.e., irreversible change) only occurs in areas of active periodontitis.

It is known that *plaque* is the primary etiologic factor of *gingivitis.* A patient's inability to clean adequately around orthodontic devices (e.g., banded teeth, brackets, wires, springs, coils, elastics, plates) will promote plaque accumulation, which can lead to gingival inflammation. Before the extensive use of bonded brackets, overgrowth of anaerobes in the patient's sulcus was typical.[57] Fortunately, the common practice of placing numerous subgingival orthodontic bands in each quadrant has gone by the wayside. Nevertheless, a shift in the subgingival microflora to a more pathogenic population that is similar to what is seen in periodontal disease sites may occur with use of orthodontic devices.[131] Even without banded bracket appliances, an active and diligent oral hygiene program for the patient that includes frequent periodic checkups by an appropriate dentist is required throughout orthodontic treatment.

Dental Restorations: Gingival Interface

The relationship between bacterial plaque accumulation and gingival inflammation has been documented since at least the 1960s.[132] A patient's susceptibility to gingival inflammation is not based solely on the mere quantity of dental plaque but also on the virulence of the resident plaque microorganisms. The bacterial composition of dental plaque is dynamic, and the pathogenicity of each specific organism is subject to change over time. It is a dental dictum that creating a confluent restorative–gingival interface is an important factor that allows patients' oral hygiene efforts to reduce the accumulation of plaque and microorganisms and to decrease—if not eliminate—the resulting gingivitis. For this reason, emphasis should be placed on the marginal integrity of dental restorations; the coronal contour of the restoration; the embrasure and contact; and the marginal location of the restoration. The proper marginal location of a restoration relative to the alveolar bone is thought to be one of the most important parameters for achieving and maintaining long-term gingival health.[92,124-126,139] Similarly, all patients who are undergoing dental therapy should be philosophically taught and technically trained to perform effective plaque-removal practices.

Assessing the Individual's Biologic and Anatomic Risk Factors

Destruction of the periodontium is more likely to occur in the presence of known risk factors. Specific risk factors include 1) infection, 2) primary occlusal trauma, 3) iatrogenic causes, and 4) intrinsic biologic anatomic aspects. The known intrinsic (unique to the individual) biologic anatomic risk factors for periodontal disease may be referred to as the individual's *biotype* (Figs. 6-13 through 6-29). These factors include the following:

- Inadequate attached gingiva surrounding the clinical crowns of the teeth
- Inadequate alveolar bone to house the roots of the full complement of teeth in each arch
- Inadequate nasal airflow with a forced mouth-breathing pattern, often in association with vertical (skeletal) excess and inadequate lip closure, with limited coverage of the incisors and the gingiva
- The effects of malocclusion associated with a jaw deformity

The individual's biotype may pose risk factors for periodontal tissue loss. These risk factors can often be overcome or at least managed through surgical, orthodontic, and dental interventions; meticulous home care; and the avoidance of toxins such as tobacco, certain medications, and certain chemicals.

Text continued on p. 200

• **Figure 6-13** A 50-year-old man was referred by his general dentist to an orthodontist, who then referred the patient for surgical evaluation. The patient had a history of juvenile rheumatoid arthritis with temporomandibular joint involvement. There is condylar erosion with loss of posterior facial height with retrusion and clockwise rotation of the mandible, which resulted in a Class II anterior open-bite malocclusion. Adequate painless vertical mouth opening is present, and no symptomatic temporomandibular disorder is present. Over the years, there has been destruction of the dentition and the periodontal apparatus with a degree of labial bone loss and gingival recession. At referral to this surgeon, the patient had a history that documented heavy snoring, restless sleeping, and daytime somnolence. An attended polysomnogram confirmed a respiratory disturbance index of 10 events per hour. There is intranasal obstruction as a result of septal deviation and inferior turbinate enlargement, a retropositioned palate as a result of maxillary retrusion, and a retropositioned tongue as a result of mandibular retrusion. The soft palate and the tongue are normal with regard to function and size. The patient had previously undergone tonsillectomy. His biologic and anatomic risk factors for periodontal disease include inadequate alveolar bone (i.e., an inadequate alveolar bone/dental root ratio); nasal obstruction and mouth breathing with lip incompetence; and malocclusion with secondary trauma. A comprehensive approach was recommended to address the patient's upper airway and breathing difficulties, his occlusion issues and his long-term dental health, and his facial aesthetics. **A,** Frontal facial and occlusal views before treatment. **B,** Profile facial view and lateral cephalometric radiograph before treatment. **C,** Panorex radiograph before treatment.

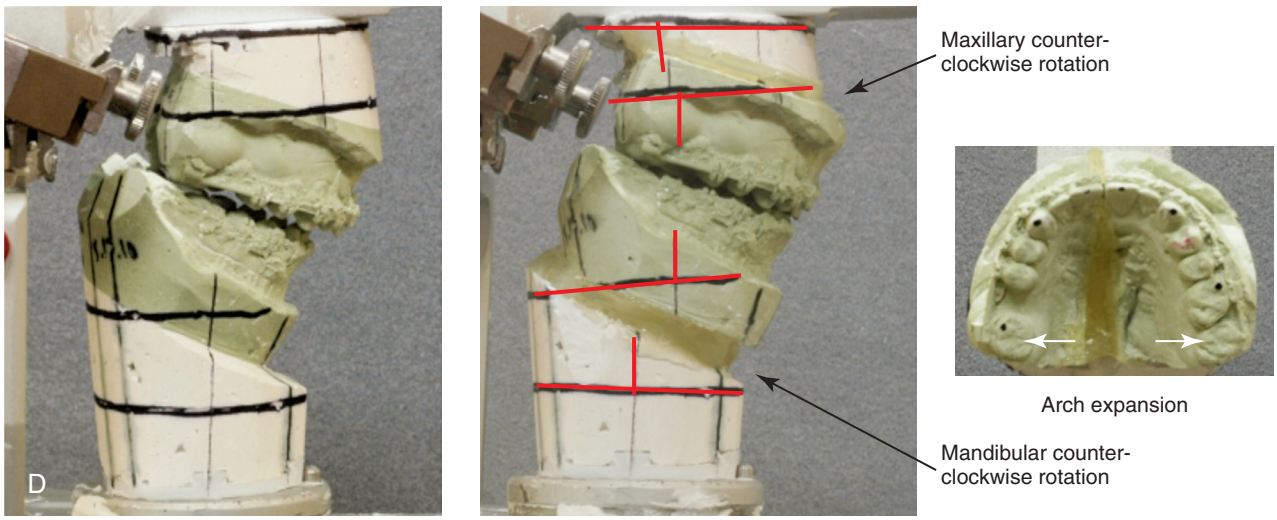
• **Figure 6-14** A 26-year-old college graduate arrived for surgical evaluation. Since the mixed dentition, he was recognized to have a developmental jaw deformity that was characterized by maxillary deficiency in combination with relative mandibular excess. Camouflage orthodontic treatment including four bicuspid extractions was carried out when he was between 12 and 16 years old. A residual Class III negative overjet with an anterior open-bite malocclusion remains. The patient had never been able to breathe well through his nose as a result of septal deviation and inferior turbinate enlargement. Secondary occlusal trauma resulted in the need for root canal therapy and crowns in all four posterior quadrants. There is labial bone loss and gingival recession. A comprehensive dental rehabilitative and reconstructive approach will require periodontal evaluation and treatment followed by orthodontic (dental) decompensation and then jaw and intranasal surgery. **A,** Frontal facial and occlusal views before treatment. **B,** Profile facial view and lateral cephalometric radiograph before treatment. **C,** Panorex radiograph before treatment.

• **Figure 6-15** A 16-year-old boy was born with Stickler syndrome, which is a type II collagen mutation. At the time of his birth, the Pierre-Robin sequence was appreciated. The patient underwent repair of the cleft palate before he was 1 year old. He had positive eye findings and required the treatment of a retinal detachment during his teenage years; a small cataract is being followed. He arrived for the evaluation of a jaw deformity and malocclusion, which were characterized by maxillomandibular deficiency with anterior open-bite malocclusion. This was combined with chronic obstructive nasal breathing and a long face growth pattern. Attempted growth modification and camouflage orthodontics earlier in life were ineffective. There was generalized root deficiency throughout the maxillary and mandibular dentition, likely as a result of the collagenopathy. The patient agreed to an orthodontic and surgical approach. Further orthodontic (dental) decompensation was cautiously carried out as a result of the compromised periodontal apparatus. The procedures included maxillary Le Fort I osteotomy in segments (vertical intrusion, horizontal advancement, and arch expansion); bilateral sagittal split ramus osteotomies (horizontal advancement and limited counterclockwise rotation); osseous genioplasty (vertical shortening and horizontal advancement); and septoplasty, inferior turbinate reduction, and nasal floor recontouring. **A,** Frontal views with smile before and after treatment. **B,** Profile views before and after treatment.

Prior to retreatment

Pre surgery

After treatment



• **Figure 6-15, cont'd C,** Occlusal views before retreatment, with orthodontics in progress, and then after treatment. **D,** Articulated dental casts that indicate analytic model planning.

• **Figure 6-16** A woman in her late 20s was born with an isolated cleft palate. She underwent palate repair during infancy, and she later developed a jaw deformity with malocclusion. Attempts to neutralize the occlusion included years of growth modification and orthodontic mechanics with four bicuspid extractions between the ages of 11 and 17 years. The patient was left with generalized labial bone loss and gingival recession, especially of the lower anterior teeth. She presented to this surgeon as an adult with a lifelong history of obstructed nasal breathing. She also had a long face growth pattern that involved the maxilla, the mandible, and the chin and that included excess vertical height and horizontal retrusion of those areas. She underwent periodontic, prosthodontic, orthodontic, surgical, speech pathology, and ear, nose, and throat evaluations, among others. She received periodontal treatment and then orthodontic decompensation. Her surgery included a Le Fort I osteotomy (horizontal advancement and clockwise rotation); bilateral sagittal split ramus osteotomies (horizontal advancement and counterclockwise rotation); osseous genioplasty (vertical reduction and horizontal advancement); and septoplasty, inferior turbinate reduction, and nasal floor recontouring (see Fig. 34-9). **A,** Frontal facial and occlusal views before retreatment. **B,** Profile view and lateral cephalometric radiograph before retreatment.

• **Figure 6-17** A 21-year-old college graduate arrived for surgical evaluation. The history and examination confirmed lifelong nasal obstruction, lip incompetence, and gummy smile. Further inspection confirmed a long face growth pattern with a Class II anterior open-bite malocclusion. The patient had been under an orthodontist's care since the mixed dentition and throughout her high school years. Growth modification, rapid palatal expansion, and full bracketing with four bicuspid extractions introduced dental compensation but without successful closure of the open bite. Early periodontal deterioration is evident. She agreed to a comprehensive dental and facial rehabilitation approach. Periodontal evaluation and treatment followed by orthodontic (dental) decompensation and then jaw and intranasal surgery were carried out (see Figure 21-5). **A,** Frontal view in repose and occlusal views before retreatment. **B,** Profile view and lateral cephalometric radiograph before retreatment.

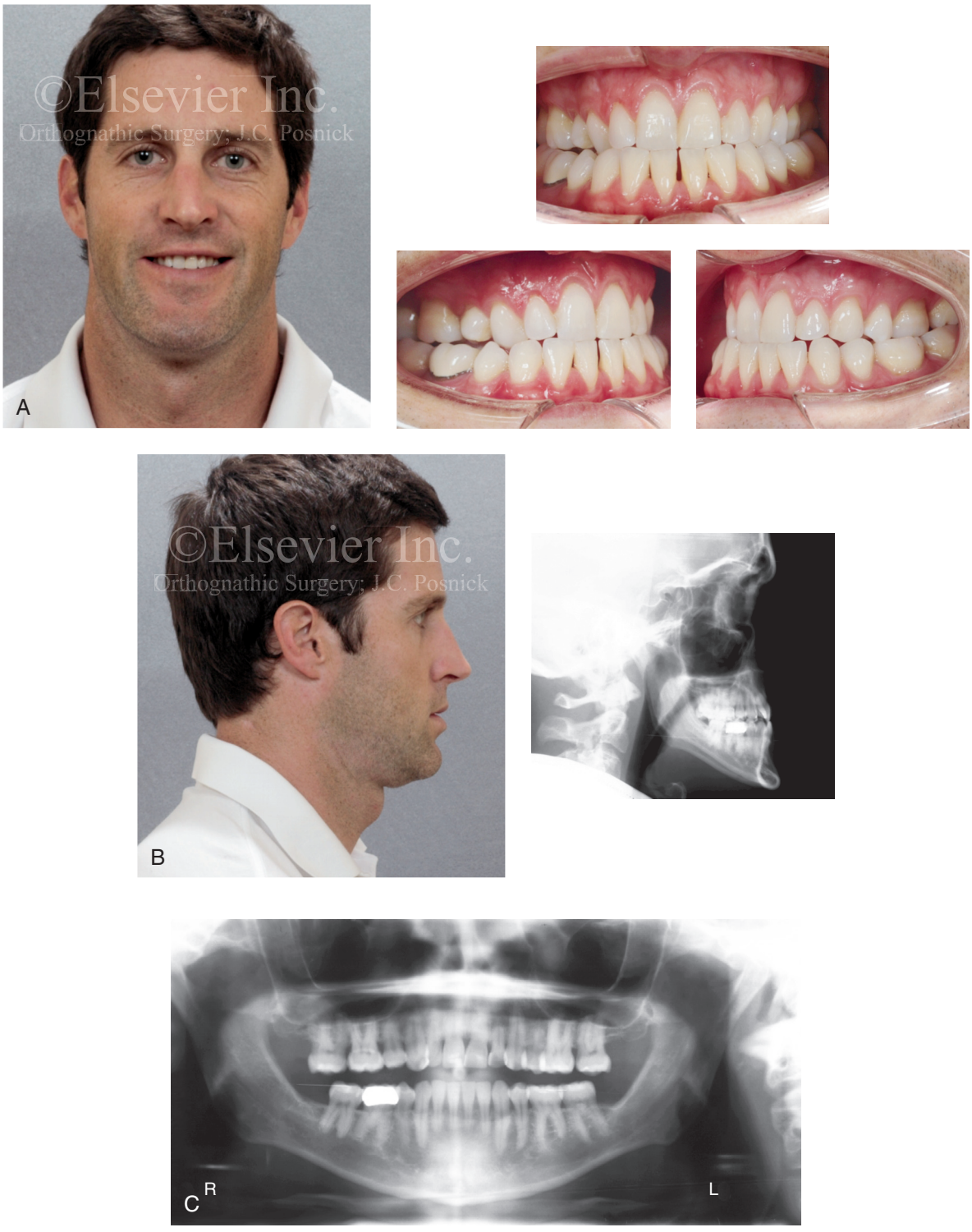
• **Figure 6-18** A woman in her mid 30s was referred by her orthodontist for surgical evaluation. She had a lifelong history of obstructed nasal breathing and a long face growth pattern. Throughout her mid childhood and teenage years, she had undergone attempted growth modification followed by orthodontic camouflage, which included four bicuspid extractions with an attempt to neutralize the occlusion. There is periodontal deterioration and dental relapse with residual malocclusion. The patient agreed to a comprehensive surgical and dental rehabilitative approach. Periodontal treatment was followed by orthodontic (dental) decompensation. Her surgery included maxillary Le Fort I osteotomy in segments (horizontal advancement, vertical shortening, counterclockwise rotation, arch expansion, and curve of Spee correction); bilateral sagittal split ramus osteotomies (horizontal advancement and counterclockwise rotation); osseous genioplasty (vertical reduction and horizontal advancement); and septoplasty, inferior turbinate reduction, and nasal recontouring (see Fig. 25-8). **A,** Frontal facial and occlusal views before retreatment. **B,** Profile view and lateral cephalometric radiograph before retreatment.

• **Figure 6-19** A woman in her late 40s with a long face growth pattern suffered deterioration of the dentition. The periodontal risk factors included inadequate attached gingiva, dental root crowding within limited alveolar bone support, nasal airway obstruction with forced mouth breathing, and lip incompetence that caused drying of the exposed gingiva. An attended polysomnogram confirmed obstructive sleep apnea. Unfavorable soft-tissue envelope distortions and premature facial aging were also discussed. She agreed to a comprehensive surgical and dental rehabilitative approach. Periodontal treatment was followed by restorative temporization and orthodontic alignment, including the extraction of the four first bicuspids. This was followed by surgery that included maxillary Le Fort I osteotomy in segments (vertical shortening, horizontal advancement, transverse widening, and limited clockwise rotation); bilateral sagittal split ramus osteotomies (horizontal advancement and limited counterclockwise rotation); osseous genioplasty (vertical shortening and horizontal advancement); an anterior approach to the soft tissues of the neck (cervical flap elevation, neck defatting, and vertical platysma muscle plication); and septoplasty, inferior turbinate reduction, and nasal floor recontouring (see Fig. 25-3). **A,** Frontal facial and occlusal views before treatment. **B,** Profile view and lateral cephalometric radiograph before treatment.

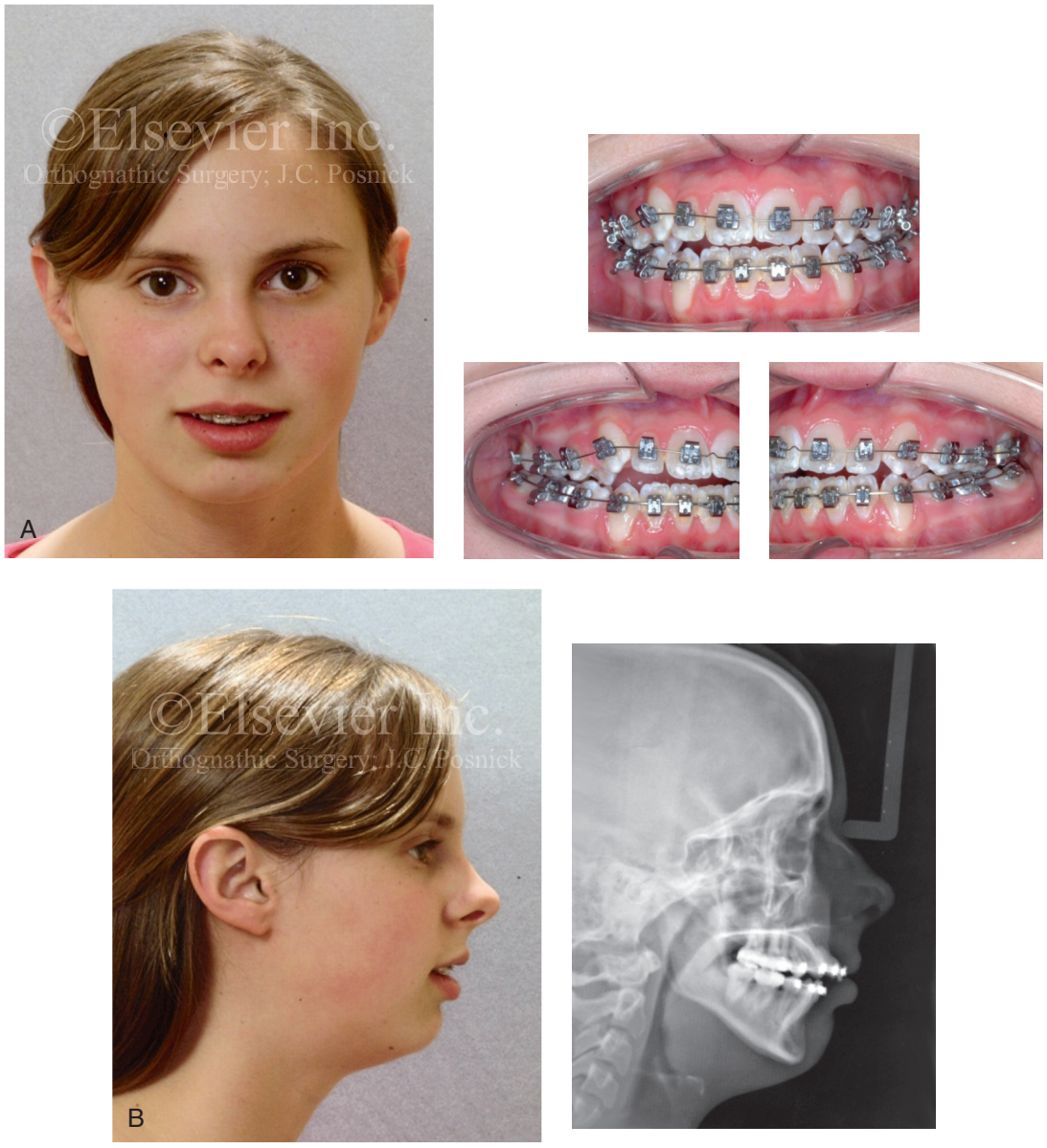
• **Figure 6-20** A 31-year-old man arrived for surgical evaluation. Since the mixed dentition, he was known to have a jaw deformity that was characterized as a maxillary deficiency with relative mandibular excess growth pattern. This results in an Angle Class III, negative overjet, anterior open bite, constricted arch-width malocclusion. The patient is congenitally missing the mandibular first premolars, and he has retained primaries. This surgeon had originally seen him 10 years earlier, when he was 21 years old. An orthodontic and surgical approach was recommended at that time. The patient then declined treatment, but, during the next 10 years, there was further labial bone loss and gingival recession of the mandibular anterior and maxillary posterior dentition. A comprehensive periodontal, orthodontic, and surgical approach was now recommended. The patient had generalized recession, localized minimal attached gingiva, and significant root exposure of teeth nos. 3, 4, 5, 12, 13, and 14. Subepithelial connective tissue grafting was recommended. This is to be followed by orthodontic decompensation and jaw surgery. The procedures are to include Le Fort I osteotomy in three segments (correction of the curve of Spee and the arch width) and bilateral sagittal split ramus osteotomies. **A,** Frontal view with smile and occlusal views at age 21. **B,** Frontal view with smile and occlusal views at age 31. **C,** Profile facial view and maxillofacial i-CAT image that indicate limited alveolar bone covering the roots of the teeth.

• **Figure 6-21** A 32-year-old man arrived for the evaluation of a longstanding jaw deformity with malocclusion, chronic obstructive nasal breathing, and periodontal issues. Since his early childhood years, he was known to have a maxillary deficiency with a relative mandibular excess growth pattern. The family elected a camouflage orthodontic approach in an attempt to neutralize the occlusion when the patient was 9 to 13 years old. He is a forced mouth breather with increased nasal airway resistance as a result of septal deviation and turbinate hypertrophy. His general dentist confirmed labial bone loss and gingival recession of the mandibular anterior and maxillary posterior dentition. He was referred to this surgeon, and a comprehensive orthognathic and dental approach was approved. Consultation with a periodontist, an orthodontist, and an ear, nose, and throat specialist was carried out. The patient was confirmed to have deviation of the septum and enlarged inferior turbinates. An attended polysomnogram is pending. The need for gingival grafting to attain improved root coverage and to generate a wider band of attachment was recommended for teeth nos. 3, 11, 14, 19, 20, 23 through 26, and 29 and 30. This will be followed by the orthodontic removal of dental compensation, including the extraction of a mandibular incisor to address the severe bone loss and gingival recession. Orthognathic and intranasal procedures to improve long-term dental health, to enhance facial aesthetics, and to open the upper airway will follow. **A,** Frontal facial and occlusal views before treatment. **B,** Panorex radiograph with treatment in progress.

• **Figure 6-22** A 30-year-old man arrived for the evaluation of a longstanding jaw deformity with malocclusion, chronic obstructive nasal breathing, and awareness of periodontal issues. Since his early childhood years, he was known to have a jaw deformity with malocclusion that included an anterior open bite. When he was between 8 and 12 years old, he underwent orthodontic camouflage treatment in an attempt to close the open bite, including full bracketing and the use of heavy anterior elastics. By the time he graduated from high school, he was conscious of a significant recurrent anterior open bite with dental crowding. Through his college years, he was aware of gingival recession on the labial aspect of many of the anterior teeth; this was apparent on the maxilla more so than on the mandible. He was sent by his general dentist for an orthodontic evaluation and then to this surgeon for an opinion. He was referred for periodontal evaluation to confirm significant labial bone loss of the anterior dentition (on the maxilla more than the mandible) with gingival recession. A comprehensive approach was recommended, including gingival grafting and four bicuspid extractions with orthodontic retraction of the anterior dentition into solid alveolar bone. This would be followed by jaw reconstruction, finishing orthodontics, and periodontal surveillance. **A,** Frontal facial and occlusal views before retreatment. **B,** Profile facial view and lateral cephalometric radiograph before retreatment. **C,** Panorex radiograph before retreatment.



• **Figure 6-23** A 34-year-old man arrived for the evaluation of a longstanding jaw deformity with malocclusion, chronic obstructive nasal breathing, and periodontal issues. Since his early childhood years, he was known to have a maxillary deficiency with relative mandibular excess growth pattern. The family elected a camouflage orthodontic approach that included four bicuspid extractions and the introduction of dental compensation in an attempt to neutralize the occlusion when the patient was 10 to 15 years old. He is a forced mouth breather as a result of the increased nasal airway resistance. He underwent septoplasty when he was 20 years old, but this did not result in significant improvement. His general dentist confirmed labial bone loss and gingival recession of the mandibular anterior and maxillary posterior dentition. He was referred to this surgeon, and a comprehensive orthognathic and dental approach was recommended. Consultation with the periodontist, the orthodontist, and the ear, nose, and throat specialist was carried out. The patient was confirmed to have residual deviation of the septum and enlarged inferior turbinates, which explained the continued difficulty that he had breathing through his nose. A degree of root resorption of the anterior dentition was likely the result of previous orthodontic mechanics. The need for gingival grafting to attain as much root coverage as possible and to generate a wider band of attachment was recommended for teeth nos. 3, 11, 14, 19, 20, 23 through 26, and 29 and 30. This will be followed by the orthodontic removal of dental compensation and then orthognathic and intranasal procedures to improve the patient's long-term dental health, to enhance his facial aesthetics, and to open his upper airway. **A,** Frontal facial and occlusal views before treatment. **B,** Profile facial view and lateral cephalometric radiograph before treatment. **C,** Panorex radiograph with treatment in progress.



• **Figure 6-24** A 16-year-old girl with a primary mandibular deficiency growth pattern. She had undergone several years of growth modification followed by orthodontic mechanics that included four bicuspid extractions in an attempt to neutralize the occlusion. The patient was left with mandibular deficiency and malocclusion that included significant overjet, anterior open bite, and proclination of the maxillary incisors. Labial bone loss with gingival recession that affected the lower anterior dentition is evident. The patient was referred to this surgeon for a comprehensive orthodontic and surgical approach (see Fig. 19-11). **A,** Frontal and occlusal views in repose at the time of referral. **B,** Profile and lateral cephalometric radiograph views at the time of referral.

• **Figure 6-25** A man in his early 20s with a primary mandibular deficiency growth pattern. He underwent many years of growth modification followed by orthodontic camouflage maneuvers that included four bicuspid extractions in an attempt to neutralize the occlusion. Deterioration of the periodontium with labial bone loss and gingival recession along the mandibular anterior dentition are evident. **A,** Frontal facial and occlusal views before retreatment. **B,** Profile view and lateral cephalometric radiograph before retreatment.

• **Figure 6-26** A woman in her early 50s was referred by a prosthodontist for surgical evaluation. There had been a gradual deterioration of the dentition, which was at least partially a result of the longstanding developmental Class II excess overjet deep-bite skeletal pattern. A head and neck evaluation confirmed a retrusive mandible, which also resulted in retroglossal airway obstruction. In addition, a desire for improved profile aesthetics was discussed. A comprehensive approach to dental rehabilitation, improvement of the upper airway, and facial rejuvenation and reconstruction was recommended. Coordinated endodontic, orthodontic, periodontic, prosthodontic, and surgical care was required. Periodontal treatment, extractions, dental implant placement, restorative temporization, and orthodontic alignment were carried out. These treatments were followed by surgery that included bilateral sagittal split ramus osteotomies (horizontal advancement and counterclockwise rotation); osseous genioplasty (vertical lengthening with interpositional grafting); and an anterior approach to the soft tissues of the neck (cervical flap elevation, neck defatting, and vertical platysma muscle plication). These procedure were followed by crown lengthening and then by final dental restorations (see Fig. 25-2). **A,** Frontal facial and occlusal views before treatment. **B,** Profile facial view and lateral cephalometric radiograph during treatment. **C,** Panorex radiograph before treatment.

• **Figure 6-27** A 50-year-old woman was referred by her general dentist to an orthodontist and then for surgical evaluation. There was a mandibular deficiency and a constricted upper jaw growth pattern. This resulted in a Class II deep-bite excess overjet malocclusion. Over the years, there was destruction to the dentition and periodontal apparatus with generalized bone loss and gingival recession. The patient was now under the care of a restorative dentist and a periodontist. To resolve the malocclusion, maxillary expansion and mandibular advancement in combination with orthodontics were recommended. At referral to this surgeon, a history documented heavy snoring, restless sleeping, and daytime somnolence. An attended polysomnogram confirmed a respiratory disturbance index of 42.7 events per hour. There was intranasal obstruction caused by septal deviation and inferior turbinate enlargement, a retropositioned palate caused by maxillary retrusion, and a retropositioned tongue as a result of mandibular retrusion. The soft palate and the tongue were of normal size and function. There were minimum tonsil and adenoid structures. Continuous positive airway pressure was tried but not well tolerated. Surgery was agreed to in combination with orthodontics to improve the malocclusion and to open the airway. The procedures carried out included a maxillary Le Fort I osteotomy (horizontal advancement, vertical lengthening, and arch form correction); bilateral sagittal split osteotomies of the mandible (horizontal advancement); osseous genioplasty (horizontal advancement); septoplasty and inferior turbinate reduction; and an anterior approach to the neck (cervical flap elevation, neck defatting, and vertical platysma muscle plication). Subjectively, there was good relief of snoring and restless sleeping as well as the resolution of daytime fatigue. Six months after surgery, an attended polysomnogram confirmed the resolution of the obstructive sleep apnea with a respiratory disturbance index of 1.9 events per hour and minimum desaturation (see Fig. 26-8). **A,** Frontal facial and occlusal views before treatment. **B,** Profile facial view and lateral cephalometric radiograph before treatment.

• **Figure 6-28** A 70-year-old woman arrived for surgical evaluation. She desired a stronger profile, less "loose" neck skin, and an improved smile. She initially requested a face lift and a chin implant. She was found to have a retrusive mandible and dental deterioration that was made worse by dental crowding and a deep-bite negative overjet malocclusion. She underwent orthodontics that included four bicuspid extractions and then surgery that included sagittal split ramus osteotomies (horizontal advancement) and osseous genioplasty (horizontal advancement) (see Fig. 25-10). **A,** Frontal views with smile before and after treatment. **B,** Oblique facial views before and after treatment.

Prior to orthodontics

After treatment including four bicuspid extractions

After treatment

• **Figure 6-28, cont'd C,** Occlusal views before treatment, with orthodontics in progress (including four bicuspid extractions), and after treatment.

• **Figure 6-29** Frontal facial and occlusal views of a 16-year-old boy who had sustained a fracture of the right condyle of the mandible early during his childhood and who presented with the resulting facial asymmetry. He previously underwent several years of orthodontics to neutralize the occlusion. There was loss of labial bone along the anterior mandibular teeth. After surgical evaluation, the patient agreed to a comprehensive orthodontic and orthognathic approach. The procedures included a Le Fort I osteotomy, a left sagittal split ramus osteotomy, the reconstruction of the right condyle with a costochondral graft, and an osseous genioplasty (see Fig. 35-8). Note the recession along the labial aspects of the mandibular incisors before surgery. Gingival grafting is indicated. *From Posnick JC: Management of facial fractures in children and adolescents.* Ann Plast Surg *33:453, 1994.*

Effects of Inadequate Attached Gingiva

There are significant intrinsic biologic variations between humans with respect to the morphologic characteristics of the gingiva; this is known as the *gingival biotype.*[52,73,138] The evaluation of the individual's gingival biotype is important to orthodontic treatment planning, because thick and thin gingival biotypes are frequently associated with varied osseous patterns. These two tissue types are likely to respond very differently to similar orthodontic forces by demonstrating different patterns of osseous remodeling. With an understanding of the nature of the individual's biologic risk factors (e.g., gingiva, alveolar bone, breathing pattern, degree of malocclusion), appropriate periodontal, orthodontic, and surgical preventative procedures and precautionary measures may be instituted to provide a more favorable tissue environment to minimize alveolar bone loss and gingival recession. Historically, Ochsenbein and Maynard discussed the importance of thick versus thin gingiva with regard to restorative treatment planning.[150] In addition, in a group of patients reviewed by Olsson and colleagues, a thick periodontal biotype (85% of population) was found to be more prevalent than a thin periodontal biotype (15% of population).[152] Thick gingival tissue is dense in appearance, with a fairly large zone (length) of attachment. The gingival topography is relatively flat, which is suggestive of a full underlying bony architecture. Thin gingival tissue tends to be delicate and almost translucent in appearance. The tissue is friable, with a minimal zone of attachment; this is suggestive of minimal bone over the labial roots of the teeth. The diametrically opposite thin and thick gingival biotypes will respond differently when subjected to inflammation, mechanical trauma, orthodontic forces, or surgical insults. Results from at least some clinical studies indicate that, as long as a tooth is being orthodontically moved with light forces and within or into (and not out of) the alveolar process, then the risk of harmful side effects on the marginal (gingival) soft tissues is minimal.[212-215] In current clinical practice, pretreatment gingival augmentation (i.e., free gingival grafts, lateral pedicled flaps, and autogenous and allogenic connective tissue grafts) to improve the presenting thin keratinized gingiva—in combination with light orthodontic force—generally achieves objectives and limits recession and alveolar bone loss.

Effects of Inadequate Alveolar Bone (Alveolar Bone/Dental Root Ratio)

There will be inherent biologic variations among individuals with regard to the extent of alveolar housing as compared with the cumulative dental root volume of the compliment of teeth within each arch; this is known as the *alveolar bone/dental root ratio.*[184,199] When the roots are crowded into

limited available alveolar housing, the periodontal tissues are less able to withstand the routine inflammatory processes and the mechanical demands that they experience. In turn, the periodontium may recede (i.e., experience bone loss and gingival recession). To prevent this, either the space within the arch must be increased (e.g., rapid palatal expansion, surgically assisted rapid palatal expansion, segmental osteotomies) or extractions must be carried out as part of orthodontic treatment to limit the risk of periodontal deterioration.[50,58,101,103,114,119,147,168,217] Orthodontic relief of dental crowding in the upper jaw by simply expanding the crown arch form in the presence of an inadequate alveolar arch form may align the dental crowns, but it will predictably result in cortical plate thinning, with the potential for dehiscence, fenestrations, loss of alveolar ridge height, and gingival recession (Fig. 6-8). Under these circumstances, successful dental uncrowding without extractions requires the initial expansion of the maxillary (skeletal) arch (e.g., rapid palatal expansion, surgically assisted rapid palatal expansion, segmental osteotomy).[108] By following these principles, the achievement of improved arch form with the preservation of periodontal health should be possible.[147]

Effects of Nasal Obstruction and Mouth Breathing With Resulting Vertical Maxillary Excess, Lip Incompetence, and Inadequate Incisor/Gingiva Coverage

There is considerable variation among humans with respect to *biologic respiratory patterns* (i.e., mouth breathing versus nasal breathing). In the presence of significant nasal obstruction, a forced mouth-breathing pattern will occur and have the potential to cause detrimental effects on the periodontium.[7,102,113,206] The existence of these two very different airway biotypes (i.e., nasal breathing and mouth breathing) and the secondary effects that may result should be recognized. Mouth breathing is an important etiologic factor for chronic gingivitis and chronic marginal periodontitis as well as for the formation of dental plaque and calculus. The presence of marginal gingivitis in individuals with mouth breathing was described in the literature by **Coleyer** in 1920. **Warwick and Hastings** (1933) also claimed that the surfaces of teeth and gingiva that were constantly exposed to air (i.e., in individuals who breathe habitually through the mouth) did not have the benefit of normal irrigation with saliva; these surfaces also did not benefit from the normal friction effects that result from the vestibular mucosa moving against the teeth and gingiva that would normally occur during lip closure.

In a normal human physiologic state, nasal breathing is a primary source of air intake; it is essential for the supply of properly cleansed, moist, warm air to the lungs. The mouth is considered the secondary emergency source of air for breathing. Individuals who primarily breathe through the mouth also tend to have an excessively open lip posture (i.e., >4 mm), and they often have reduced lip coverage of the incisors and the gingiva. Epidemiologic studies suggest an increased susceptibility to gingival inflammation among mouth breathers. The exact mechanism of these effects is not entirely known, but it is thought that the gingiva of the anterior teeth in particular is prone to drying out as a result of a lack of saliva. This scenario will also lead to an absence of the natural protective action of the saliva and the lips.

Gulati and colleagues undertook a study to assess the effects of mouth breathing, lip seal, and upper lip coverage on the gingival health of children.[98] School-aged children between the ages of 10 and 14 years (n = 240) were selected for the study. After clinical examination, they were divided into two major groups: mouth breathers and normal breathers. These two groups were further subdivided into six subgroups on the basis of lip-seal ability and upper incisor lip coverage. The results of the study indicate a gingival index that is higher in mouth breathers with lip incompetence than in normal breathers. Increased lip separation and decreased upper lip coverage (i.e., excess exposure of the maxillary incisors) were also associated with higher plaque and gingival indices.

Alexander completed clinical research with the intent of understanding habitual mouth breathing and its effect on gingival health.[7-9] The study included 200 patients with a mean age of 30 years who were attending a clinic for routine dental treatment. Each of the subjects underwent systematic examinations to evaluate the presence of gingival inflammation, supragingival and subgingival calculus, and bacterial plaque. Each detailed examination was carried out under standardized conditions. Individuals were questioned carefully regarding the manner in which they usually breathe (i.e., mouth breathing versus nasal breathing). Mean gingival inflammation scores, calculus surface indices, and bacterial indices (with erythrosine disclosing solution) were calculated for each of their individual teeth. The study results confirm that habitual mouth breathing is highly associated with an increase in gingival inflammation and the prevalence of supragingival and subgingival calculus (see Figs. 6-15 through 6-22).

Fukuda and colleagues completed a study to evaluate the influence of mouth breathing on the periodontal tissues.[76] Ten patients with chronic marginal periodontitis who were found to be habitual mouth breathers were included in the study, and they were evaluated for 30 days. During the experimental time frame, the study patients underwent taping of the lips to accomplish lip seal and to limit the mouth breathing option while sleeping. Oral examinations were completed and biopsy specimens of the gingiva were taken before and after nighttime lip tapings. The study indicates that, with satisfactory lip seal (i.e., lip taping), forced primary nasal breathing was achieved in these individuals while they were sleeping. This resulted in the following:

1. Halitosis and the uncomfortable feeling in the mouth upon awakening were decreased significantly.
2. The depth of the periodontal pockets and the labiolingual width of the gingiva were both decreased. The improvement was primarily seen on the labial side of the maxillary dentition.

3. The vacuolar degeneration of the epithelial cells was decreased as confirmed by histopathologic examination. However, inflammation of the connective tissue did not show improvement during the 30-day treatment time.

Effects of Malocclusion on Periodontal Health

Bollen completed a systematic review of the literature to answer the question, "Does a malocclusion affect periodontal health?"[35] An electronic search included all publications from 1960 through 2006. A total of 25 studies that evaluated the association between a malocclusion and periodontal health were of sufficient quality for inclusion in the review.* Only 5 of these 25 studies adjusted for factors such as age, socioeconomic status, and oral hygiene. The total number of subjects included in the 25 studies was 35,300, and they had a mean age of 22 years (range, 3 to 60 years). Pertinent results of the review included the following:

1. Nineteen of the 25 studies found greater periodontal disease among subjects with greater malocclusion.
2. Six studies considered the interaction between malocclusion and "periodontopathies." These studies found significantly greater periodontal problems among subjects with malocclusions as compared with subjects without malocclusions ($P < .0001$).
3. Two studies looked at the interaction between malocclusion and gingivitis. These studies found greater levels of gingivitis among subjects with malocclusions as compared with subjects without malocclusions ($P < .0001$).

The conclusions drawn by Bollen suggest that there is often a correlation between the presence of malocclusion and periodontal disease. However, the extent of this correlation, which is found in large population studies, should not be interpreted as demonstrating a clear cause and effect for any given individual.[4,5]

Potential Detrimental Effects of Orthodontic "Camouflage" Maneuvers on the Periodontium

Orthodontic camouflage maneuvers and dental restorative procedures carried out in an attempt to neutralize the malocclusion may be additive to the individual's biologic risk factors with regard to his or her potential for progressive deterioration of the periodontium (see Figs. 6-16 through 6-25).

*References 23, 28, 32, 33, 44-46, 53, 55, 64, 65, 72, 77, 79-86, 95, 96, 105, 109, 110, 112, 118, 120, 123, 137, 143, 145, 149, 163, 187, 188-190, 196, 201, 208

Long Face Growth Pattern

In the individual with a *long face growth pattern,* orthodontic camouflage maneuvers may attempt to correct maxillary arch–width deficiency via the buccal tipping of the posterior teeth (see Figs. 6-15 through Fig 6-19). If more than a few millimeters are required, this may result in the remodeling of the alveolar process with dehiscence and fenestration of the buccal cortex, followed by gingival recession.[27,205,207,209]

Orthodontic mechanics that are carried out in an attempt to manage the skeletal component of an anterior open bite often "extrude" the incisors. A biologic consequence may be the remodeling of the alveolar process with dehiscence and fenestration of the mandibular labial cortex, followed by gingival recession. Maxillary incisor root resorption may also occur.

Maxillary Deficiency With Relative Mandibular Excess Growth Pattern

With a *maxillary deficient and relative mandibular excess growth pattern,* orthodontic camouflage maneuvers often attempt to correct upper arch–width deficiency via the buccal tipping of the posterior teeth (see Figs. 6-14 and 6-20 through 6-23). If more than a few millimeters are required, this may result in the remodeling of the alveolar process with dehiscence and fenestration of the buccal cortex, followed by gingival recession.[27,205,207,209]

Orthodontic mechanics that are carried out to manage the skeletal component of the negative overjet often result in the proclination of the maxillary incisors and retroclined mandibular anterior teeth. This approach may result in the remodeling of the alveolar process with dehiscence and fenestration of the mandibular labial cortex, followed by gingival recession. The remodeling of the alveolar process with a loss of maxillary labial cortex may also be seen.[185,194,210]

Primary Mandibular Deficiency Growth Pattern

In the individual who presents with a *primary mandibular deficiency growth pattern,* orthodontic camouflage maneuvers often attempt to uncrowd the mandibular arch by "flaring" the anterior dentition and to then decrease the overjet by proclining the incisors even further (see Figs. 6-24 through Fig. 6-28). This may result in the remodeling of the alveolar process with dehiscence and fenestrations of the labial cortex, followed by gingival recession.[10-15,18,26,64,65,72,178,195,211]

Another common error occurs when mandibular advancement surgery is planned to correct the excess overjet. Often an inter–arch-width discrepancy will be unmasked (i.e., in approximately 30% of cases). It is not uncommon for clinicians to then avoid maxillary surgery through the process of orthodontic buccal tipping the maxillary posterior teeth to relieve the crossbites. If more than a few

millimeters are required, this may result in remodeling of the alveolar process with dehiscence and fenestration of the buccal cortex followed by gingival recession[180] (see Figs. 6-24 through 6-28).

Establishing a Healthy Periodontium and Periodontal Therapeutics

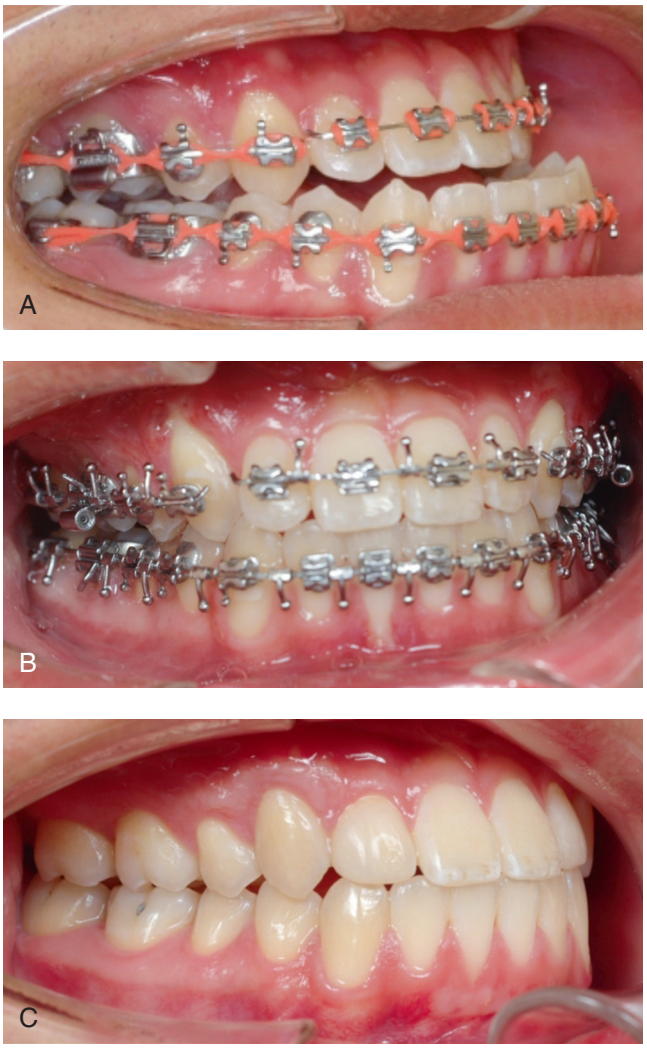
Before the initiation of orthodontic treatment, the clinician should assess for adequate attached gingiva (i.e., the need for grafting); adequate alveolar bone (i.e., the need for extractions or alveolar expansion); caries and plaque control (i.e., the need for hygiene instruction, deep scaling, and root planing); the adequacy of dental restorations (i.e., the need for restorative dentistry); the adequacy of nasal breathing (i.e., the need to open the airway through intranasal surgery); and the adequacy of lip closure (i.e., the need for jaw surgery).[16,17] When a jaw discrepancy is present (with or without combined nasal obstruction and lip incompetence), the orthodontic objectives should be modified to include the coordination of surgical treatment to achieve the desired end results.

A primary focus of *periodontal therapeutics* is to reduce inflammation by creating an environment that can be maintained as free of plaque as possible.[2,3,22,87,136,159,164,166] After this is accomplished, the individual can be trained to perform the task of deplaquing the dentition and developing an effective habit of doing so at least once every 24 hours.[179,186] Plaque-retaining traps will limit the individual's ability to effectively deplaque the dentition.[91] These traps include calculus, crowded teeth, malaligned teeth, restorative overhangs, overcontoured teeth, root anomalies, subgingival caries, and pocketing.[97] Consultation with a periodontist is useful, because his or her skills include the ability to diagnose and remove plaque traps and also to effectively educate the affected individual with regard to how best to maintain clean teeth.[130] Limiting plaque traps may also require orthodontic procedures to reposition dental crowns into more healthy relationships and to place roots more fully within the alveolar bone. This may be combined with procedures to surgically align the jaws (e.g., orthognathic correction) and to open the nasal airway (e.g., septoplasty, inferior turbinate reduction).[48,51,71,74,75,78,88,90,94,140,158,162,167,168] Misguided orthodontic tooth movement carried out in isolation or in combination with surgery can result in injuries that includes the loss of alveolar crestal height, the dehiscence of or fenestrations through the labial and lingual plates, gingival recession, root resorption, tooth mobility, and inadequate occlusion (see Figs. 6-29 and 6-30).[42,60,61,127,128,146,148,153]

Adult Orthodontic Treatment

Throughout the more advanced countries in the world, there has been a continuous increase in the number of adults who are seeking orthodontic treatment. There is a growing desire to maintain the dentition throughout life and an awareness of the importance of a comprehensive dental rehabilitative approach to do so. The orthodontic movement of teeth and the level of orthodontic force applied have potential effects on the surrounding alveolar bone, the PDLs, and the gingival attachments.

With aging, there tends to be progressive reduction of the periodontium, an increase in the root–tooth ratio, and a decrease in the resistance to spontaneous tooth migration. For these reasons, a reduced baseline periodontium may be



• **Figure 6-30** A 17-year-old high school student was referred by his orthodontist for surgical evaluation. The Class III negative overjet anterior open-bite malocclusion required retreatment with orthodontics, including maxillary bicuspid extractions and jaw surgery. With a lifelong history of nasal obstruction and consistent physical findings, intranasal procedures were also required. With orthodontic (dental) decompensation complete, the patient's surgery included a maxillary Le Fort I osteotomy in segments (arch expansion, horizontal advancement, and vertical lengthening); sagittal split ramus osteotomies (limited corrections); osseous genioplasty (vertical reduction and minimal horizontal advancement); and septoplasty and inferior turbinate reduction. Occlusal views **A,** before retreatment, **B,** with orthodontics in progress, and **C,** after the completion of treatment. The patient required and underwent gingival grafting on the labial aspect of the right maxillary canine after the removal of the orthodontic appliances.

present more frequently in adults than in adolescents. These periodontal findings must be taken into consideration before the initiation of an orthodontic program. With age, there may also be an increase in bone porosity (i.e., decreased density). The initial reaction of the PDL and the alveolar bone to orthodontic loading in the adult may be delayed. However, after tooth displacement gets under way, treatment can be carried out in the same way in the adult as it is in a teenager or a young adult. There is no known age limit to the orthodontic movement of teeth (see Fig. 6-28). By contrast, it is known that the precondition of the periodontium should be one of health and anatomic adequacy to mitigate the risks to the periodontium during orthodontic therapy. The presenting periodontium—whether normal or diminished—should be healthy when tooth movement is initiated.

Conclusions

The periodontium includes the investing and supporting tissues of the teeth, and it consists of the attachment apparatus and the dentogingival unit. The degeneration of the periodontium is likely to accelerate in the presence of specific intrinsic biologic factors (i.e., the individual's biotype), which may include a baseline jaw discrepancy with malocclusion; the crowding of the dental roots within limited alveolar bone; a forced mouth-breathing pattern; and an inadequate amount of attached gingiva at the clinical crown interface of the teeth. Active infection or inflammation of the periodontium, non-biologic restorations at the dental–gingival interface, para-occlusal habits, local toxins, and poor oral hygiene also negatively affect the periodontium.

Before the initiation of orthodontic treatment, the clinician should assess for the patient's specific risk factors for periodontal disease and address them. When a jaw discrepancy is present, the orthodontic objectives should be modified to include the coordination of surgical treatment to best preserve the periodontium and to achieve the desired end result.

References

1. Addy M, Dummer PMH, Griffiths G, et al: Prevalence of plaque gingivitis and caries in 11-12-year-old children in South Wales. *Community Dent Oral Epidemiol* 14:115–118, 1986.
2. Addy M: Plaque control as a scientific basis for the prevention of caries. *J R Soc Med* 79(Suppl):6–9, 1986.
3. Addy M, Griffiths G, Dummer PMH, et al: The distribution of plaque and gingivitis and the influence of toothbrushing hand in a group of 11-12-year-old school children. *J Clin Periodontol* 14:564–572, 1987.
4. Addy M, Griffiths GS, Duramer PMH, et al: The association between tooth irregularity and plaque accumulation, gingivitis, and caries in 11-12-year-old children. *Eur J Orthod* 10:76–83, 1988.
5. Ainamo J: Relationship between malalignment of the teeth and periodontal disease. *Scand J Dent Res* 80:104–110, 1972.
6. Albander JM: A 6-year study on the pattern of periodontal disease progression. *J Clin Periodontol* 17:467–471, 1990.
7. Alexander AG: Habitual mouth breathing and its effect on gingival health. *Paradontologie* 24:49–55, 1970.
8. Alexander AG, Tipnis AK: The effect of irregularity of the teeth and the degree of overbite and overjet on gingival health. *Br Dent J* 128:539–544, 1970.
9. Alexander AG: The effect of lack of function of teeth on gingival health, plaque and calculus accumulation. *J Periodontol* 41:438, 1970.
10. Allais D, Melsen B: Does labial movement of lower incisors influence the level of the gingival margin? A case-control study of adult orthodontic patients. *Eur J Orthod* 25(4):343–352, 2003.
11. Alstad S, Zachrisson BU: Longitudinal study of periodontal condition associated with orthodontic treatment in adolescents. *Am J Orthod* 76:277–286, 1979.
12. Andlin-Sobocki A, Bodin L: Dimensional alterations of the gingiva related to changes of facial/lingual tooth position in permanent anterior teeth of children: a 2-year longitudinal study. *J Clin Periodontol* 20:219–224, 1993.
13. Andlin-Sobocki A, Persson M: The association between spontaneous reversal of gingival recession in mandibular incisors and dentofacial changes in children: a 3-year longitudinal study. *Eur J Orthod* 16:229–239, 1994.
14. Årtun J, Osterberg SK, Kokich VG: Long-term effect of thin interdental alveolar bone on periodontal health after orthodontic treatment. *J Periodontol* 57:341–346, 1986.
15. Årtun J, Krogstad O: Periodontal status of mandibular incisors following excessive proclination. A study in adults with surgically treated mandibular prognathism. *Am J Orthod Dentofacial Orthop* 91:225–232, 1987.
16. Årtun J, Osterberg SK: Periodontal status of teeth facing extraction sites long-term after orthodontic treatment. *J Periodontol* 58(1):24–29, 1987.
17. Årtun J, Urbye KS: The effect of orthodontic treatment on periodontal bone support in patients with advanced loss of marginal periodontium. *Am J Orthod Dentofacial Orthop* 93(2):143–148, 1988.
18. Årtun J, Grobety D: Periodontal status of mandibular incisors after pronounced orthodontic advancement during adolescence: a follow-up evaluation. *Am J Orthod Dentofacial Orthop* 119:2–10, 2001.
19. Asikainen S, Jousimies-Somer H, Kanervo A, Summanen P: Certain bacterial species and morphotypes in localized juvenile periodontitis and in matched controls. *J Periodontol* 58:224–230, 1987.
20. Atherton JD, Kerr NW: Effect of orthodontic tooth movement upon the gingiva. *Br Dent J* 124:555–560, 1968.
21. Atherton JD: Gingival response to orthodontic tooth movement. *Am J Orthod* 58:179–186, 1970.
22. Axelsson P, Lindhe J, Nystrom B: On the prevention of caries and periodontal disease: results of a 15-year longitudinal study in adults. *J Clin Periodontol* 18(3):182–189, 1991.
23. Badzian-Kobos K, Stepien-Szczepanska J, Strzelecka E: Oral hygiene, condition of periodontium, and malocclusion in children aged 7-15 years from a region of mining and power production. *Czas Stomatol* 40:309–315, 1987.
24. Baer PN, Cocarro PJ: Gingival enlargement coincident with orthodontic therapy. *J Periodontol* 34:436–439, 1964.
25. Baker DL, Seymour GJ: The possible pathogenesis of gingival recession: a histological study of induced recession in the rat. *J Clin Periodontol* 3:208–219, 1976.
26. Beagrie CS, Thompson, GW, Basu MK: Tooth position and anterior bone loss in skulls. *Br Dent J* 129:471–474, 1970.
27. Beagrie GS, James GA: The association of posterior tooth irregularity and periodontal disease. *Br Dent J* 113:239–243, 1962.

28. Behlfelt K, Ericsson L, Jacobson L, Linder-Aronson S: The occurrence of plaque and gingivitis and its relationship to tooth alignment within the dental arches. *J Clin Periodontol* 8:329–337, 1981.
29. Bergström J, Eliasson S, Preber H: Cigarette smoking and periodontal bone loss. *J Periodontol* 62:242–246, 1991.
30. Bergström J, Preber H: Tobacco use as a risk factor. *J Periodontol* 65:545–550, 1994.
31. Bernimoulin JP, Curiloviec Z: Gingival recession and tooth mobility. *J Clin Periodontol* 4(2):107–114, 1977.
32. Bibb CA: Occlusal evaluation and therapy in the management of periodontal disease. In Newman MG, Takei HH, Carranza FA, editors: *Carranza's clinical periodontology*, ed 9, Philadelphia, 2002, W.B. Saunders Company, pp 698–701.
33. Bilimoria KF: Malocclusion: its role in the causation of periodontal disease. *J All India Dent Assoc* 35:293–300, 1963.
34. Bolin A, Eklund G, Frithiof L, Lavstedt S: The effect of changed smoking habits on marginal alveolar bone loss: a longitudinal study. *Swed Dent J* 17(5):211–216, 1993.
35. Bollen AM, Cunha-Cruz J, Bakko DW, et al: The effects of orthodontic therapy on periodontal health: a systematic review of controlled evidence. *J Am Dent Assoc* 139(4):413–422, 2008.
36. Bondemark L: Interdental bone changes after orthodontic treatment: a 5-year longitudinal study. *Am J Orthod Dentofacial Orthop* 114(1):25–31, 1998.
37. Boyd RL: Are your patients healthier periodontically because of orthodontic treatment? *Bull Pac Coast Soc Orthod* 56:54–57, 1984.
38. Boyd RL, Leggott PJ, Quinn RS, et al: Periodontal implications of orthodontic treatment in adults with reduced or normal periodontal tissues versus those of adolescents. *Am J Orthod Dentofacial Orthop* 96(3):191–198, 1989.
39. Boyd RL: Periodontal screening examination guide. In *American Association of Orthodontists risk management program*, St. Louis, 1992, American Association of Orthodontists, p 13.
40. Bragd L, Dahlen G, Wikstrom M, Slots J: The capability of *Actinobacillus actinomycetemcomitans, Bacteroides gingivalis* and *Bacteroides intermedius* to indicate progressive periodontitis; a retrospective study. *J Clin Periodontol* 14(2):95–99, 1987.
41. Brägger U, Lang NP: Significance of bone in periodontal disease. *Semin Orthod* 2(1):32–38, 1996.
42. Brezniak N, Wasserstein A: Root resorption after orthodontic treatment: Part 2. Literature review. *Am J Orthod Dentofac Orthop* 103:138–146, 1993.
43. Brown IS: The effect of orthodontic therapy on certain types of periodontal defects: I. Clinical findings. *J Periodontol* 44:742–756, 1973.
44. Buckley LA: The relationship between malocclusion and periodontal disease. *J Periodontol* 43:415–417, 1972.
45. Buckley LA: The relationship between irregular teeth, plaque, calculus and gingival disease. *Br Dent J* 148:67–69, 1980.
46. Buckley LA: The relationship between malocclusion, gingival inflammation, plaque and calculus. *J Periodontol* 52:35–40, 1981.
47. Burch JG, Bagci B, Sabulski D, Landrum C: Periodontal changes in furcations resulting from orthodontic uprighting of mandibular molars. *Quintessence Int* 23:509–513, 1992.
48. Burke JL, Provencher RF, McKean TW: Small segmental and unitooth ostectomies to correct dentoalveolar deformities. *J Oral Surg* 35:453, 1977.
49. Burkett LW: Effects of orthodontic treatment on the soft periodontal tissues. *Am J Orthod* 49:671, 1963.
50. Carroll WJ, Haug RH, Bissada NF, et al: The effects of the Le Fort I osteotomy on the periodontium. *J Oral Maxillofac Surg* 50:128–132, 1992.
51. Clarke NG, Hirsch RS: Personal risk factors for generalized periodontitis. *J Clin Periodontol* 22(2):136–145, 1995.
52. Coatoam GW, Behrents RG, Bissada NF: The width of keratinized gingiva during orthodontic treatment: its significance and impact on periodontal status. *J Periodontol* 52:307–313, 1981.
53. Cohen W: A study of occlusal interferences in orthodontically treated occlusions and untreated normal occlusions. *Am J Orthod* 51:647–689, 1965.
54. Cooper MB, Landay MA, Seltzer S: The effect of excessive occlusal forces on the pulp. II. Heavier and longer term forces. *J Periodontol* 42:353–359, 1971.
55. Davies TM, Shaw WC, Addy M, Dummer PH: The relationship of anterior overjet to plaque and gingivitis in children. *Am J Orthod Dentofacial Orthop* 93:303–309, 1988.
56. Davies TM, Shaw WC, Worthington HV, et al: The effect of orthodontic treatment on plaque and gingivitis. *Am J Orthod Dentofacial Orthop* 99(2):155–161, 1991.
57. Diamanti-Kipioti A, Gusberti FA, Lang NP: Clinical and microbiological effects of fixed orthodontic appliances. *J Clin Periodontol* 14:326–333, 1987.
58. Diedrich PR: Guided tissue regeneration associated with orthodontic therapy. *Semin Orthod* 2(1):39–45, 1996.
59. Dominiak M, Kalecinska E, Krzyszton E, et al: Correlation between temporomandibular dysfunction, disturbances of occlusion, and gingival recession in a group of youth students. *Bull Group Int Rech Sci Stomatol Odontol* 47:40–46, 2006.
60. Dorfman HS: Mucogingival changes resulting from mandibular incisor tooth movement. *Am J Orthod* 74:286–297, 1978.
61. Dorfman HS, Turvey TA: Alterations in osseous crestal height following interdental osteotomies. *Oral Surg Oral Med Oral Pathol* 48:120–125, 1979.
62. Driel WD, Leeuwen EJ, Hoff JW, et al: Time-dependent mechanical behavior of the periodontal ligament. *Proc Inst Mech Eng H* 214:497–504, 2000.
63. Eliasson LA, Hugoson A, Kurol J, Siwe H: The effects of orthodontic treatment on periodontal tissues in patients with reduced periodontal support. *Eur J Orthod* 4(1):1–9, 1982.
64. Emslie RD: The incisal relationship and periodontal disease. *Paradontologie* 12:15, 1958.
65. Engelking G, Zachrisson BU: Effects of incisor repositioning on monkey periodontium after expansion through the cortical plate. *Am J Orthod* 82:23–32, 1982.
66. Engström C, Granström G, Thilander B: Effect of orthodontic force on periodontal tissue metabolism. A histologic and biochemical study in normal and hypocalcemic young rats. *Am J Orthod Dentofacial Orthop* 93:486–495, 1988.
67. Ericsson I, Thilander B, Lindhe J, Okamoto H: The effect of orthodontic tilting movements on the periodontal tissues of infected and non-infected dentitions in dogs. *J Clin Periodontol* 4:278–293, 1977.
68. Ericsson I, Thilander B, Lindhe J: Periodontal condition after orthodontic tooth movements in the dog. *Angle Orthod* 48:210–218, 1978.
69. Ericsson I, Thilander B: Orthodontic forces and recurrence of periodontal disease. *Am J Orthodont* 74:41, 1978.
70. Ericsson I, Lindhe J: Recession in sites with inadequate width of the keratinized gingiva: an experimental study in the dog. *J Clin Periodontol* 11(2):95–103, 1984.
71. Feliu JL: Long-term benefits of orthodontic treatment on oral hygiene. *Am J Orthod* 82(6):473–477, 1982.
72. Ferro A, Aronna P: Isolated gingival recession "without cause" localized to the lower incisors in Class II, 1 dental malocclusion: further studies. *Arch Stomatol (Napoli)* 29:1049–1062, 1988.
73. Forsberg A: A clinical study of the periodontal tissues of the upper incisors in two age groups. *Acta Odontologica Scand Suppl* 9(8):1–105, 1951.
74. Foushee DG, Moriarty JD, Simpson DM: Effects of mandibular orthognathic treatment on mucogingival tissue. *J Periodontol* 56:727–733, 1985.
75. Fox ME, Stephens WF, Wolford LM, Deeb ME: Effects of interdental osteotomies on the periodontal and osseous supporting tissues. *Int J Adult Orthodon Orthognath Surg* 6:39–46, 1991.
76. Fukuda T, Yokomichi K, Kato H, Ishikawa J: Studies on the influence of mouth breathing to the periodontal tissue. The effect of oral screen and lip-seal tape on the gingiva in mouth breathers. *Nihon Shishubyo Gakkai Kaishi* 2:280, 1975.

77. Gardner JH: A survey of malocclusions and some aetiological factors on 1,000 Sheffield school children. *Dent Practit* 6:187–201, 1956.
78. Garib DG, Henriques JFC, Janson G, et al: Rapid maxillary expansion—tooth-tissue-borne vs tooth-borne expanders: a CT evaluation of dentoskeletal effects. *Angle Orthod* 75:548–557, 2005.
79. Geiger AM: Occlusal studies in 188 consecutively treated cases of periodontal disease. *Am J Orthod* 48:330–360, 1962.
80. Geiger AM: Occlusion in periodontal disease. *J Periodontol* 36:387–392, 1965.
81. Geiger AM, Wasserman BH, Thompson RH, et al: Relationship of occlusion and periodontal disease: Part I. A system for evaluating periodontal status. *J Periodontol* 42:371–378, 1971.
82. Geiger AM, Wasserman BH, Thompson RH, Turgeon LR: Relationship of occlusion and periodontal disease: Part V. Relation of classification of occlusion to periodontal status and gingival inflammation. *J Periodontol* 43:554–560, 1972.
83. Geiger AM, Wasserman BH, Turgeon LR: Relationship of occlusion and periodontal disease: Part VI. Relation of anterior overjet and overbite to periodontal destruction and gingival inflammation. *J Periodontol* 44:150–157, 1973.
84. Geiger AM, Wasserman BH, Turgeon LR: Relationship of occlusion and periodontal disease: Part VIII. Relationship of crowding and spacing to periodontal destruction and gingival inflammation. *J Periodontol* 45:43–49, 1974.
85. Geiger AM, Wasserman BH: Relationship of occlusion and periodontal disease: Part IX. Incisor inclination and periodontal status. *Angle Orthod* 46:99–110, 1976.
86. Geiger AM, Wasserman BH: Relationship of occlusion and periodontal disease: Part X. Relation of cross-bite to periodontal status. *J Periodontol* 47:785–789, 1977.
87. Genco RJ: Current view of risk factors for periodontal diseases. *J Periodontol* 67(Suppl 10):1041–1049, 1996.
88. Goldberg D, Turley P: Orthodontic space closure of edentulous maxillary first molar area in adults. *Int J Adult Orthodon Orthognath Surg* 4:255–266, 1989.
89. Goldie RS, King GJ: Root resorption and tooth movement in orthodontically treated, calcium-deficient, and lactating rats. *Am J Orthod* 85:424–430, 1984.
90. Goldman HM: The development of physiologic gingival contours by gingivoplasty. *Oral Surg Oral Med Oral Pathol* 3:879–888, 1950.
91. Goldman HM, Cohen DW: *Periodontal therapy*, ed 4, St. Louis, 1968, The C.V. Mosby Company.
92. Gould MSE, Picton DC: The relationship between irregularities of the teeth and periodontal disease: a pilot study. *Br Dent J* 121:20, 1966.
93. Graves DT, Fine D, Teng YT, et al: The use of rodent models to investigate host-bacteria interactions related to periodontal diseases. *J Clin Periodontol* 35:89–105, 2008.
94. Greenbaum KR, Zachrisson BU: The effect of palatal expansion therapy on the periodontal supporting tissues. *Am J Orthod* 81:12–21, 1982.
95. Grewe JM, Chadha JM, Hagan D, Zermeno JA: Oral hygiene and occlusal disharmony in Mexican-American children. *J Periodontal Res* 4:189–192, 1969.
96. Griffiths GS, Addy M: Effects of malalignment of teeth in the anterior segments on plaque accumulation. *J Clin Periodontol* 8:481–490, 1981.
97. Grossi SG, Genco RJ, Machtei EE, et al: Assessment of risk for periodontal disease: risk indicators for alveolar bone loss. *J Periodontol* 66(1):23–29, 1995.
98. Gulati MS, Grewal N, Kaur A: A comparative study of effects of mouth breathing and normal breathing on gingival health in children. *J Indian Soc Pedod Prev Dent* 16(3):72–83, 1998.
99. Haber J: Cigarette smoking: a major risk factor for periodontitis. *Compendium* 15(8):1004–1008, 1994.
100. Hamp SE, Lundstrijm F, Nyman S: Periodontal conditions in adolescents subjected to multiband orthodontic treatment with controlled oral hygiene. *Eur J Orthod* 4:77, 1982.
101. Handelman CS: Nonsurgical rapid maxillary alveolar expansion in adults: a clinical evaluation. *Angle Orthod* 67:291–305, 1997.
102. Hanson ML, Andrianopoulos MV: Tongue thrust, occlusion, and dental health in middle-aged subjects: a pilot study. *Int J Orofacial Myology* 13:3–9, 1987.
103. Harry MR, Sims MR: Root resorption in bicuspid intrusion: a scanning electron microscope study. *Angle Orthod* 52:235, 1982.
104. Heimlich AC: Selective grinding as an aid to orthodontic therapy. *Angle Orthod* 21:76–88, 1951.
105. Helm S, Petersen PE: Causal relation between malocclusion and periodontal health. *Acta Odontol Scand* 47:223–228, 1989.
106. Henrikson PA: Periodontal disease and calcium deficiency: an experimental study in the dog. *Acta Odontol Scand* 26(Suppl 50):1–132, 1968.
107. Hollender L, Rönnerman A, Thilander B: Root resorption, marginal bone support and clinical crown length in orthodontically treated patients. *Eur J Orthod* 2:197–205, 1980.
108. Hom BM, Turley PK: The effects of space closure of the mandibular first molar area in adults. *Am J Orthod Dentofacial Orthop* 85:457–469, 1984.
109. Horup N, Melsen B, Terp S: Relationship between malocclusion and maintenance of teeth. *Community Dent Oral Epidemiol* 15:74–78, 1987.
110. Ingervall B, Jacobsson U, Nyman S: A clinical study of the relationship between crowding of teeth, plaque and gingival condition. *J Clin Periodontol* 4:214–222, 1977.
111. Iyer VS: Reaction of gingiva to orthodontic force: a clinical study. *J Periodontol* 33:26–28, 1962.
112. Jacobson L, Linder-Aronson S: Crowding and gingivitis: a comparison between mouthbreathers and nosebreathers. *Scand J Dent Res* 80:500–504, 1972.
113. Jacobson L: Mouth breathing and gingivitis I. Gingival condition in children with epipharyngeal adenoids. *J Periodontal Res* 3:269–277, 1973.
114. Jager A, Polley J, Mausberg R: Effects of orthodontic expansion of the mandibular arch on the periodontal condition of the posterior teeth. *Dtsch Zahnarztl Z* 45(2):113–115, 1990.
115. Janson G, Bombonatti R, Brandao AG, et al: Comparative radiographic evaluation of the alveolar bone crest after orthodontic treatment. *Am J Orthod Dentofacial Orthop* 124(2):157–164, 2003.
116. Janson M: Gingival and periodontal relationships after orthodontic therapy: a study of class-II patients. *Dtsch Zahnarztl Z* 39(3):254–256, 1984.
117. Jenkins SM, Dummer PM, Addy M: Radiographic evaluation of early periodontal bone loss in adolescents: an overview. *J Clin Periodontol* 19(6):363–366, 1992.
118. Kalamparov K, Gantsev GA, Ershov VN: Relationship between dentomaxillary deformities and periodontal diseases in children. *Stomatologiia (Mosk)* 51:47–50, 1972.
119. Karring T, Numan S, Thilander B, Magnusson I: Bone regeneration in orthodontically produced alveolar bone dehiscences. *J Periodont Res* 17:309–315, 1982.
120. Katz RV: An epidemiologic study of the relationship between various states of occlusion and the pathological conditions of dental caries and periodontal disease. *J Dent Res* 57:433–439, 1978.
121. Kessler M: Interrelationships between orthodontic and periodontics. *Am J Orthod* 70:154–177, 1976.
122. KIoehn JS, Pfeiffer JS: The effect of orthodontic treatment on the periodontium. *Angle Orthodont* 44:127, 1974.
123. Klassman B, Zacher HW: Treatment of a periodontal defect resulting from improper tooth alignment and local factors. *J Periodontol* 40:401, 1969.
124. Kois JC: The restorative-periodontal interface: biological parameters. *Periodontol 2000* 11:29–38, 1996.
125. Kokich VG: Esthetics: the orthodontic-periodontic restorative connection. *Semin Orthod* 2(1):21–30, 1996.
126. Kraus BS, Jordan RE, Abrams L: *Dental anatomy and occlusion*, Baltimore, 1969, Williams & Wilkins Company.

127. Kwon H-J, Pihlstrom B, Waite DE: Effects on the periodontium of vertical bone cutting for segmental osteotomy. *J Oral Maxillofac Surg* 43:952–955, 1985.
128. Langford SR, Sims MR: Root surface resorption, repair, and periodontal attachment following rapid maxillary expansion in man. *Am J Orthod* 81:108–115, 1982.
129. Lau D, Grimths GS, Shaw WC: Reproducibility of an index for recording the alignment of individual teeth. *Br J Orthod* 11:80–84, 1984.
130. Lee H, Silness J: Periodontal disease in pregnancy: I. Prevalence and severity. *Acta Odontol Stand* 21:533–551, 1963.
131. Listgarten MA, Hellden K: Relative distribution of bacteria at clinically health and periodontally diseased sites in humans. *J Clin Periodontol* 5:115–132, 1978.
132. Loe H, Theilade D, Jensen SB: Experimental gingivitis in man. *J Periodontol* 36:177–187, 1965.
133. Loesche WJ: The role of spirochetes in periodontal disease. *Adv Dent Res* 2:275–283, 1988.
134. Lundeen HC, Gibbs CH: *The function of teeth*, Gainesville, Fla, 2005, L and G Publishers.
135. Magnusson I, Marks RG, Clark WB, et al: Clinical, microbiological and immunological characteristics of subjects with "refractory" periodontal disease. *J Clin Periodontol* 18:291–299, 1991.
136. Marshall-Day CD: The epidemiology of periodontal disease. *J Periodontol* 22:13, 1951.
137. Massler M, Sen Savara BS: Relation of gingivitis to dental caries and malocclusion in children 14-17 years of age. *J Periodontol* 22:87–96, 1951.
138. Maynard JG, Ochsenbein LD: Mucogingival problems, prevalence and therapy in children. *J Periodontol* 46:543–552, 1975.
139. McCombie F, Stothard D: Relationships between gingivitis and other dental conditions. *J Can Dent Assoc* 30:506–513, 1964.
140. Melsen B, Agerbaek N, Eriksen J, Terps S: New attachment through periodontal treatment and orthodontic intrusion. *Am J Orthod Dentofacial Orthop* 94(2):104–116, 1988.
141. Melsen B, Agerbaek N, Makenstam G: Intrusion of incisors in adult patients with marginal bone loss. *Am J Orthod Dentofacial Orthop* 96:232–241, 1989.
142. Melsen B: Biological reaction of alveolar bone to orthodontic tooth movement. *Angle Orthod* 69:151–158, 1999.
143. Miller J, Hobson P: The relationship between malocclusion, oral cleanliness, gingival conditions and dental caries in school children. *Br Dent J* 111:43–52, 1961.
144. Motegi E, Nomura M, Miyazaki H, et al: Gingival recession in long-term post-orthodontic patients. *J Dent Res* 81:A372, 2002.
145. Musialowicz KJ: Relation between malocclusion and periodontal diseases in school children from urban and rural areas covered and not covered by planned stomatological care. *Ann Acad Med Stetin* 29:371–385, 1983.
146. Newman WG: Possible etiologic factors in external root resorption. *Am J Orthod* 67:522, 1975.
147. Northway WM, Meade JB, Jr: Surgically assisted rapid maxillary expansion: a comparison of technique, response, and stability. *Angle Orthod* 67:309–320, 1997.
148. Nyman S, Karring T, Bergenholtz G: Bone regeneration in alveolar bone dehiscences produced by jiggling forces. *J Periodontal Res* 17:316–322, 1982.
149. O'Leary TJ, Badell MC, Bloomer RS: Occlusal characteristics and tooth mobility in periodontally healthy young males classified orthodontically. *J Periodontol* 46:553–558, 1975.
150. Ochsenbein C, Maynard JG: The problem of attached gingiva in children. *ASDC J Dent Child* 41:263–272, 1974.
151. Ogaard B: Marginal bone support and tooth lengths in 19-year-olds following orthodontic treatment. *Eur J Orthod* 10(3):180–186, 1988.
152. Olsson M, Lindhe J: Periodontal characteristics in individuals with varying forms of the upper central incisors. *J Clin Periodontol* 118:78–82, 1991.
153. Omnell ML, Tong DC, Thomas T: Periodontal complications following orthognathic surgery and genioplasty in 19-year-old: a case report. *Int J Adult Orthodon Orthognath Surg* 9:133, 1994.
154. Paolantonio M, Festa F, di Placido G, et al: Site-specific subgingival colonization by *Actinobacillus actinomycetemcomitans* in orthodontic patients. *Am J Orthod Dentofacial Orthop* 115(4):423–428, 1999.
155. Papapanou PN, Wennström JL, Gröndahl K: Periodontal status in relation to age and tooth type: a cross-sectional radiographic study. *J Clin Periodontol* 15:463–478, 1988.
156. Parker GR: Transseptal fibers and relapse following bodily retraction of teeth: a histologic study. *Am J Orthod* 61:331–334, 1972.
157. Pearson LE: Gingival heights of lower central incisors orthodontically treated and untreated. *Angle Orthod* 38:337–339, 1968.
158. Pedrazzoli V, Kilian M, Karring T, et al: Effect of surgical and nonsurgical periodontal treatment on periodontal status and subgingival microbiota. *J Clin Periodontol* 18:598–604, 1991.
159. Pfeifer JS: The reaction of alveolar bone to flap procedures in man. *Periodontics* 3:135–140, 1965.
160. Pihlstrom BL, Ramfjord SP: Periodontal effect of nonfunction in monkeys. *J Periodontol* 42:748, 1971.
161. Polson AM: Long-term effect of orthodontic treatment on the periodontium. In McNamara JA, Ribbens KA, editors: *Orthodontic treatment and the periodontium. Monograph 15, Craniofacial Growth Series*, Ann Arbor, Mich, 1984, Center for Human Growth and Development, The University of Michigan, pp 89–100.
162. Pontoriero R, Celenza F, Ricci G, Carvenale G: Rapid extrusion with fiber resection: a combined orthodontic-periodontic treatment modality. *Int J Periodontics Restorative Dent* 7:31–43, 1987.
163. Poulton DR, Aaronson SA: The relationship between occlusion and periodontal status. *Am J Orthod* 47:690–699, 1961.
164. Preber H, Bergström J: The effect of non-surgical treatment on periodontal pockets in smokers and non-smokers. *J Clin Periodontol* 13:319–323, 1985.
165. Preber H, Bergström J: The effect of cigarette smoking on periodontal healing following surgical therapy. *J Clin Periodontol* 17:324–328, 1990.
166. Pritchard JF: Gingivectomy, gingivoplasty, and osseous surgery. *J Periodontol* 32:257–262, 1961.
167. Pritchard JF: The effect of bicuspid extraction orthodontics on the periodontium. Findings in 100 consecutive cases. *J Periodontol* 44:534–542, 1975.
168. Proffit WR, Phillips C, Fields HW, Turvey TA: The effect of orthognathic surgery on occlusal force. *J Oral Maxillofac Surg* 47:457–463, 1989.
169. Reitan K: Continuous bodily tooth movement and its histological significance. *Acta Odontol Stand* 6:115, 1947.
170. Reitan K: Tissue rearrangement during retention of orthodontically rotated teeth. *Angle Orthod* 29:105–113, 1959.
171. Ren Y, Maltha JC, Von den Hoff JW, Kuijpers-Jagtman AM: Age effect on orthodontic tooth movement in rats. *J Dent Res* 82:38–42, 2003.
172. Ren Y, Maltha JC, Kuijpers-Jagtman AM: The rat as a model for orthodontic tooth movement: A critical review and a proposed solution. *Eur J Orthod* 26:483–490, 2004.
173. Ribeiral MBC, Bolognese AM, Feres EJ: Periodontal evaluation after orthodontic treatment. *J Dent Res* 78(5):979, 1999.
174. Roberts WE, Goodwin WC, Jr, Heiner SR: Cellular response to orthodontic force. *Dent Clin North Am* 25:3–17, 1981.
175. Robertson PB, Schultz LD, Levy BM: Occurrence and distribution of interdental gingival clefts following orthodontic movement into bicuspid extraction sites. *J Periodontol* 48:232–235, 1977.
176. Roth RH: Five-year clinical evaluation of the Andrews straight wire appliance. *J Clin Orthod* 10:836–850, 1976.
177. Ruben RP, Frankl SN, Wallace S: The histopathology of periodontal disease in children. *J Periodontol* 42:473, 1971.
178. Ruf S, Hansen K, Pancherz H: Does orthodontic proclination of lower incisors in children and adolescents cause gingival recession? *Am J Orthod Dentofacial Orthop* 114:100–106, 1998.

179. Rugg-Gunn AJ, MacGregor IDM, Edgar WM, Ferguson MW: Toothbrushing behaviour in relation to plaque and gingivitis in adolescent school children. *J Periodontal Res* 14:231–238, 1979.
180. Ryon K: Effects of force, magnitude and direction of tooth movement on different alveolar bone types. *Angle Orthod* 34:244, 1964.
181. Sadowsky C, BeGole EA: Long-term status of temporomandibular joint function and functional occlusion after orthodontic treatment. *Am J Orthod* 78:201–212, 1980.
182. Sadowsky C, BeGole EA: Long-term effects of orthodontic treatment on periodontal health. *Am J Orthod* 80(2):156–172, 1981.
183. Safkan-Seppala B, Ainamo J: Periodontal conditions in insulin-dependent diabetes mellitus. *J Clin Periodontol* 19(1):24–29, 1992.
184. Salonen LW, Frithiof L, Wouters FR, Hellden LB: Marginal alveolar bone height in an adult Swedish population: a radiographic cross-sectional epidemiologic study. *J Clin Periodontol* 18(4):223–232, 1991.
185. Sarikaya S, Haydar B, Ciger S, Ariyurek M: Changes in alveolar bone thickness due to retraction of anterior teeth. *Am J Orthod Dentofacial Orthop* 122:15–26, 2002.
186. Schneider HG, Brendel AK: Oral hygiene study of children under orthodontic treatment. *Stomatol DDR* 31:258–264, 1981.
187. Schneider HG, Mauersberger I, Bruchmann M: Relation between periodontal diseases and malocclusions. *Stomatol DDR* 31:494–500, 1981.
188. Sergl HG, Krause H: Studies on the effects of malocclusions on the periodontium. *Dtsch Zahnarztl Z* 28:149–154, 1973.
189. Shaw WC, Addy M, Ray C: Dental and social effects of malocclusion and effectiveness of orthodontic treatment: a review. *Community Dent Oral Epidemiol* 8:36–45, 1980.
190. Shefter GJ, McFall WT, Jr: Occlusal relationships and periodontal status in human adults. *J Periodontol* 55:368–374, 1984.
191. Shinberg OE, Saakian S, Zapashnik EK: The functional periodontal overload in bite anomalies in adults. *Stomatologiia (Mosk)* Nov-Dec:42–44, 1991.
192. Sjolien T, Zachrisson BW: Periodontal bone support and tooth length in orthodontically treated persons. *Am J Orthod* 64:28–37, 1973.
193. Slots J: Microflora in the healthy gingival sulcus in man. *Scand J Dent Res* 85:247–254, 1977.
194. Sperry TP, Spiedel TM, Isaacson RJ, Worms FW: The role of dental compensations in the orthodontic treatment of mandibular prognathism. *Angle Orthod* 47:293–299, 1977.
195. Steiner GG, Pearson JK, Ainamo J: Changes of the marginal periodontium as a result of labial tooth movement in monkeys. *J Periodontol* 52:314–320, 1981.
196. Sten IB: Tooth malpositions and periodontal pathosis: an evaluation of etiology and considerations in treatment. *J Peridontol* 29:253, 1958.
197. Stuteville OH: Injuries caused by orthodontic appliances and methods of preventing these injuries. *J Am Dent Assoc* 24:1494–1507, 1937.
198. Tad S, Zachrisson BU: Longitudinal study of periodontal condition associated with orthodontic treatment in adolescents. *Am J Orthod* 76:277–286, 1979.
199. Thilander B, Nyman S, Karring T, Magnusson I: Bone regeneration in alveolar bone dehiscences related to orthodontic tooth movements. *Eur J Orthod* 5:105–114, 1983.
200. Thilander B: Infrabony pockets and reduced alveolar bone height in relation to orthodontic therapy. *Semin Orthod* 2(1):55–61, 1996.
201. Thompson RH, Geiger AM, Turgeon LR: Relationship of occlusion and periodontal disease. Part III. Relation of periodontal status to general background characteristics. *J Periodontol* 43:540–546, 1972.
202. Tirk TM, Guzman CA, Nalchajian R: Periodontal tissue response to orthodontic treatment studied by panoramix. *Angle Orthod* 37:94–103, 1967.
203. Trossello VK, Gianelly AA: Orthodontic treatment and periodontal status. *J Periodontol* 50:665–671, 1979.
204. Vanarsdall RL, Corn H: Soft-tissue management of labially positioned unerupted teeth. *Am J Orthod* 72(1):53–64, 1977.
205. Vanarsdall RL, Jr: Periodontal/orthodontic interrelationships. In Graber TM, Vanarsdall RL, editors: *Orthodontics: current principle and techniques*, ed 2, St. Louis, 1994, Mosby, pp 712–749.
206. Wagaiyu EG, Ashley FP: Mouth breathing, lip seal and upper lip coverage and their relationship with gingival inflammation in 11-14 year-old schoolchildren. *J Clin Periodontol* 18:698–702, 1991.
207. Wainwright WM: Faciolingual tooth movement: influence on the root and cortical plate. *Am J Orthod* 65:278, 1973.
208. Wasserman BH, Geiger AM, Turgeon LR: Relationship of occlusion and periodontal disease: Part VII. Mobility. *J Periodontol* 44:572–578, 1973.
209. Watson WG: Expansion and fenestration or dehiscence. *Am J Orthod* 77:330–332, 1980.
210. Wehrbein H, Fuhrmann RA, Diedrich PR: Periodontal conditions after facial root tipping and palatal root torque of incisors. *Am J Orthod Dentofacial Orthop* 106:455–462, 1994.
211. Wehrbein H, Fuhrmann RA, Diedrich PR: Human histologic tissue response after long-term orthodontic tooth movement. *Am J Orthod Dentofacial Orthop* 107(4):360–371, 1995.
212. Wennstrom JL, Lindhe J, Sinclair F, Thilander B: Some periodontal tissue reactions to orthodontic tooth movement in monkeys. *J Clin Periodontol* 14(3):121–129, 1987.
213. Wennström JL: The significance of the width and thickness of the gingiva in orthodontic treatment. *Dtsch Zahnarztl Z* 45:136–141, 1990.
214. Wennström JL, Lindskog-Stokand B, Nyman S, et al: Periodontal tissue response to orthodontic movement of teeth with infrabony pockets. *Am J Orthod Dentofacial Orthop* 103:313–319, 1993.
215. Wennström JL: Mucogingival considerations in orthodontic treatment. *Semin Orthod* 2(1):46–54, 1996.
216. World Health Organization: *Epidemiology, etiology and prevention of periodontal disease*, Geneva, 1978, Report of WHO Scientific Group, Technical Report Series, p 621.
217. Yurkstas A, Curby W: Force analysis of prosthetic appliance during function. *J Prosthet Dent* 3:82–87, 1953.
218. Zachrisson BW, Alnaes L: Periodontal condition in orthodontically treated and untreated children: 1. Loss of attachment, gingival pocket depth and clinical crown heights. *Angle Orthod* 43:402–411, 1973.
219. Zachrisson BW, Alnaes L: Periodontal condition in orthodontically treated and untreated individuals. *Angle Orthod* 44:48–55, 1974.
220. Zachrisson S, Zachrisson B: Gingival condition associated with orthodontic treatment. *Angle Orthod* 42:26–34, 1972.
221. Zambon JJ, Grossi SG, Machtei EE, et al: Cigarette smoking increases the risk for subgingival infection. *J Periodontol* 67(Suppl 10):1050–1054, 1996.

7

Psychosocial Considerations in the Evaluation and Treatment of Dentofacial Deformities

JEFFREY C. POSNICK, DMD, MD

- Psychosocial Aspects to Consider Before Surgery
- Body Dysmorphic Disorder
- Psychological Functioning in the Individual With a Facial Deformity
- Patient and Family Decision for or Against the Correction of a Dentofacial Deformity
- Patient and Family Education Regarding Convalescence After Orthognathic Surgery
- Patient-Centered Assessment of Surgical and Orthodontic Outcomes
- Controversies Surrounding "Successful" Orthognathic Outcomes
- Conclusions

Individuals who present with dentofacial deformities do not comprise a monolithic group. They may arrive for evaluation during childhood, adolescence, early adulthood, or it may be later in life. The etiology of the jaw deformity may be congenital, developmental, or posttraumatic, or it may have occurred after tumor resection that required radiation therapy. A combination of orthodontic treatment and orthognathic surgery—often in conjunction with additional dental rehabilitation—may be required to correct the skeletal deformities, to improve head and neck function (i.e., chewing, breathing, speech, swallowing, lip control), to enhance facial aesthetics, and to restore the dentition.

The initial discussion regarding the need for orthognathic surgery may come as a surprise to the patient and his or her family. This is especially true for the individual whose jaw dysmorphology occurred gradually during facial development, without special attention being drawn to it all of a sudden as would occur from a traumatic event, a birth defect, or after tumor resection.[9,10,37,68,71,80,81,101,103] Although they have typically been aware of the malocclusion and the associated facial characteristics for at least several years, the patient and his or her family generally assume that orthodontics alone or some other minimally invasive approach is all that will be required for treatment.[87] The orthodontist may have already exhausted the patient and his or her family's energy and resources with several years of treatment to "avoid surgery" before finally "giving up." The family may have inaccurate preconceived ideas about the surgery and the patient's expected convalescence. The decision-making role of the parents and the compliance (i.e., the attitude) of the adolescent patient will also be important. Any dysfunctional family interactions involving the mother, the father, the patient, or the patient's siblings, stepparents, or grandparents at this moment of stress (i.e., when a decision is needed) may also come into play. From the adolescent's point of view, social interactions with his or her peers and concerns about the interpretation (by others) of any change in his or her appearance—as well as his or her evolving self-image and identity—will also have an impact on the situation in ways that are ongoing and somewhat unpredictable.

Frequently, the treating clinicians (i.e., the surgeon, the orthodontist, the restorative dentist, and the general physician) judge success or failure according to specific anatomic or numeric benchmarks, such as achieving a Class I occlusion, normative cephalometric radiographic measurements, and successful wound healing. Although patients and their families do consider stable occlusion and a lack of wound healing complications to be important aspects of success, there are likely to be other motives and objectives that cannot be overlooked if patient satisfaction is to be achieved. It is not uncommon for an orthognathic surgical result to be labeled as suboptimal by the surgeon, the orthodontist, or the restorative dentist as a result of an imperfect occlusion, whereas the patient's satisfaction rating will be more

favorable. It is just as likely that the treating clinicians may feel that a successful outcome has been achieved (i.e., satisfactory occlusion and uncomplicated wound healing), whereas the patient may feel that the aesthetic objectives have not been fully met.

Research confirms a correlation between an adolescent's self-reporting of improved appearance ratings and a decrease in social, physical, and psychological problems after successful reconstruction.[58,67,73,78] Patel and Kapp-Simon prospectively studied 70 teenagers who were slated to undergo orthognathic surgery.[69] Before surgery, 78% stated that a functional reason was their primary motivation for undergoing orthognathic surgery. Not surprisingly, before surgery, the adolescents rated their appearance significantly less favorable than their parents did. Two years after surgery, the adolescents reported significantly increased self-confidence, and a majority (58%) felt that they had achieved improvements in their facial appearance as a result of surgery. They stated that improvements in their facial appearance would be a motivating factor for them to undergo similar surgery again. It would appear that, when all is said and done, adolescents—like adults—rate the enhancement of their facial appearance as a high priority when measuring the success or failure of their orthognathic procedures. Experience confirms it is better to openly discuss facial aesthetic aspects before surgery. This encourages the clinician to set realistic objectives, and it forces the patient and the family to confront their outcome expectations in advance.

Both adolescents and adults undergoing orthognathic surgery should be screened to assess emotional baseline functioning as well as psychological readiness for surgery. The pre-surgical mental health screening should clarify any past or current symptoms of depression, anxiety disorders, panic attacks, aggression, drug/alcohol use, eating disorders, school-related problems, family or social (peer) dysfunctional relationships.[4]

Psychosocial Aspects to Consider Before Surgery

Personal Motivation

The individual's personal motivation to seek the correction of a dentofacial deformity is an important factor for clinicians to consider (Figs. 7-1 through 7-5).* In 2000, Rivera and colleagues surveyed 142 patients regarding their reasons for choosing to undergo orthognathic surgery.[86] Seventy-one percent indicated facial aesthetic concerns, 47% stated functional issues, and 28% confirmed temporomandibular disorders as their reasons for seeking treatment. In 2007, Narayanan and colleagues surveyed 50 patients who sought orthognathic surgery and found that, for the majority, facial aesthetics rather than the correction of their occlusions was the primary motivating factor.[59] In 2007, Stirling and colleagues conducted a questionnaire and interview survey of individuals who had undergone orthognathic surgery. The 46 interviewed patients stated that their primary motivations generally included both "improvement of the occlusion and to gain a more normal facial appearance."[97] More recently, Proothi, Drew, and Sachs completed a retrospective survey of 501 individuals who underwent orthognathic surgery to determine why they sought the procedures.[83] The survey group was 43% male and 57% female, and the individuals ranged in age from 12 to 45 years. Seventy-six percent stated that facial appearance was negatively affected by their pretreatment jaw condition, but only 15% claimed that their appearance was their primary motivation for undergoing surgical evaluation. Thirty-six percent stated that their occlusion was the primary motivating force. Interestingly, 33% admitted to having significant speech difficulties, and 15% complained of swallowing problems that resulted from their presenting jaw deformity.

All adolescents more or less struggle with issues related to self-identity, social acceptance, and physical appearance. The added factor of a dentofacial deformity with impaired head and neck function and negative effects on physical appearance—coupled with the need to make a decision for or against surgical correction—may compound these psychological issues. If the individual already has baseline depression, anxiety, poor self-confidence, or other social issues, the presence of a dentofacial deformity with the need for surgical correction is likely to exacerbate these symptoms. The extent to which an exacerbation of baseline symptoms occurs may depend on how much the lack of self-confidence and social problems relate to the dentofacial deformity.

A typical sequence of events is a pre-adolescent individual being referred by his or her general dentist to an orthodontist for the correction of a malocclusion. The orthodontist may have then attempted growth-modification techniques followed by a camouflage approach to "improve" the occlusion for a period of time that ranges from 2 to 8 years before finally "giving up" and suggesting a surgical referral. The adolescent and his or her family may also have pushed for a surgical referral in response to concerns about facial aesthetics and a lack of progress with regard to the full correction of the malocclusion by the orthodontist who initially felt that he or she could solve the problem with orthodontics alone. With today's evidence-based thinking, knowledgeable clinicians should recognize the need for a surgical approach from the outset.

The adolescent and his or her parents' motivation for correction may be clearly stated to the surgeon at the initial introduction, or it may be only indirectly discussed. The patients' and their families' perceptions of the surgical outcomes will be important to achieving a smooth recovery and a favorable outcome. The results—both functional and aesthetic—should be clearly and realistically articulated by the surgeon, openly discussed, and agreed to by all parties before proceeding.

*References 2, 3, 6, 12, 25, 26, 34, 42, 46, 61, 63, 64, 75, 76, 83, 86, 97, 102, 104, 105

Text continued on p. 218

• **Figure 7-1** A 17-year-old boy who was born with a nonprogressive congenital myopathy was referred by an orthodontist for surgical evaluation. The lack of masticatory muscle strength resulted in a constant open mouth posture. The long face growth pattern is characterized by vertical maxillary excess; a clockwise-rotated retrusive mandible; a vertically long and retrusive chin; and an Angle Class II anterior open-bite malocclusion. The patient underwent a combined orthodontic and surgical approach. Four bicuspid extractions relieved dental crowding. This was followed by surgery that included a maxillary Le Fort I osteotomy (vertical intrusion and horizontal advancement); bilateral sagittal split osteotomies of the mandible (horizontal advancement and counterclockwise rotation); and osseous genioplasty (vertical reduction and horizontal advancement). **A,** Profile views before and after reconstruction. **B,** Frontal views with smile before and after reconstruction.

• **Figure 7-2** A 15-year-old girl was referred by her orthodontist for surgical evaluation. She had a long face growth pattern and a Class II anterior open-bite malocclusion. She had been treated for several years with orthodontics alone, and this included four bicuspid extractions. The anterior open bite was essentially closed, with only minor degrees of malocclusion. The history and physical examination confirmed lifelong nasal obstruction, heavy snoring, and the suggestion of sleep apnea. Facial aesthetic concerns of the patient and her family included an awareness of a gummy smile, severe lip strain, a weak profile, and an irregular shape and position of the lips. The family agreed to an orthognathic approach. The procedures performed included a maxillary Le Fort I osteotomy (vertical shortening and horizontal advancement with counterclockwise rotation); bilateral sagittal split osteotomies (horizontal advancement and counterclockwise rotation); osseous genioplasty (horizontal advancement and vertical shortening); and septoplasty, inferior turbinate reduction, and nasal floor recontouring. **A,** Oblique facial views before and after reconstruction. **B,** Frontal views with smile before and after reconstruction.

NOTE: Improved sleep at night resulted in less daytime fatigue and better concentration at school. Positive psychosocial effects were appreciated and easily adjusted to by the patient.

• **Figure 7-3** A 16-year-old girl was referred by her orthodontist for surgical evaluation. She had a long face growth pattern in combination with asymmetrical mandibular deficiency. This resulted in difficulty with speech articulation, swallowing, chewing, and lip closure/posture. Facial aesthetic awareness of a gummy smile, lip incompetence, a weak profile, and lip irregularities was discussed. With orthodontic decompensation complete, orthognathic surgery was carried out. The procedures performed included a maxillary Le Fort I osteotomy in three segments, with first bicuspid extractions (vertical retrusion, arch form correction, and counterclockwise rotation); bilateral sagittal split ramus osteotomies (horizontal advancement, counterclockwise rotation, and asymmetry correction); and septoplasty, inferior turbinate reduction, and nasal floor contouring. **A,** Profile views before and after treatment. **B,** Frontal views with smile before and after treatment.

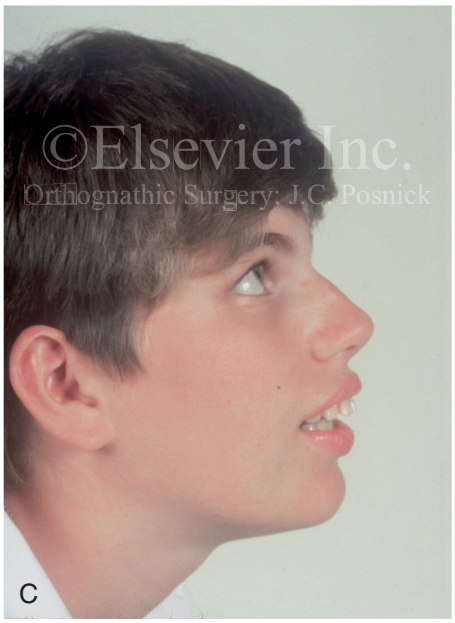
NOTE: Improvements in function (chewing, speech, swallowing, and lip closure) were immediately appreciated and accepted by the patient. The patient's psychosocial adjustment to the consistent positive comments from her peers required some accommodation. This included adjusting to not being visually recognizing as the same person by many past acquaintances.

• **Figure 7-4** A 14-year-old girl was referred by her orthodontist for surgical evaluation of a dentofacial deformity. She had a long face growth pattern that presented with excessive vertical height and significant horizontal projection and narrowness of the maxilla and the midface. She had limited airflow to the nose, severe lip incompetence, and the impression of a weak chin. Several years of orthodontics have already been carried out, and she agreed to surgical reconstruction. The procedures performed included a maxillary Le Fort I osteotomy in three segments with first bicuspid extractions (vertical intrusion, horizontal setback, and arch correction) as well as septoplasty, inferior turbinate reduction, and nasal recontouring. **A,** Profile views before and after reconstruction. **B,** Frontal views with smile before and after reconstruction.

NOTE: Improvements in speech articulation, swallowing, chewing, and lip closure were easily adapted to by the patient. After some time, her self-esteem improved, and she became less shy and withdrawn as compared with her behavior before surgery.

• **Figure 7-5** A 16-year-old congenitally blind girl presented with maxillary and mandibular protrusion and anterior open-bite malocclusion. Although she was unable to visualize the deformity, she felt self-conscious after years of teasing as a result of her facial appearance and her difficulties with chewing, swallowing, and speech articulation. The patient underwent a combined orthodontic and surgical approach. The procedures performed included a maxillary Le Fort I osteotomy (vertical intrusion and horizontal setback) and bilateral sagittal split osteotomies of the mandible (horizontal setback). **A,** Frontal views before and after reconstruction. **B,** Oblique facial views before and after reconstruction.

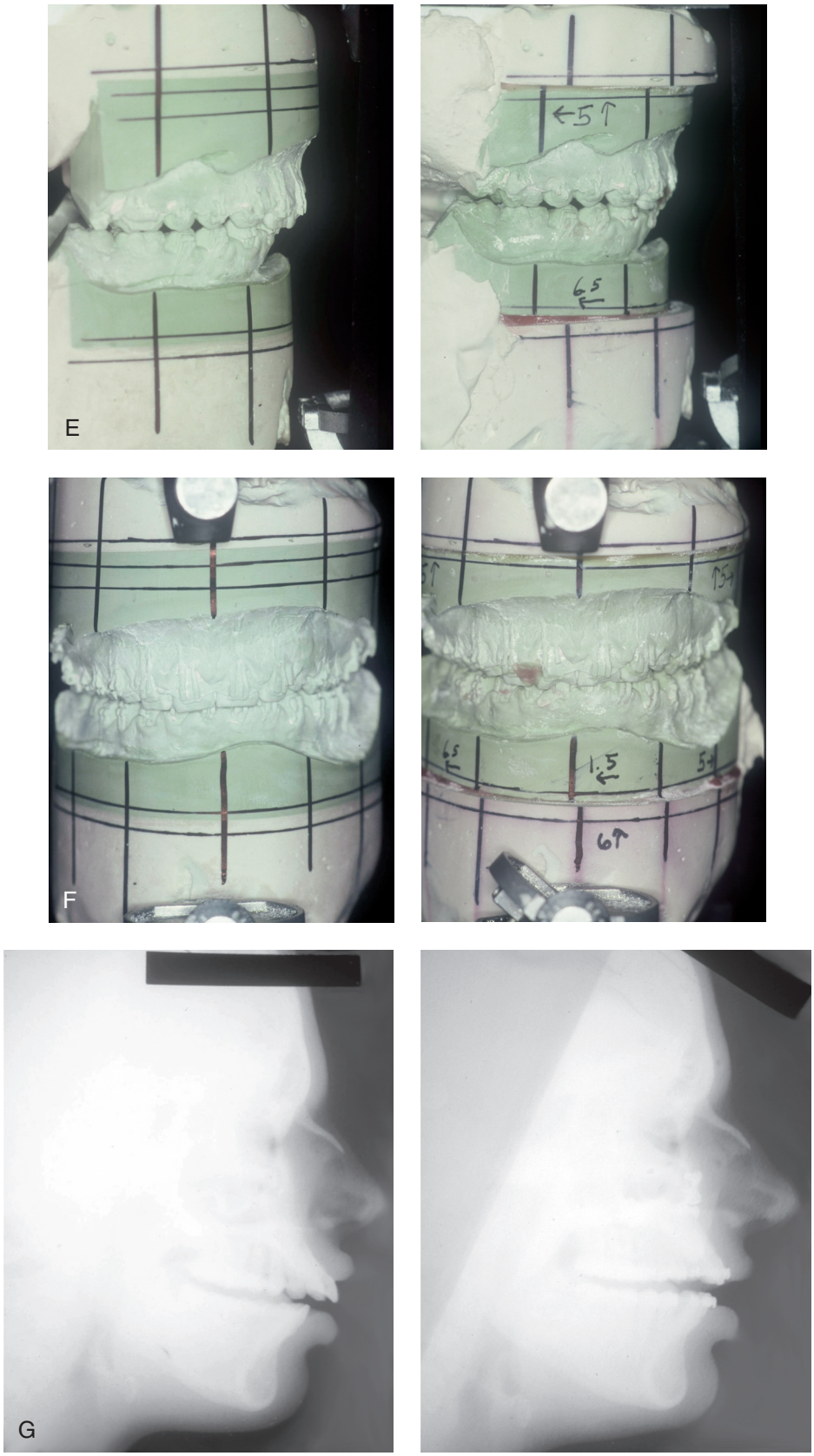
Continued



Pre treatment

After treatment

• **Figure 7-5, cont'd C,** Profile views before and after reconstruction. **D,** Occlusal views before and after reconstruction.



• **Figure 7-5, cont'd E** and **F,** Articulated dental casts that indicate analytic model planning. **G,** Lateral cephalometric radiographs before and after treatment.

Baseline Mental Health

Individuals who do not have sufficient preoperative psychological or emotional stability are more likely to experience depression, anxiety, or panic attacks and to demonstrate difficulty complying with treatment demands during the early postoperative phase.[16,17,21,27] They will also have greater difficulty with an outcome that does not meet their preconceived and often incompletely articulated expectations. If so, after surgery, they may become easily frustrated and then angry, and this may involve a loss of confidence in the surgeon. They may break off communication and seek additional opinions to justify their concerns. Some may even resort to litigation should a perceived complication or suboptimal outcome occur. Recognizing and addressing these patient-specific tendencies before surgery is the best way to limit or mitigate problems after surgery.

After an initial screening examination, a more in-depth psychological evaluation may be beneficial.[8,18,24,96] The patient and his or her family's motivation and the patient's readiness for surgery should also be assessed. Unless a sufficient support system is in place, the stress of the necessary convalescence is likely to worsen any premorbid conditions in the patient. An initial screening that results in red flags for potential emotional problems is best followed up with a more focused psychological evaluation. The patient's therapist should give clearance that the individual is considered emotionally stable enough to undergo the proposed treatment. When the treating clinicians (i.e., the surgeon, the orthodontist, and the general physician) have knowledge of the patient's premorbid tendencies, coping strategies can be instituted proactively to minimize postoperative stress. The treating mental health professional should be available to the patient, the family, and the surgical team during hospitalization, after discharge, and throughout the patient's convalescence.

High-Risk Social Behaviors

High-risk social behaviors that would be expected to affect a successful outcome should be identified before surgery. Examples include prior dependence on alcohol or on prescription or non-prescription illegal drugs. The prevalence of these past and current behavior patterns in the general population is significant and cannot be ignored.

Baseline Eating Disorders

Clinical studies indicate that between 0.5% and 1.0% of late adolescent and young adult women meet the criteria for anorexia nervosa, and between 1% and 3% meet the criteria for bulimia nervosa.[92] Eating disorders are multidetermined problems, and they are most commonly recognized among female adolescents. Risk factors may include a perfectionist personality type, a family history of an eating disorder, a history of trauma, a tendency toward feelings of inadequacy, and low self-esteem despite external accomplishments. Patients with eating disorders often exhibit rigid cognitive patterns that are characterized by an all-or-nothing way of thinking.

Orthognathic procedures will significantly alter the diet (i.e., liquids only) and appetite level, with an expected change in nutritional intake for at least 5 weeks. Significant weight loss during the initial 2 weeks of recovery is to be expected, even among healthy individuals. The question is raised as to whether or not these dietary and postsurgical metabolic changes will tend to exacerbate a baseline eating disorder. Maine and Goldberg completed a study to determine if an elective dental manipulation—which, by necessity, results in eating discomfort and dietary restrictions—would likely influence the recovery of or result in a relapse for an individual with a baseline eating disorder.[54] Three questions concerning dental and maxillofacial procedures were added to a standard questionnaire assessment of a consecutive series of 97 individuals who were entering an eating disorder therapy program. The additional questions were designed to determine the influence of dental and maxillofacial interventions on individuals with baseline eating disorders. All 97 study subjects complied with the questionnaire; 75 of the study patients were 25 years old or younger, and 53 were diagnosed with anorexia nervosa.

Interestingly, 9.3% of the study patients who had undergone either significant orthodontic therapy or the removal of the wisdom teeth in the past indicated that the dental procedure—which, by necessity, caused eating discomfort and diet alterations—had either exacerbated or resulted in a relapse of their baseline eating disorders. This study confirms that a history of an eating disorder should alert the orthodontist and the orthognathic surgeon to potential postoperative difficulties. With an awareness of the individual's baseline conditions, the orthodontist, the surgeon, the primary care physician, and members of the psychosocial team may establish guidelines for postoperative behavioral and nutritional objectives to closely monitor to ensure a smooth recovery.

Social Interaction Issues

Ongoing family or social peer conflicts or greater-than-average worries expressed by the individual or his or her family regarding the surgery, the preparation for the surgery, or the anticipated recovery period are red flags.[1,29,33,41] When considering the timing of surgery for the adolescent or young adult, the family should take into account the anticipated impact on the patient's everyday school and social activities, including examination schedules, college applications and interviews, and participation in sports activities. The surgeon's ability to provide a clear picture of the expected recovery time and the patient's ability to return to partial and regular activities is important and will vary according to the patient's age and stage in life (e.g., adolescent, college or graduate student, middle-aged adult with family and work commitments). Clarifying anticipated

postoperative limitations in physical, cognitive, and emotional activities is important to setting the stage for a successful outcome.

Body Dysmorphic Disorder

It is estimated that more than 5% of individuals who are seeking cosmetic surgery consults have body dysmorphic disorder (BDD).[14,19,65,91] BDD is a somatoform disorder that involves a preoccupation with a slight or imagined defect in some aspect of the individual's physical appearance that leads to a significant disruption of daily functions. It is important to distinguish an individual with a legitimate cosmetic or functional concern from one who is hyperconcerned about minor imperfections of some aspect of his or her face.

Cunningham and colleagues documented that a percentage of individuals who request orthognathic surgery will have underlying BDD.[19] A surgical procedure to improve facial aesthetics typically does not change or fully satisfy the appearance concerns of these individuals. Orthognathic procedures may even exacerbate these symptoms, despite the clinician's accurate assessment of the presenting deformity and the likelihood of the achievement of a favorable morphologic and functional result. No matter how skilled the surgeon and how perfect the end result, dissatisfaction should be expected when treating a patient with BDD; therefore, surgery should be avoided unless extensive counseling is performed before the proposed procedure. The development of an effective psychosocial screening interview method or the use of a self-reporting assessment survey to help identify individuals with BDD is needed in clinical practice.

Psychological Functioning in the Individual With a Facial Deformity

A spectrum of studies report that individuals with facial deformities have a higher prevalence of psychological problems that may interfere with the ability of these individuals to function socially. The psychological problems described include (1) a reduced quality of life; (2) a tendency to internalize behavior; (3) low self-esteem; (4) an increased tendency toward depression; and (5) anxiety.[13,100] Interestingly, studies confirm that it is the patient's satisfaction with his or her own appearance rather than an objective standard of the severity of the deformity that influences these psychological problems. The individual's personal dissatisfaction with his or her facial appearance seems to be a better predictor of higher rates of anxiety and depression and lower rates of favorable quality of life and self-esteem as compared with the clinician's impression of how the individual looks.

Versnel and colleagues conducted research that involved adults with facial disfigurement and confirmed that a subset of these individuals demonstrated psychological dysfunctions: 11% had anxiety; 6% had depression; 10% had externalized problems; and 25% had a tendency to internalize problems.[100] The authors clarified that either an actual or a perceived facial deformity can be a significant stressor in life to which the affected individual must adapt.[13,55,72,84,89,93] The study also looked at differences between congenital and acquired facial deformities. Many of the study patients that acquired their facial deformities later in life (i.e., those that were related to trauma or tumors) had a more difficult time, which implied that a significant percentage of those with congenital conditions had eventually adapted to their physical problems. The authors surmised that, because the congenital patients had no knowledge of how life would have been without facial disfigurement, their adaptation was different and often more complete.

An individual's fear of a negative facial appearance evaluation by others during their everyday life is considered a good predictor of psychological functioning. Therefore, psychological support for the individual with either an obvious facial deformity that cannot be further improved or a perceived facial disfigurement should focus on educating the individual to rely less on the opinions of others. From another perspective, surgery carried out for the purpose of enhancing facial appearance must reach or at least approximate the individual's self-perceived expectations. This is the reason why surgery to improve facial aesthetics in the individual with BDD will not result in patient satisfaction; the unrealistic expectations of these patients can never be fully reached.

Patient and Family Decision for or Against the Correction of a Dentofacial Deformity

The patient and the family members or significant others that comprise his or her immediate social network need a clear and consistent explanation of the following:

- The surgical objectives, anticipated treatment outcomes, and limitations with regard to predicting exact results
- How the surgery will fit in with the overall treatment plan (e.g., orthodontics, dental rehabilitation, speech therapy, airway management)
- The potential risks, complications, and limitations of each planned procedure
- What to expect during hospitalization (e.g., discomfort, swelling, hygiene needs, limitations in activity and diet, limitations in mouth opening and lip seal control, difficulties with speech and communication)
- What to expect after discharge from the hospital (i.e., ongoing convalescence needs)
- What the patient and the family must do to maximize a favorable outcome
- When the patient can expect to resume a more normal routine
- The expected time required for full healing

Information should be provided in a number of different ways, which may include (1) verbal discussion; (2) a demonstration of surgical technique on a model skull; (3) showing similar patients' before and after photographs to demonstrate the potential range of results; (4) reviewing and discussing the patient's own photographs, dental models, and radiographs; and (5) providing reinforcement with detailed and readable written material.[32,66] The option of discussing details with another patient of the same approximate age and sex who has undergone similar surgery may also be helpful.

Patient and Family Education Regarding Convalescence After Orthognathic Surgery

Early after surgery, while in hospital, the individual who (1) appears to be more irritable and non-communicative; (2) complains of greater-than-expected levels of pain with demands for continued narcotics; and (3) is having more-than-average difficulty complying with personal care needs (i.e., achieving hydration and caloric requirements, oral and body hygiene requirements, and adequate ambulation) may be a candidate for a demanding convalescence.[77,79] The adolescent may comply just enough (e.g., ambulation, fluid intake) to get out of the hospital. After he or she gets home, unless the parents are able to remain firm concerning patient care needs, the adolescent's feeling of self-consciousness or uncertainty about temporary levels of facial swelling, drooling, loss of appetite, and difficulty with breathing and speech can lead to further social withdrawal and non-compliance. This may also lead to overreliance on narcotic medication with the assumption that pain is the problem. Unless this type of patient is closely monitored, dehydration, hypoglycemia, and depression will set in. Even if this phase of convalescence is managed, some adolescents express unwillingness to return to school within the expected time frame as a result of concern about the reactions of their peers. Many will lose this reluctance when a friend is invited to their home to break the ice. As the recovering patient perceives positive affirmation from their family and friends regarding the changes brought about by surgery, their social hesitation generally subsides.

The clinicians (i.e., the surgeon, the orthodontist, and the treating dentist) often judge success on the basis of measurable standards of (1) an "improved" profile; (2) achieving normative cephalometric radiograph measurements; (3) a corrected occlusion; and (4) achieving specific wound-healing parameters. Discussions with the patient and family should not overlook psychosocial aspects. Individuals with baseline evidence of mental health issues are likely to have a greater-than-average need for positive reinforcement, and they may also require more formal psychological support throughout the perioperative process. The success of treatment should not be thought of solely in terms of improving head and neck function or the achievement of strict numerical parameters. The potential for orthognathic procedures to have profound effects on the individual's psychosocial health—whether positive or negative—should not be underestimated (see Figs. 7-1 through 7-5). During convalescence and after the return to school or work, the clinician should ask the patient about his or her interactions with others regarding the recent surgery to determine if a more in-depth discussion is required.

Patient-Centered Assessment of Surgical and Orthodontic Outcomes

Many public health experts believe that the ultimate determination of a treatment's quality and success is its effectiveness for producing not just physical health as judged by clinicians but also for resulting in the individual's personal satisfaction.[5,22,23,38,44,45,51,56,57,59,82,94,95,98] Traditional surgical outcome measurements such as mortality and improvement in physiologic parameters (e.g., blood pressure) find limited application in the treatment of dentofacial deformities. At the same time, traditional orthodontic parameters of accomplishing specific change in millimeter distances or degrees of angulation at standard anatomic sites do not necessarily correlate with patient satisfaction. Despite the importance of satisfaction as a proxy for quality, the concept of patient-perceived well-being can be difficult to define. When developing a patient questionnaire for use as a self-administered survey after orthognathic surgery, consideration should be given to the features of care that influence the patient's satisfaction. These features may be categorized into one of the following domains: (1) aesthetic outcomes; (2) functional outcomes; or (3) psychological outcomes. A review of the literature concerning patient-centered outcome assessments after orthognathic surgery sheds light on the issue of how to achieve patient-based satisfaction after treatment.

Orthognathic Surgery and Health-Related Quality of Life: Review of Study

Posnick and colleagues reported on data from a health-related quality-of-life survey that was completed postoperatively by a consecutive series of patients who had undergone bimaxillary orthognathic surgery in combination with intranasal and other adjunctive procedures. The survey was filled out by each study patient only after recovery from surgery and after all orthodontic therapy and dental rehabilitation was complete (i.e., a minimum of 6 months after surgery).[82]

A postsurgical patient satisfaction questionnaire was designed by the authors to assess each patient's overall satisfaction with the procedures that he or she underwent, patient-perceived functional improvements, and patient-perceived negative effects. The self-assessment survey was mailed to a consecutive series of 42 individuals who

underwent bimaxillary orthognathic surgery (i.e., Le Fort I osteotomy and sagittal split ramus osteotomies of the mandible) in combination with intranasal (i.e., septoplasty and inferior turbinate reduction) and other patient-specific procedures by a single surgeon (Posnick) at one institution during a 12-month time frame. Patients who underwent orthognathic surgery for the correction of a syndrome, a cleft, or a posttraumatic deformity during the same time frame were excluded from the study. The study group included 15 males and 27 females. The mean age was 25 years, and patient ages ranged from 15 to 55 years. Seven of the 42 patients were less than 18 years old: four were 15 years old, 1 was 16 years old, and two were 17 years old. The most frequent pattern of jaw deformity that was identified in the study group was a long face growth pattern (17 out of 42 patients [40%]), which was characterized as vertical maxillary excess combined with mandibular deficiency. This was followed by primary mandibular deficiency (9 out of 42 patients [21%]). Simultaneous maxillofacial procedures that were completed in addition to those listed previously varied in accordance with patient-specific needs. Thirty-eight percent of the patients (16 out of 42) underwent the simultaneous removal of wisdom teeth. Seventeen percent of the patients (7 out of 42) also underwent suction-assisted lipectomy of the neck. Eighty-six percent of the patients (36 out of 42) underwent osseous genioplasty. The postsurgical patient satisfaction questionnaire made use of a 7-point Likert scale for the respondents to rate their levels of satisfaction or acceptance for each of nine questions that were asked.

To assess overall patient satisfaction three questions were asked. The first question was phrased as follows: "If you had to make the decision again, how likely would you be to undergo this same surgery?" Eighty-three percent (35 out of 42 patients) responded positively to this question; 12% (5 out of 42 patients) were neutral, and 5% (2 out of 42 patients) reported that they would not have undergone orthognathic surgery if they had to do it all over again. The second question was written as follows: "How likely would you be to recommend the same surgery to others?" Two individuals (5%) stated that they would not recommend the same surgery to others. One of the 42 patients (2%) was neutral, whereas 93% of the patients (39 out of 42) stated that they would recommend the same surgery to others. The patients were then asked the following question: "Considering everything, how satisfied are you now with the results of surgery?" Eighty-eight percent (37 out of 42 patients) were satisfied; 1 patient (2%) was neutral, and 4 patients (10%) had at least some degree of uncertainty. These results showed that more than 70% of subjects gave a positive response to all three global satisfaction questions. According to the survey, only 2 out of the 42 consecutive patients (5%) who were treated were dissatisfied (i.e., they answered negatively to all three global satisfaction questions). Interestingly, one of the two dissatisfied patients was judged by the three treating clinicians (i.e., the surgeon, the orthodontist, and the restorative dentist) to have a satisfactory result (i.e., an acceptable occlusion, an improved upper airway, and enhanced facial aesthetics). The second dissatisfied patient developed idiopathic condylar resorption approximately 3 months after surgery, with a resulting residual malocclusion.

The Pearson correlation coefficient highlights several points related to this study group. The global satisfaction questions within the survey (question nos. 1, 2, and 3) all correlate highly with each other. Of the three global satisfaction questions asked, question no. 3 correlates closest with the specific head and neck function questions (question nos. 4 through 9). Of the specific function questions that were asked (question nos. 4 through 9), the perceived residual lip and chin region numbness (question no. 9) had the weakest correlation with overall patient satisfaction. In other words, most individuals were overall satisfied with the results of their orthodontics and orthognathic procedures, despite the fact that they would have preferred a greater return of lip and chin region sensation.

The following question was also asked: "Overall, how satisfied are you with your current bite?" At the completion of treatment, 5 out of 42 patients (12%) were not entirely satisfied with their occlusion, whereas 2% (1 out of 42 patients) gave a neutral response to this question. The majority of patients (36 out of 42 [86%]) expressed satisfaction with their occlusal result after the completion of treatment.

Speech articulation issues are not generally considered a major factor in a patient's decision to undergo jaw surgery. The following question about this issue was asked: "Overall, how satisfied are you with your current speech articulation?" The data suggests that 34 out of 42 patients (80%) were satisfied with their postoperative speech articulation. Nine percent (4 out of 42 patients) had no strong feelings, whereas 4 out of 42 patients (9%) were not completely satisfied with their speech at the completion of treatment.

Patients in this study group were also asked the following: "Overall, how satisfied are you with your current lip posture and lip closure?" Eighty-six percent (36 out of 42) of the patients were satisfied with static lip posture and their ability to actively close their lips. Twelve percent (5 out of 42 patients) were not entirely satisfied; they apparently felt that their ability to control their lips was not completely normal.

The patients were then asked this question: "Overall, how satisfied are you with your current breathing?" Eighty-one percent (38 out of 42 patients) were overall satisfied with their breathing after treatment. Five percent (2 out of 42) were neutral with regard to their assessment of their own breathing, whereas 2 out of 42 patients (5%) remained dissatisfied with at least some aspects of their breathing.

Each patient was asked the following question: "Overall, how accepting are you with your current level of TMJ [temporomandibular joint] function/facial pain?" Eighty-eight percent (37 out of 42 patients) were accepting of their current TMJ function and of any degree of facial pain that they may have. Five percent (2 out of 42 patients) were

neutral about this issue, whereas 3 out of 42 patients (7%) were not completely accepting of their level of TMJ dysfunction or facial pain after treatment.

Patients were asked to respond to the following question: "Overall, how accepting are you with your current level of lower lip/chin sensation?" This question registered the highest level of patient dissatisfaction. Twenty-one percent of patients (9 out of 42) wished that their postsurgical sensory loss was not as much. Twenty-four percent of patients (10 out of 42) were neutral on this issue, whereas 23 out of 42 (55%) were accepting of their level of lower lip and chin region sensory return after treatment.

Controversies Surrounding "Successful" Orthognathic Outcomes

Interestingly, although 5 out of 42 (12%) of the patients studied by Posnick and colleagues were not completely satisfied with their occlusions, only 2 out of 42 (5%) of the patients were dissatisfied with the overall experience of having undergone the orthognathic procedures and the orthodontic treatment. Therefore, it is apparent that the occlusion achieved is not the only and not even the most important factor when a patient is judging surgical success or failure. Posnick's study group data also showed that, despite the fact that 21% (9 out of 42) of the patients were not entirely comfortable with the degree of sensory return to the lower lip and chin region, only 5% (2 out of 42) were dissatisfied with the postoperative surgical results overall.

Not surprisingly, orthognathic surgery patients' self-verification needs (i.e., the need to feel accepted) are generally an important factor in their judgment of successful treatment. This need cannot be overlooked if a favorable result is to be achieved.* For example, patients who undergo orthognathic procedures may expect to draw attention to their new appearance. If orthodontic treatment and orthognathic surgery result in positive and praising feedback from those in their social circles, self-verification is fulfilled, and they are likely to be satisfied. Some orthognathic patients may have realistic expectations but do not have a supportive social network of family and friends to fulfill their self-verification needs. Negative appearance–related commentary (e.g., teasing by peers at school, or more subtle forms of negative feedback by adults) has been shown to significantly affect body image and psychosocial functioning after surgery. If the comments of others are not positive or do not meet personal expectations (which can be unrealistically high), self-verification needs are not fulfilled, and the patient is likely to be dissatisfied. One concrete self-verification need after orthognathic surgery is a "normal" occlusion. The feedback that an individual receives from his or her treating clinicians (i.e., the orthodontist, the restorative dentist, and the surgeon) about his or her occlusion would be expected to influence that individual's level of satisfaction.

Kiyak and colleagues completed a study that evaluated the psychological changes in patients who were undergoing orthognathic surgery.[46-51] They used a standardized questionnaire before and at intervals that extended for 2 years after surgery. The data for all six measurement periods (2 weeks preoperatively, in the hospital, and 2 weeks, 4 months, 8 months, and 2 years postoperatively) were available for 46 patients. Several important findings from this research included the following:

1. Complaints of functional head and neck problems decreased significantly from before surgery to 24 months after surgery.
2. A significant percentage of patients (49%) continued to report lip paresthesia 2 years after surgery.
3. The incidence of early postsurgical problems or complaints (i.e., at 2 weeks) had no consistent effect on the satisfaction expressed with longer-term surgical outcomes (i.e., at 2 years).
4. The satisfaction expressed by patients with surgical outcomes at 4 months remained high throughout the postoperative course (i.e., at 2 years).
5. Self-esteem appeared to rise in anticipation of surgery (i.e., at 2 weeks preoperatively) but to decline at 8 months after surgery. The patients' self-esteem then rose again as documented at 24 months after surgery. Interestingly, on average, self-esteem never reached the high expectations of the surgical outcome that the patients had expressed at 2 weeks before surgery.
6. Body image showed a decline at 9 months after surgery as compared with its highest point. Fortunately, the overall body image at 2 years after surgery was significantly more positive than it was at 2 weeks before surgery.

On the basis of this data, Kiyak's group suggests that, if feasible, clinicians should maintain periodic contact with their orthognathic surgery patients for at least 2 years after surgery.[46-51] Although clinician interactions with the patient immediately after surgery generally fall to the surgeon (i.e., at 0 to 5 weeks), during the intermediate stage after surgery (i.e., 1 to 12 months), clinician feedback primarily falls to the orthodontist and the general dentist. According to the study, at 6 to 12 months after surgery, psychological unevenness for the patient is not uncommon. The patient and the orthodontist may experience conflict regarding the importance of "finishing the bite" and the timing of the completion of treatment. The patient may become adamant that the braces be removed for a specific social event (e.g., graduation, yearbook photographs), whereas the orthodontist may wish to continue detailing the bite. The orthodontist may focus on dental aspects that are considered to be of less immediate importance from the patient's perspective. At the same time, the patient may have concern about persistent paresthesia (i.e., of the inferior alveolar and infraorbital

*References 7, 11, 15, 20, 28, 30, 31, 35, 36, 39, 40, 43, 47-50, 52, 60, 62, 70, 74, 85, 88, 90, 99

nerves) and whether this is a complication or an expected consequence of the jaw surgery. If accurate information was given before surgery and if the lines of communication remain open between the patient and the surgeon, a favorable resolution of this issue is likely. The orthodontist should be aware of the expected progression of sensory recovery and suggest a return visit to the surgeon if he or she perceives a potential conflict. If instead the orthodontist, the restorative dentist, or the office personnel (e.g., the hygienist, the dental assistant) make off-the-cuff disparaging remarks about the patient's facial aesthetics, occlusion, or incomplete facial sensory return, then the patient may assume the worst.

At the conclusion of their study (i.e., 24 months after surgery), Kiyak and colleagues found that a majority of patients had achieved their personal dental (occlusal) or jaw function objectives.[46-51] Nevertheless, a minority of the subjects continued to have lingering concerns. If the patient perceived that the facial aesthetic improvements did meet his or her personal expectations, then overall satisfaction was high, regardless of any residual functional complaints (e.g., paresthesia, dysesthesia, temporomandibular disorder, occlusal imperfection). The majority of the orthognathic patients in the study by Kiyak and colleagues did in fact perceive facial aesthetic improvements and were therefore pleased.

The authors made several suggestions. First, it is helpful to prepare patients thoroughly before surgery and to explain not just the temporary functional impairment that they will experience soon after surgery but also the psychological changes toward themselves and toward others that are to be expected throughout recovery. Second, it is important to emphasize that these changes will likely continue to evolve for as long as 2 years after surgery. Third, if the patient is unprepared for the anticipated facial aesthetic changes, then dissatisfaction is also more likely. For these reasons, both the surgeon and the orthodontist should maintain open lines of communication with the patient and each other long after primary skeletal healing has occurred. Anticipating the persistence of lower lip and chin paresthesia as a consequence of the mandibular osteotomies should be communicated clearly during the preoperative discussion and then reinforced at several postoperative intervals.

Lee and colleagues completed a study to evaluate the patient-centered assessment of health at 6 weeks and then 6 months after orthognathic surgery.[53] Their 36 study patients were evaluated at intervals that included baseline/presurgery; 6 weeks postoperatively; and 6 months postoperatively. A standardized health-related survey was given at each time interval. The results of this study demonstrated a significant deterioration in both the physical and mental health of the patients soon after surgery (at 6 weeks) as compared with baseline. This matches the expected signs and symptoms that are typically observed soon after surgery. At 6 months after surgery, there was a statistical return to baseline levels in combination with significant improvements in several of the parameters that were measured. The authors concluded that significant positive changes in quality of life occurred after orthognathic surgery in the majority of patients when their healing was complete. There was a marked but transient deterioration in many aspects related to physical and emotional well-being during the early postoperative period (i.e., the first 6 weeks after surgery). This was followed by significant improvements that were documented by 6 months after surgery.

In a discussion of their results, Lee and colleagues stated that patients who report unexpected negative effects after surgery are more likely to be dissatisfied with their overall treatment. Soon after surgery, Lee's group documented difficulties with the orthognathic patient's ability to carry out daily physical activities, work performance, and social activities with friends and family. Patients experienced moderate degrees of pain, tiredness or lack of energy, and emotional impairment during the early recovery period. All of these changes are predictable and temporary, and they should be explained to the patient and the patient's family before surgery and then reinforced during the postoperative visits. It was found that, even during the early recovery period (i.e., the initial 6 weeks), as long as reasonable preoperative education was provided, there is generally improvement in the patient's psychological status. The authors believe that this is a reflection of the patient's perceived improvements in both occlusion and facial appearance meeting his or her personal expectations despite difficulties experienced as part of the normal healing process. By 6 months after orthognathic surgery, there was a trend toward further improvements in emotional well-being. By then, the overall study group consciously experienced marked improvements in facial aesthetics, oral and jaw function, and social abilities.

Conclusions

A review of the literature regarding patient satisfaction after orthognathic surgery confirms the need for the following: (1) careful preoperative patient selection; (2) clear communication of realistic and agreed-upon objectives; and (3) including the family or the immediate social network and all collaborating clinicians in the process. Although the majority of patients and their families will be pleased with the results that are achieved, a small percentage will be dissatisfied, despite the clinician's perception that the results achieved were at least an improvement and even when no specific complications occurred. In addition, as with any form of treatment or surgery, complications may occur despite the best of care. Until the science of patient selection parallels clinical realities, anticipating a small percentage of dissatisfied patients and families remains the norm. The meticulous documentation of the interactions between the patient and the doctor as well as the maintenance of open lines of communication between the patient and all treating clinicians beyond the initial healing period remain essential components of patient management when performing orthognathic procedures.

References

1. Adams GR: The effects of physical attractiveness on the socialization process. In Lucker GW, Ribbens KA, McNamara JA, editors: *Psychological aspects of facial form. Craniofacial Growth Series Monograph No. 11*, Ann Arbor, Mich, 1981, University of Michigan Press, pp 25–47.
2. Athanasiou AE, Melsen B, Eriksen J: Concerns, motivations and experience of orthognathic surgery patients: a retrospective study of 152 patients. *Int J Adult Orthodon Orthognath Surg* 4:47–55, 1989.
3. Bell R, Kiyak HA, Joondeph DR, et al: Perceptions of facial profile and their influence on the decision to undergo orthognathic surgery. *Am J Orthod* 88:323–332, 1985.
4. Bellucci CC, Kapp-Simon KA: Psychological considerations in orthognathic surgery. *Clin Plastic Surg* 34:e11–e16, 2007.
5. Bennett ME, Phillips CL: Assessment of health-related quality of life for patients with severe skeletal disharmony: a review of the issues. *Int J Adult Orthodon Orthognath Surg* 14:65, 1999.
6. Berscheid E: An overview of the psychological effects of physical attractiveness. In Lucker GW, Ribbons KA, McNamara JA, editors: *Psychological aspects of facial form, Craniofacial Growth Series Monograph No. 11*, Ann Arbor, Mich, 1981, University of Michigan Press, pp 1–23.
7. Bertolini F, Russo V, Sansebastiano G: Pre- and postsurgical psycho-emotional aspects of the orthognathic surgery patient. *Int J Adult Orthodon Orthognath Surg* 15:16–23, 2000.
8. Blinder D, Rotenberg L, Taicher S: Conversion disorder after maxillofacial trauma and surgery. *Int J Oral Maxillofac Surg* 25:116, 1996.
9. Broder HL: Psychological research of children with craniofacial anomalies: review, critique and implications for the future. *Cleft Palate Craniofac J* 34:402, 1997.
10. Bull R, Rumsey N: *The social psychology of facial appearance*, New York, 1988, Springer-Verlag.
11. Chen B, Zhang ZK, Wang X: Factors influencing postoperative satisfaction of orthognathic surgery patients. *Int J Adult Orthodon Orthognath Surg* 17:217–222, 2002.
12. Cheng LHH, Roles D, Telfer MR: Orthognathic surgery: the patient's perspective. *Br J Oral Maxillofac Surg* 36:261, 1998.
13. Clifford E, Crocker EC, Pope BA: Psychological findings in the adulthood of 98 cleft lip-palate children. *Plast Reconstr Surg* 50:234–237, 1972.
14. Crerand C, Franklin M, Sarwer DB: Body dysmorphic disorder and cosmetic surgery. *Plast Reconstr Surg* 118:167, 2006.
15. Crowell NT, Sazima HJ, Elder ST: Survey of patients' attitudes after surgical correction of prognathism: study of 33 patients. *J Oral Surg* 28:818–822, 1970.
16. Cucalon A, Smith RJ: Relationship between compliance by adolescent orthodontic patients and performance on psychological tests. *Angle Orthod* 60:107, 1990.
17. Cunningham SJ, Hunt NP, Feinmann C: Psychological aspects of orthognathic surgery: a review of the literature. *Int J Adult Orthodon Orthognath Surg* 10:159, 1995.
18. Cunningham SJ, Feinmann C, Horrocks EN: Psychological problems following orthognathic surgery. *J Clin Orthod* 29:755, 1995.
19. Cunningham SJ, Bryant CJ, Manisali M, et al: Dysmorphophobia: recent developments of interest to the maxillofacial surgeon. *Br J Oral Maxillofac Surg* 34:368, 1996.
20. Cunningham SJ, Crean SJ, Hunt NP, et al: Preparation, perceptions and problems: a long-term follow-up study of orthognathic surgery. *Int J Adult Orthod Orthognath Surg* 11:41–47, 1996.
21. Cunningham SJ, Feinmann C: Psychological assessment of patients requesting orthognathic surgery and the relevance of body dysmorphic disorder. *Br J Orthod* 25:293, 1998.
22. Cunningham SJ, Garratt AM, Hunt NP: Development of a condition-specific quality of life measure for patients with dentofacial deformity: I. Reliability of the instrument. *Community Dent Oral Epidemiol* 28:195, 2000.
23. Cunningham SJ, Garratt AM, Hunt NP: Development of a condition-specific quality of life measure for patients with dentofacial deformity: II. Validity and responsiveness testing. *Community Dent Oral Epidemiol* 30:81, 2002.
24. De Sousa A: Psychological issues in oral and maxillofacial reconstructive surgery. *J Oral Maxillofac Surg* 46:661, 1998.
25. Eagly AH, Ashmore RD, Makhijani IVIG, Longo LC: What is beautiful is good, but…: a meta-analytic review of research on the physical attractiveness stereotype. *Psychol Bull* 110:109–128, 1991.
26. Finlay PM, Atkinson JM, Moos KF: Orthognathic surgery: patient expectations; psychological profile and satisfaction with outcome. *Br J Oral Maxillofac Surg* 33:9–14, 1995.
27. Fitts WH: *Manual for the Tennessee Department of Mental Health Self-Concept Scale*, Nashville, Tenn, 1965, Department of Mental Health.
28. Flanary CM, Alexander JM: Patient responses to the orthognathic surgery experience: factors leading to dissatisfaction. *J Oral Maxillofac Surg* 41:770, 1983.
29. Flanary CM, Barnwell GM, Jr, Alexander JM: Patients' perception of orthognathic surgery. *Am J Orthod* 88:137, 1985.
30. Flanary CM, Barnwell GM, VanSickels JE, et al: Impact of orthognathic surgery on normal and abnormal personality dimensions: a 2-year follow-up study of 61 patients. *Am J Orthod Dentofac Orthop* 98:313, 1990.
31. Fonsecca RJ, Baily LT, Proffitt WR, et al: *Patient selection for orthognathic surgery*. In *Oral and Maxillofacial Surgery,* vol 2, Philadelphia, 2000, Saunders, pp 3–4.
32. Forssell H, Finne K, Forssell K, et al: Expectations and perceptions regarding treatment: a prospective study of patients undergoing orthognathic surgery. *Int J Adult Orthodon Orthognath Surg* 13:107, 1998.
33. Frost V, Peterson G: Psychological aspects of orthognathic surgery: how people respond to facial change. *Oral Surg Oral Med Oral Pathol* 71:538, 1991.
34. Garvill J, Garvill H, Kahnberg KE, Lundgren S: Psychological factors in orthognathic surgery. *J Craniomaxillofac Surg* 20:28–33, 1992.
35. Haimovitz D, Lansky LM, O'Reilly P: Fluctuations in body satisfaction across situations. *Int J Eat Disord* 13:77, 1993.
36. Hakman ECJ: Psychological studies on orthognathic surgery. In De Beaufort I, Hilhorst M, Holm S, editors: *In the eye of the beholder. Ethics and medical change of appearance*, Copenhagen, 1996, Scandinavian University Press, pp 48–75.
37. Harper DC: Children's attitudes to physical differences among youth from Western and non-Western cultures. *Cleft Palate Craniofac J* 32:114, 1995.
38. Hatch JP, Rugh JD, Clark GM, et al: Health-related quality of life following orthognathic surgery. *Int J Adult Orthodon Orthognath Surg* 13:67, 1998.
39. Hatch JP, Rugh JD, Bays RA, et al: Psychological function in orthognathic surgical patients before and after bilateral split osteotomy with rigid and wire fixation. *Am J Orthod Dentofacial Orthop* 115:536, 1999.
40. Heldt L, Haffke EA, Davis LF: The psychological and social aspects of orthognathic treatment. *Am J Orthod* 82:318, 1982.
41. Holman AR, Brumer S, Ware WH, et al: The impact of interpersonal support on patient satisfaction with orthognathic surgery. *J Oral Maxillofac Surg* 53:1289, 1995.
42. Hoppenreijs TJM, Hakman ECJ, Van't Hof MA, et al: Psychological implications of surgical-orthodontic treatment in patients with anterior open bite. *Int J Adult Orthodon Orthognath Surg* 14:101–112, 1999.
43. Hunt OT, Johnston CD, Hepper PG, et al: The psychosocial impact of orthognathic

surgery: a systematic review. *Am J Orthod Dentofac Orthop* 120:490, 2001.
44. Hutton CE: Patients' evaluation of surgical correction of prognathism: survey of 32 patients. *J Oral Surg* 25:225–228, 1967.
45. Juggins KJ, Nixon F, Cunningham SJ: Patient- and clinician-perceived need for orthognathic surgery. *Am J Orthod Dentofacial Orthop* 128:697, 2005.
46. Kiyak HA, Hohl T, Sherrick P, et al: Sex differences in motives for and outcomes of orthognathic surgery. *J Oral Surg* 39:757–764, 1981.
47. Kiyak HA, McNeill RW, West RA, et al: Predicting psychologic responses to orthognathic surgery. *J Oral Maxillofac Surg* 40:150–155, 1982.
48. Kiyak HA, West RA, Hohl T, et al: The psychological impact of orthognathic surgery: a 9-month follow-up. *Am J Orthod* 81:404–412, 1982.
49. Kiyak HA, Hohl T, West RA, et al: Psychologic changes in orthognathic surgery patients: a 24-month follow up. *J Oral Maxillofac Surg* 42:506, 1984.
50. Kiyak HA, McNeill RW, West RA: The emotional impact of orthognathic surgery and conventional orthodontics. *Am J Orthod* 88:224, 1985.
51. Kiyak HA, Zeitler DL: Self-assessment of profile and body image among orthognathic surgery patients before and two years after surgery. *J Oral Maxillofac Surg* 46:365–371, 1988.
52. Lazaridou-Terzoudi T, Kiyak HA, Moore R, et al: Long-term assessment of psychological outcome of orthognathic surgery. *J Oral Maxillofac Surg* 61:545, 2003.
53. Lee S, McGrath C, Samman N: Impact of orthognathic surgery on quality of life. *J Oral Maxillofac Surg* 66: 1194–1199, 2008.
54. Maine M, Goldberg MH: The role of third molar surgery in the exacerbation of eating disorders. *J Oral Maxillofac Surg* 59:1297–1300, 2001.
55. Marcusson A, List T, Paulin G, Akerlind I: Reliability of a multidimensional questionnaire for adults with treated complete cleft lip and palate. *Scand J Plast Reconstr Surg Hand Surg* 35: 271–278, 2001.
56. Modig M, Andersson L, Ward I: Patients' perception of improvement after orthognathic surgery: pilot study. *Br J Oral Maxillofac Surg* 44:24, 2006.
57. Montegi E, Hatch JP, Rugh JD, Yamagichi H: Health-related quality of life and psychosocial function 5 years after orthognathic surgery. *Am J Orthod Dentofacial Orthop* 124:138, 2003.
58. Murray JE, Mulliken JB, Kaban IB, et al: Twenty-year experience in maxillofacial surgery: an evaluation of early surgery on growth function and body image. *Ann Surg* 190:320, 1979.
59. Narayanan V, Guhan S, Sreekumar K, et al: Self-assessment of facial form oral function before and after orthognathic surgery: a retrospective study. *Indian J Dent Res* 19:12, 2008.
60. Newton JT, Minhas G: Exposure to "ideal" facial images reduces facial satisfaction: an experimental study. *Community Dent Oral Epidemiol* 33:410, 2005.
61. Nurminen L, Pietila T, Vinkka-Puhakka H: Motivation for and satisfaction with orthodontic-surgical treatment: a retrospective study of 28 patients. *Eur J Orthod* 21:79, 1999.
62. Oland J, Jensen J, Melsen B: Factors of importance for the functional outcome in orthognathic surgery patients: a prospective study of 118 patients. *J Oral Maxillofac Surg* 68:2221, 2010.
63. Oland J, Jensen J, Elklit A, et al: Motives for surgical-orthodontic treatment and effect of treatment on psychosocial well-being and satisfaction: a prospective study of 118 patients. *J Oral Maxillofac Surg* 69:104–113, 2011.
64. Oland J, Jensen J, Papadopoulos MA, Melsen B: Does skeletal facial profile influence preoperative motives and postoperative satisfaction? A prospective study of 66 surgical-orthodontic patients. *J Oral Maxillofac Surg* 69:2025–2032, 2011.
65. O'Regan J, Dewey M, Slade P, Lovius B: Self-esteem and aesthetics. *Br J Orthod* 18:111–118, 1991.
66. Ostler S, Kiyak HA: Treatment expectations versus outcomes among orthognathic surgery patients. *Int J Adult Orthodon Orthognath Surg* 6:247–255, 1991.
67. Ouellette PL: Psychological ramifications of facial change in relation to orthodontic treatment and orthognathic surgery. *J Oral Surg* 36:787, 1978.
68. Padwa BL, Evans CA, Pillemer FC: Psychosocial adjustment in children with hemifacial microsomia and other craniofacial deformities. *Cleft Palate Craniofac J* 28:354, 1991.
69. Patel PK, Kapp-Simon KA: Orthognathic surgery in adolescence: the patient's perspective [abstract]. American Association of Plastic Surgeons 83rd Annual Meeting. Chicago, 2004.
70. Pavone I, Rispoli A, Acocella A, et al: Psychological impact of self-image dissatisfaction after orthognathic surgery: a case report. *World J Orthod* 6:141, 2005.
71. Pertschuk MJ, Whitaker LA: Psychosocial adjustment and craniofacial malformations in childhood. *Plast Reconstr Surg* 75:177, 1985.
72. Peter JP, Chinsky RR: Sociological aspects of cleft palate adults: I. Marriage. *Cleft Palate J* 11:295–309, 1974.
73. Peterson LJ, Topazian RG: The preoperative interview and psychological evaluation of the orthognathic surgery patient. *J Oral Surg* 32:583, 1974.
74. Phillips C, Hill BJ, Cannac C: The influence of video imaging on patients' perceptions and expectations. *Angle Orthod* 65:263–270, 1995.
75. Phillips C, Broder HL, Bennett ME: Dentofacial disharmony: motivations for seeking treatment. *Int J Adult Orthodon Orthognath Surg* 12:7, 1997.
76. Phillips C, Bennett ME, Broder HL: Dentofacial disharmony: psychological status of patient seeking a treatment consultation. *Angle Orthod* 68:547–566, 1998.
77. Phillips C, Kiyak HA, Bloomquist D, et al: Perceptions of recovery and satisfaction in the short term after orthognathic surgery. *J Oral Maxillofac Surg* 62:535, 2004.
78. Phillips J, Whitaker LA: The social effects of craniofacial deformity and its correction. *Cleft Palate J* 16:7, 1979.
79. Pillemer FG, Cook KV: The psychosocial adjustment of pediatric craniofacial patients after surgery. *Cleft Palate J* 26:201, 1989.
80. Pope AW, Speltz ML: Research on psychosocial issues of children with craniofacial anomalies: Progress and challenges. *Cleft Palate Craniofac J* 34:371, 1997.
81. Pope AW, Ward J: Self-perceived facial appearance and psychosocial adjustment in preadolescents with craniofacial anomalies. *Cleft Palate Craniofac J* 34:396, 1997.
82. Posnick JC, Wallace J: Complex orthognathic surgery assessment of patient satisfaction. *J Oral Maxillofac Surg* 66:934, 2008.
83. Proothi M, Drew SJ, Sachs SS: Motivating factors for patients undergoing orthognathic surgery evaluation. *J Oral Maxillofac Surg* 68:1555–1559, 2010.
84. Ramstad T, Ottem E, Shaw WC: Psychological adjustment in Norwegian adults who had undergone standardized treatment of complete cleft lip and palate: I. Education, employment and marriage. *Scand J Plast Reconstr Surg Hand Surg* 29:251–257, 1995.
85. Rispoli C, Acocella A, Pavone I, et al: Psychoemotional assessment changes in patients treated with orthognathic surgery: pre- and postsurgery report. *World J Orthod* 5:48, 2004.
86. Rivera SM, Hatch JP, Dolce C, et al: Patients' own reasons and patient-perceived recommendations for orthognathic surgery. *Am J Orthod Dentofacial Orthop* 18:134, 2000.
87. Robinson E, Rumsey N, Partridge J: An evaluation of the impact of social interaction skills training for facially disfigured people. *Br J Plast Surg* 49:281–289, 1996.
88. Sarver DM, Johnston MW, Matukas VJ: Video imaging for planning and counseling in orthognathic surgery. *J Oral Maxillofac Surg* 46:939–945, 1988.
89. Sarwer DB, Bartlett SP, Whitaker LA, et al: Adult psychological functioning of individuals born with craniofacial anomalies. *Plast Reconstr Surg* 103:412–418, 1999.
90. Scott AA, Hatch JP, Rugh JD, et al: Psychosocial predictors of satisfaction among orthognathic surgery patients. *Int J Adult Orthodon Orthognath Surg* 15:7–15, 2000.

91. Secord PF, Jourard SM: The appraisal of body cathexis: body cathexis and the self. *J Consult Psychol* 17:343–347, 1953.
92. Shisslak CM, Crago M, Estes LS: The spectrum of eating disorders. *Int J Eating Disord* 18:209, 1995.
93. Sinko K, Jagsch R, Prechtl V, et al: Evaluation of esthetic, functional, and quality of life outcome in adult cleft lip and palate patients. *Cleft Palate Craniofac J* 42:355–361, 2005.
94. Slade GD, Spencer AJ: Development and evaluation of the Oral Health Impact Profile. *Community Dent Health* 11:3, 1994.
95. Slade GD: Assessing change in quality of life using the Oral Health Impact Profile. *Community Dent Oral Epidemiol* 26:52, 1998.
96. Stewart TD, Sexton J: Depression: a possible complication of orthognathic surgery. *J Oral Maxillofac Surg* 45:847, 1987
97. Stirling J, Latchford G, Morris DO, et al: Elective orthognathic treatment decision making: a survey of patient reasons and experiences. *J Orthod* 34:114, 2007.
98. Travess HC, Newton JT, Sandy JR, et al: The development of a patient-centered measure of the process and outcome of combined orthodontic and orthognathic treatment. *J Orthod* 31:220, 2004.
99. Upton PM, Sadowsky PL, Sarver DM, et al: Evaluation of video imaging prediction in combined maxillary and mandibular orthognathic surgery. *Am J Orthod Dentofacial Orthop* 112:656, 1997.
100. Versnel SL, Plomp RG, Passchier J, et al: Long-term psychological functioning of adults with severe congenital facial disfigurement. *Plast Reconstr Surg* 129:110–117, 2012.
101. Wictorin L, Hillerstrom K, Sorensen S: Biological and psycho-social factors in patients with malformations of the jaws. I. A study of 95 patients prior to treatment. *Scand J Plast Reconstr Surg* 3:138–143, 1969.
102. Williams AC, Shah H, Sandy JR, et al: Patients' motivations for treatment and their experiences of orthodontic preparation for orthognathic surgery. *J Orthod* 32:191, 2005.
103. Williams DM, Bentley R, Cobourne MT, et al: Psychological characteristics of women who require orthognathic surgery: comparison with untreated controls. *Br J Oral Maxillofac Surg* 47:191, 2009.
104. Wilmot JJ, Barber HD, Chou DG, et al: Associations between severity of dentofacial deformity and motivation for orthodontic-orthognathic surgery treatment. *Angle Orthod* 63:283, 1993.
105. Zhou Y, Hagg U, Rabie AB: Severity of dentofacial deformity, the motivations and the outcome of surgery in skeletal Class III patients. *Chin Med J (Engl)* 115:1031, 2002.

8

Speech, Mastication, and Swallowing Considerations in the Evaluation and Treatment of Dentofacial Deformities

JEFFREY C. POSNICK, DMD, MD

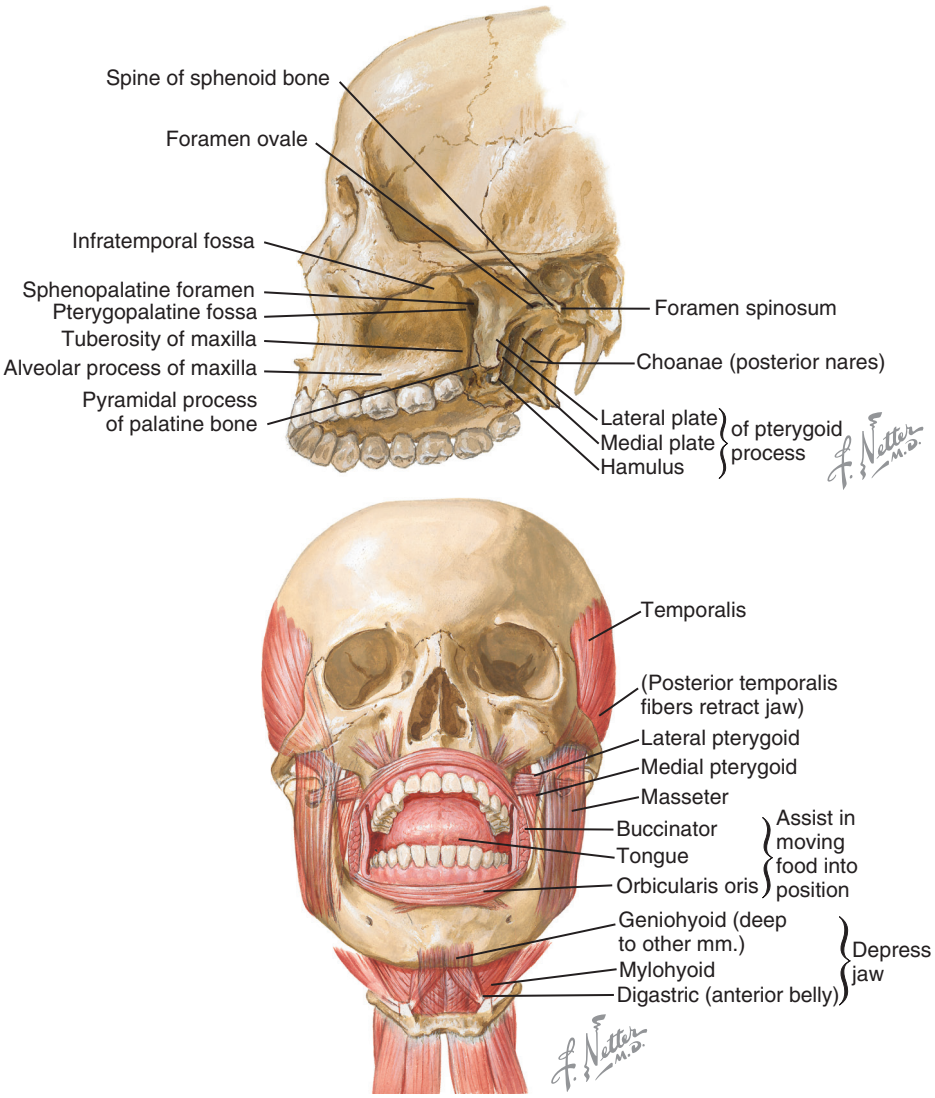
- The Neuromuscular Masticatory System
- Mastication Mechanism
- Swallowing Mechanism
- Speech: Mechanism, Formation, and Assessment
- Effects of Jaw Deformities with Malocclusion on Speech
- Effects of Successful Orthodontics and Orthognathic Surgery on Speech
- Conclusions

The physiologic and functional mechanisms of human speech, mastication, and swallowing are essential for an individual to maintain his or her normal performance levels. These are highly coordinated and complex actions that are closely interrelated. These functions are susceptible to physical impairments, such as the presence of a dentofacial deformity. This chapter will review the mechanisms of normal speech, mastication, and swallowing. The potential for negative sequelae that stem from the presence of a jaw deformity with malocclusion and the benefits of successful reconstruction are also reviewed.[11,151]

The Neuromuscular Masticatory System

The *masticatory system* is an integrated complex that is primarily made up of bones, muscles, ligaments, and teeth. Movement of the structures is neurologically coordinated for efficient function and for the maintenance of the component parts over the individual's lifetime.[1-3] Controlled contracture and relaxation of the head and neck musculature are necessary to move the mandible, the soft palate, the lips, and the tongue efficiently for effective function (i.e., speech, swallowing, and chewing). A neurologic control system regulates and coordinates the activities of the entire masticatory system.

The dynamic balance of muscles in the head and neck is made possible through feedback that is provided by sensory receptors.[10] To create a precise mandibular movement, input from the various sensory receptors is received by the central nervous system through the afferent fibers.[41] The brain stem and cortex work together to assimilate and organize this input, and then provide modulated motor activities through the efferent nerve fibers.[80] Within the brain stem, neurons control rhythmic muscle activities for effective breathing, swallowing, and chewing.[25] These neurons are called the *central pattern generator* (CPG).[96] The CPG is responsible for the timed and integrated activity among antagonistic muscles that is needed to accomplish these functions. For example, during chewing, the suprahyoid and infrahyoid muscles contract at the same time that the elevator muscles relax; this allows the mouth to open and accept food. With the bolus of food in the mouth, the CPG causes contraction of the elevator muscles while relaxing the suprahyoid and infrahyoid muscles, thereby producing closure of the mouth onto the food bolus. This chewing mechanism is repeated until the particles of food are small enough to be efficiently swallowed. All of this assumes that the individual has normal functioning of the tongue and lips, adequate numbers and locations of teeth, and intact temporomandibular joints and jaws (Fig. 8-1).[43] Ideally, with all of these component structures in place, chewing can be accomplished without excess stress to any of the component parts. Although chewing is typically a subconscious activity, it can be brought into conscious control at any time. Likewise, breathing and swallowing are generally carried out as subconscious activities, but they can also be brought under voluntary control to be refined (Table 8-1).



• **Figure 8-1** Mastication is the process of chewing food in preparation for deglutition (swallowing) and digestion. All muscles of mastication originate on the skull and insert on the mandible. All muscles of mastication are innervated by the mandibular division of the trigeminal nerve. All muscles of mastication are derivatives of the first pharyngeal arch. Movements of the mandible are classified as follows:

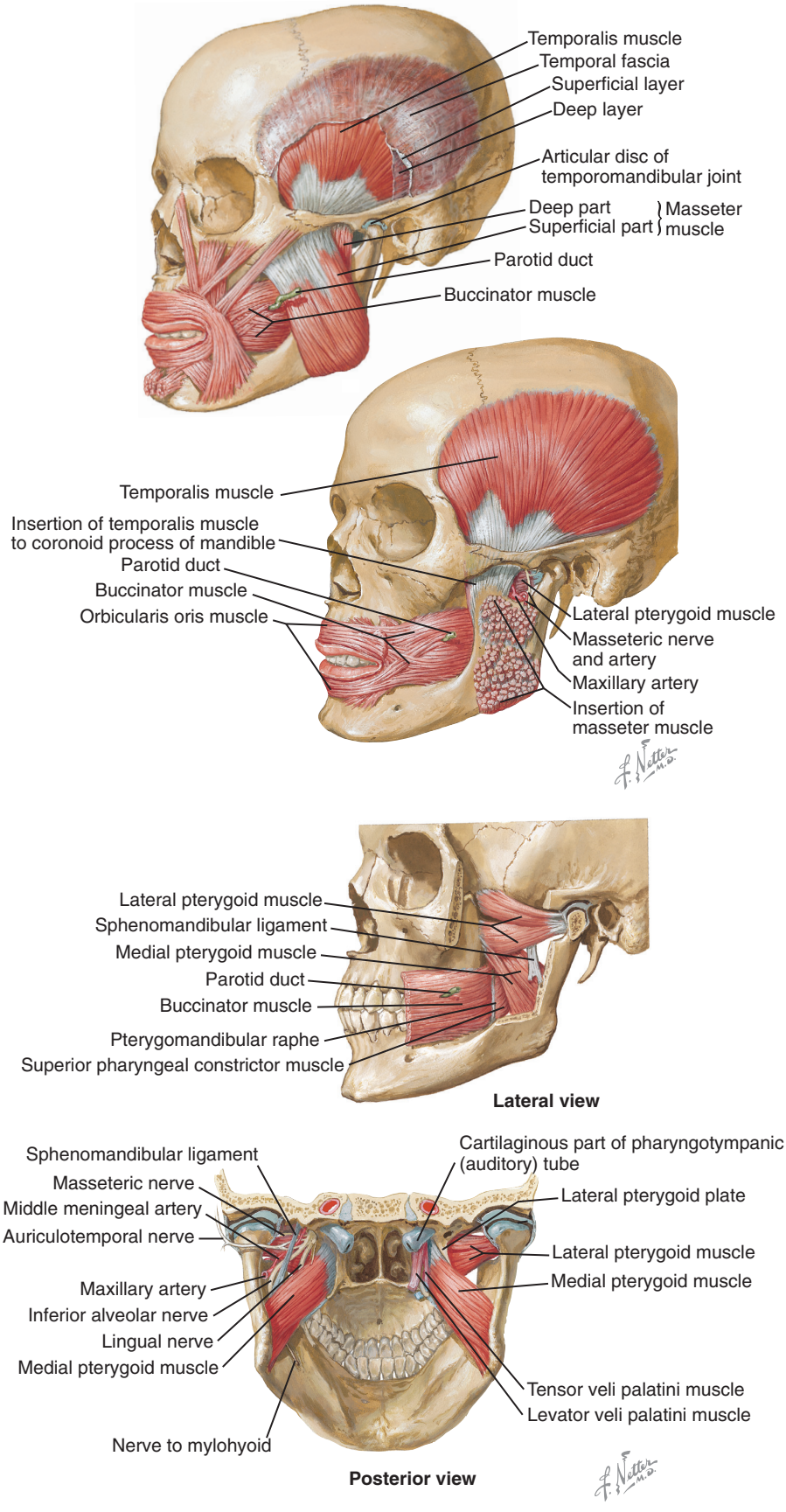
- Elevation
- Depression
- Protrusion
- Retrusion
- Side-to-side (lateral) excursion

The bones involved include the base of the skull and the mandible. The skull base and the mandible articulate at the temporomandibular joint between the squamous portion of the temporal bone and the condyle of the mandible.

The muscles involved in the masticatory process include four paired muscles:

- The masseter
- The temporalis
- The medial pterygoid
- The lateral pterygoid

Although the buccinators are not strictly considered muscles of mastication, they help to keep the bolus of food against the teeth during this process. The illustrations shown here demonstrate the masticatory muscles' bony insertions and locations of insertion as well as their overall anatomy. *Netter illustration from www.netterimages.com* *© Elsevier Inc. All rights reserved.*



• **Figure 8-1, cont'd**

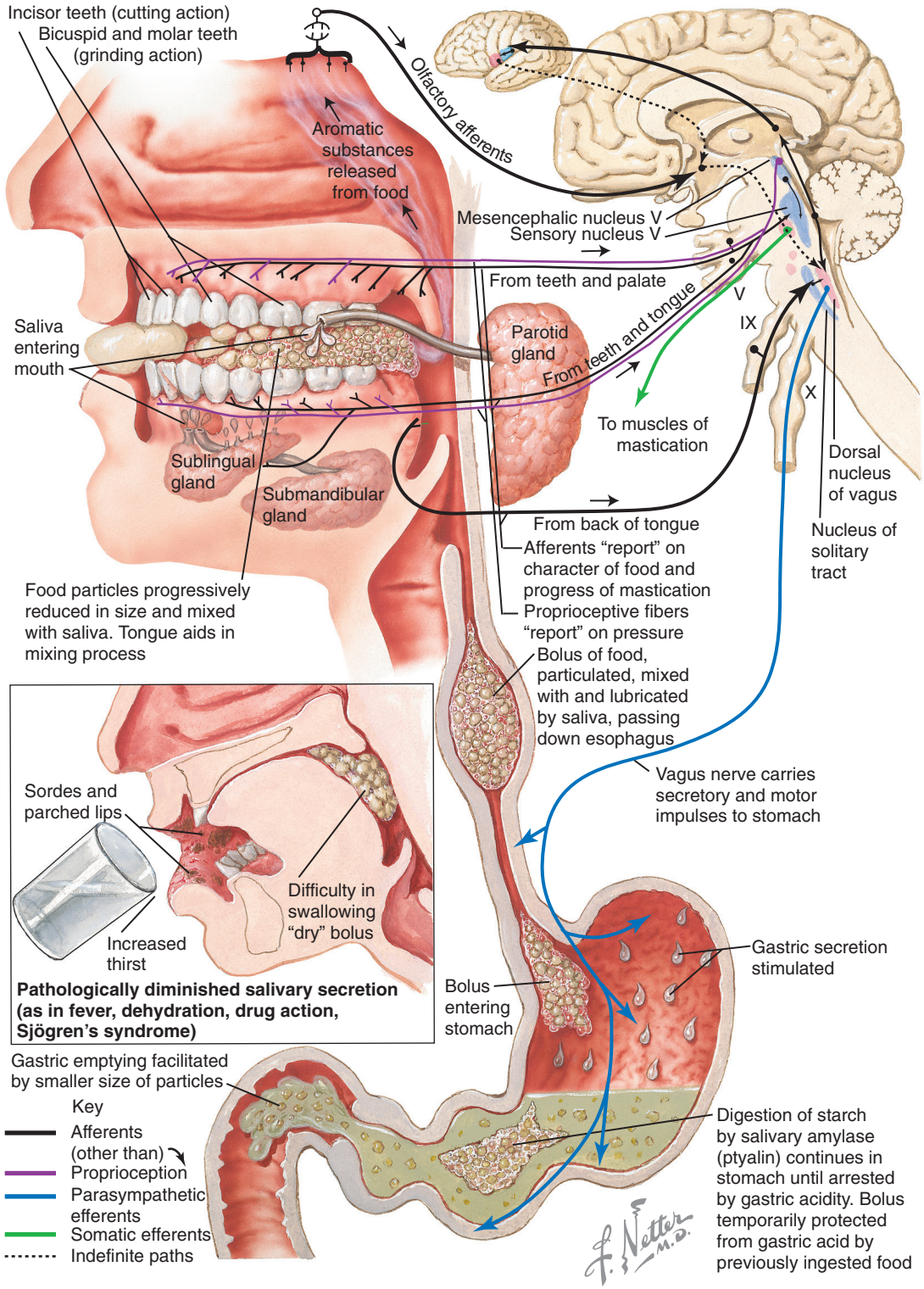
TABLE 8-1 Muscles of Mastication

Muscle of Mastication	Origin	Insertion	Main Actions
Masseter: superficial head	• Inferior border of the anterior two thirds of the zygomatic arch	• Angle of mandible • Inferior and lateral parts of the mandibular ramus	• Elevates mandible • Protrudes mandible • Aids in lateral excursion of mandible
Masseter: deep head	• Medial border of the zygomatic arch • Inferior border of the posterior one third of the zygomatic arch	• Superolateral mandibular ramus • Coronoid process	
Temporalis	• Temporalis fossa, along the inferior temporal line, including the temporal fascia	• Coronoid process, extending inferiorly on the anterior border of the mandibular ramus	• Elevates mandible • Protrudes mandible • Aids in lateral excursion of mandible
Medial pterygoid: deep head	• Medial surface of the lateral pterygoid plate	• Medial surface of the ramus and the angle of the mandible	• Elevates mandible • Protrudes mandible • Lateral excursion of mandible
Medial pterygoid: superficial head	• Maxillary tuberosity	• Medial surface of the ramus and the angle of the mandible	• Elevates mandible • Protrudes mandible • Lateral excursion of mandible
Lateral pterygoid: upper head	• Greater wing of the sphenoid • Infratemporal crest	• Articular discs and the capsule of the temporomandibular joint	• Depresses mandible • Protrudes mandible • Lateral excursion of mandible
Lateral pterygoid: lower head	• Lateral surface of the lateral pterygoid plate	• Pterygoid fovea on the neck of the condyle	• Depresses mandible • Protrudes mandible • Lateral excursion of mandible

Mastication Mechanism

Mastication is defined as the act of chewing food (Fig. 8-2). It represents the initial stage of digestion. During mastication, the food bolus is broken down into small particles for ease of swallowing. For most, it is considered an enjoyable activity that involves the senses of taste, touch, and smell. It is a complex function that requires the involvement of the muscles, the teeth, and the periodontal supportive structures. It also requires functioning temporomandibular joints and jaws, and it makes use of the lips, the cheeks, the tongue, the palate, and the salivary glands. It is an activity that is usually automatic and often taken for granted, although it can be easily brought into voluntary (i.e., conscious) control. It is also susceptible to pathologic conditions and damaging habits. Furthermore, modifications of this system (i.e., compensatory patterns) will be introduced when component parts are not in proper working order (e.g., in the presence of dentofacial deformity with malocclusion).

In the normal setting, mastication is made up of rhythmic, well-controlled separations and closures of the maxillary and mandibular teeth. This activity is under the control of the CPG, which is located in the brain stem. Each opening and closing movement of the mandible represents a chewing stroke.[79] The *chewing stroke* can be divided into the *opening phase* and the *closing phase*. The closing phase may be further subdivided into the *crushing phase* and the *grinding phase*. During mastication, the chewing stroke is repeated until the food is broken down into small enough parts for swallowing. After the food has been incised (usually with the anterior teeth) and then brought further into the mouth, the crushing of the bolus is concentrated on the posterior teeth.



• **Figure 8-2** Mastication prepares food via "chewing" for deglutition and digestion. The illustrations and legends show an overview of the process. *Netter illustration from www.netterimages.com* *© Elsevier Inc. All rights reserved.*

It has been documented that the tooth contact ability, the number of teeth, and the quality of the teeth influence the chewing stroke. During mastication, sensory information is sent back to the central nervous system.[5,158] This feedback mechanism allows for the alteration of the chewing stroke in response to the particular food being chewed and the quality of the anatomic structures that are available for chewing.[65] Generally, with maximum full intercuspation (i.e., tall cusps, deep fossae, and ideal contacts), a predominantly vertical chewing stroke is seen. When there is a flattened occlusal surface (e.g., as a result of occlusal equilibrium or a grinding habit that involves a loss of enamel), a broader chewing stroke can be documented.[54,81] The presence of malocclusion may produce irregular and less repeatable chewing strokes in the individual.[6] Studies document that, in approximately 75% of subjects, there is a preferred chewing side.[6] Chewing activities normally occur on the side with the greatest number of pain-free tooth contacts noticed during lateral glide.

The maximum biting force that can be applied to the teeth varies from individual to individual.[156] With tougher foods, chewing preferably occurs predominantly on the first molar and second premolar areas. In general, males can bite more forcefully than females. Typically, in the presence of normal anatomy, the maximal amount of force applied to a molar is several times that which can be applied to an incisor.[60] The maximum biting force generally increases with age, up to adolescence. Nevertheless, the amount of force placed on the teeth during mastication varies greatly from individual to individual.[103,114]

When food is introduced into the mouth, the lips must guide and control intake. After the food is in place, the lips must seal off the oral cavity. The lips are especially necessary when liquid is being introduced.[58] This is more difficult for the individual with a long face growth pattern that causes mentalis strain and lip incompetence. When the food is in the mouth, the tongue plays a major role in maneuvering the bolus for sufficient chewing. The tongue typically initiates the process of breaking up the food by pressing the bolus against the hard palate; thus, the presence of an oronasal fistula in a patient with a cleft palate can be problematic. The tongue then pushes the food onto the occlusal surfaces of the teeth, where it can be effectively crushed during the chewing stroke. During the opening phase of the next chewing stroke, the tongue repositions the partially crushed food again onto the teeth for further breakdown. While the tongue is repositioning the food that has been displaced lingually, the buccinator muscle in the cheek region is compressing the food that has shifted into the vestibule back over the molars. The displaced food is continuously repositioned on the occlusal surfaces of the teeth until the particles are small enough to be swallowed efficiently. The tongue is also used to separate the food particles that require more chewing (and are therefore replaced over the molars) from those that are now small enough for swallowing. This sorting process is essential to prevent choking on large food particles. After the eating process is complete, the tongue is used to clean the teeth and to remove any food residue that has been trapped in locations such as the floor of the mouth (i.e., the sulcus) or in the labial vestibule.

In the presence of normal anatomy, the ingestion of a bolus of food is made easier by the active lowering of the mandible, the opening of the lips, and the depression of the tongue.[25] All of these activities increase the size of the oral cavity to accommodate the bolus that is to be ingested. Not surprisingly, temporomandibular joint ankylosis or masticatory muscle pain with trismus will limit mouth opening and alter the process. The ingestion of fluid is usually via sucking (e.g., with a straw). In this case, the lips remain sealed around the delivery device, and the exit to the back of the oral cavity is closed by the tongue and the soft palate. In the individual with a long face growth pattern (i.e., lip incompetence and an anterior open bite), this will be more difficult. The lowering of the mandible and the depression and retraction of the tongue are accomplished by bracing the cheeks laterally and the mouth floor inferiorly. These actions will generate a subatmospheric pressure within the oral cavity to facilitate the flow of fluids into the oral cavity. This process will be more difficult in the presence of an oronasal fistula (i.e., an incompletely repaired cleft palate). This form of suction is also a useful mechanism for driving the entry of saliva into the oral cavity from the salivary glands.[112]

Swallowing Mechanism

Swallowing is a process that occurs through a series of coordinated muscular contractions that move the bolus of food from the oral cavity through the esophagus and into the stomach (Fig. 8-3). It requires voluntary, involuntary, and reflex muscular activity. The decision to swallow generally depends on the following: (1) the degree of fineness of the food (2) the intensity of the taste extracted and (3) the degree of lubrication of the bolus. During the swallowing mechanism, the lips are closed to seal the anterior aspect of the oral cavity. The teeth are brought into their maximum intercuspal position for the stabilization of the mandible. With the mandible fixed, the contraction of the suprahyoid and infrahyoid muscles will control the movement of the hyoid bone for effective swallowing.

The normal adult swallow involves the use of the teeth for mandibular stability; this is called the *somatic swallow.* When adequate teeth are not present (e.g., in the infant), the mandible must be braced by other means. In the infant swallow, which is also called the *visceral swallow,* the mandible is braced by placing the tongue between the dental arches.

In the normal growing child, as the posterior teeth erupt, the occluding teeth are able to brace the mandible, and the transition to an adult swallow occurs. In the presence of malocclusion, either from a developmental jaw deformity (i.e., an anterior open bite or a Class II malocclusion with significant overjet) or from the loss of teeth from either

tumor or trauma, the infant swallow is maintained or reassumed (Fig. 8-4). In the presence of an anterior open bite, it is necessary for the individual to thrust the tongue forward to close the gap during the swallowing mechanism. This is an appropriate *compensatory swallowing pattern*.

Studies confirm that this repetitive *swallowing cycle* occurs approximately 600 times during a 24-hour cycle in the average individual.[33] The general breakdown includes cycles during eating, cycles between meals while awake, and cycles during sleep. Lower levels of salivary flow during sleep result in less need for swallowing.[112]

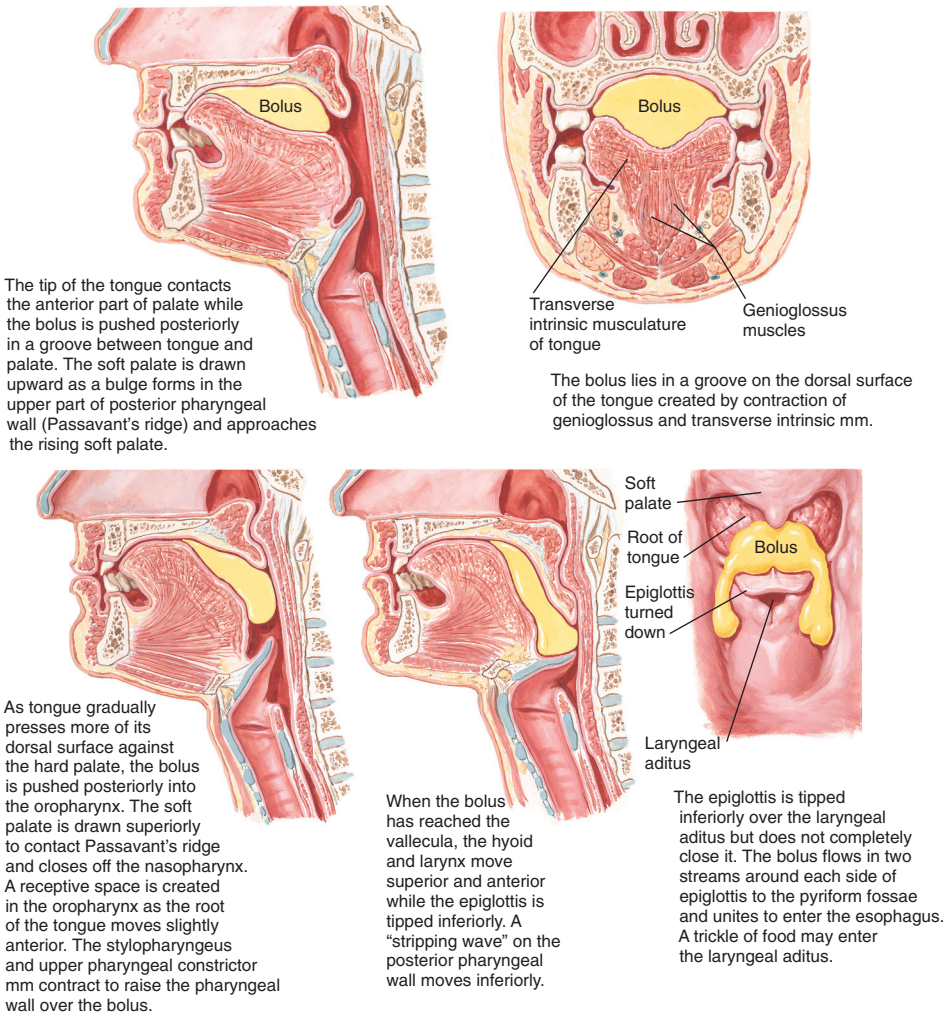
Although swallowing is one continuous act, for descriptive purposes, it is divided into three stages.

Swallowing: First Stage

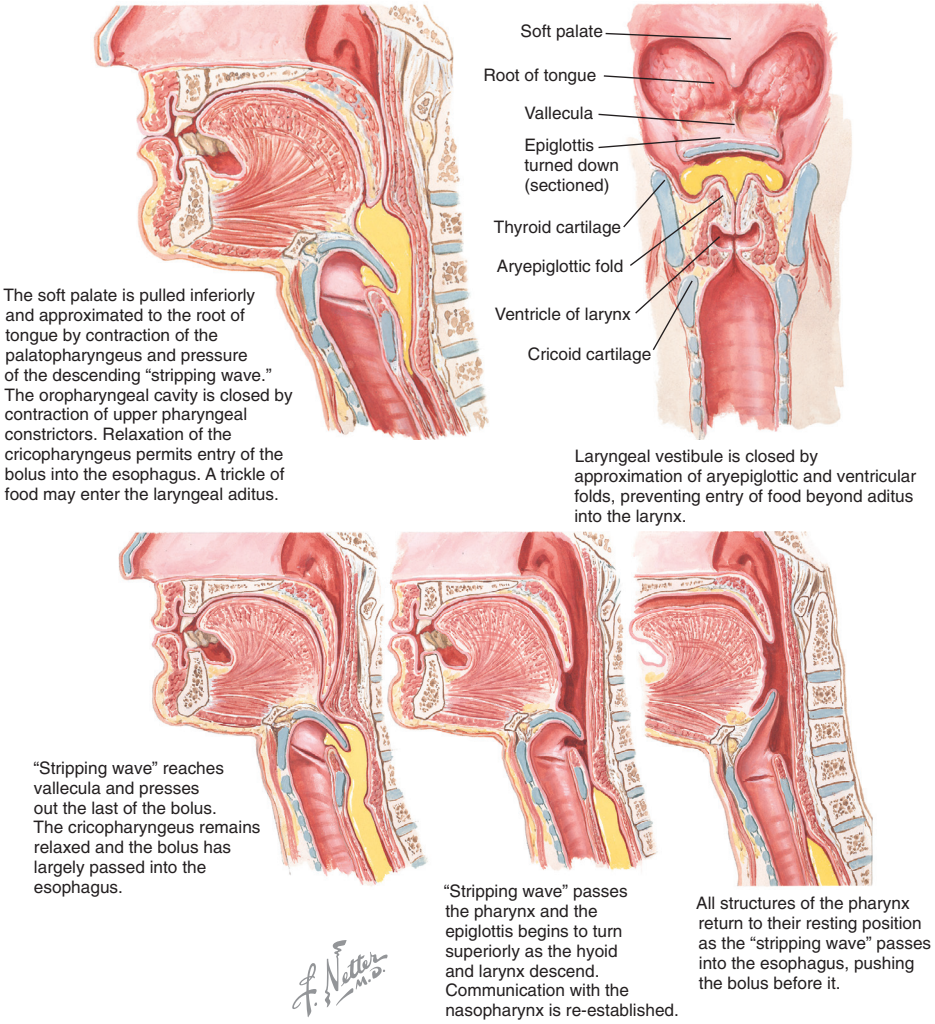
The first stage of swallowing is voluntary. It begins with the selective separation of the masticated food into a specific bolus. This task is accomplished primarily by the tongue. The bolus is positioned on the dorsum of the tongue and then pressed lightly against the intact hard palate. The tip of the tongue rests on the hard palate anteriorly, just behind the incisors. The lips are sealed, and the teeth are brought together; this essentially dams off the anterior oral cavity. The presence of the bolus pressed against the mucosa of the intact hard palate initiates a reflex wave of contraction of the tongue that pushes the bolus backward. As the bolus reaches the back of the tongue and with the soft palate now occluding against the nasopharynx, the bolus is transferred into the pharynx.

Swallowing: Second Stage

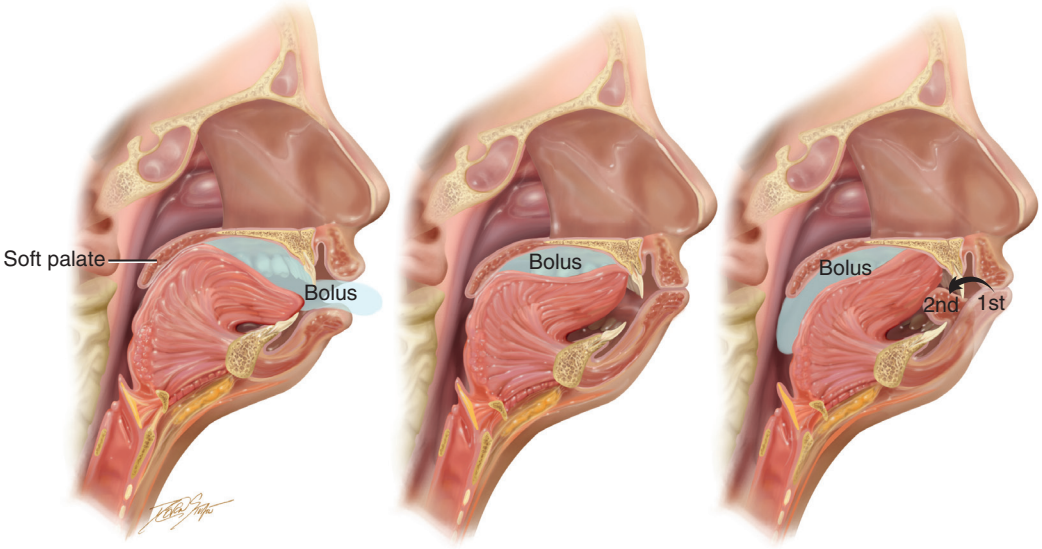
When the bolus has reached the pharynx, a peristaltic wave caused by the contracture of the pharyngeal constrictor muscles carries it down the pharynx. To prevent regurgitation into the nasal cavity, the soft palate must rise and touch the posterior pharyngeal wall, thereby sealing off the nasal



• **Figure 8-3** Deglutition or swallowing is a combination of voluntary and involuntary muscle contractions that move a bolus of food from the oral cavity to the esophagus. These illustrations review the details of the process. *Netter illustration from www.netterimages.com © Elsevier Inc. All rights reserved.* *Continued*



• **Figure 8-3, cont'd**



• **Figure 8-4** Illustrations of a compensatory swallowing mechanism in an individual with a Class II excess overjet malocclusion. This illustration is representative of the patient shown in Figure 8-10.

cavity. Simultaneously, the epiglottis must block the pharyngeal airway to the trachea to prevent the food from going into the larynx or the trachea and ending up in the lungs. This process of walling off potential openings keeps the food moving down the esophagus. During this stage of swallowing, the pharyngeal muscular activity opens the pharyngeal orifices of the eustachian tubes, which would otherwise be closed.

Swallowing: Third Stage

The third stage of swallowing describes the passing of the bolus through the length of the esophagus and into the stomach. Rapid peristaltic waves carry the bolus through the esophagus. As the bolus reaches the lower esophagus, the sphincter relaxes, thereby allowing for the entrance of the bolus into the stomach. Interestingly, in the upper portion of the esophagus, the muscles are mainly voluntary and can also be used to return the bolus into the mouth when necessary for more complete mastication. In the lower section of the esophagus, the muscles are entirely involuntary.

Evaluation of Swallowing

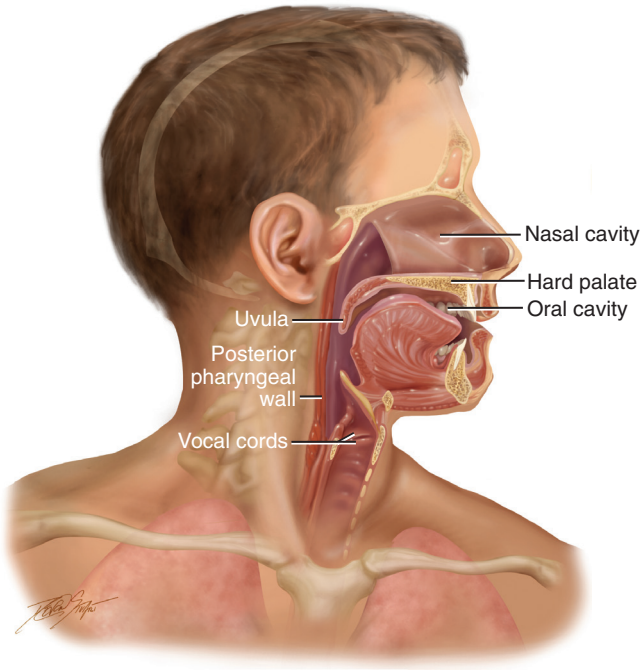
When a complaint about swallowing is registered, a special evaluation may be required. Such an evaluation should consist of a complete medical history, a detailed description of the complaint, and a physical examination of the peripheral deglutitory motor and sensory system, including trial swallows under observation. Diagnostic studies—including videofluorography, manometry, electromyography, and fiber-optic endoscopy—are indicated in selected cases.

Speech: Mechanism, Formation, and Assessment

The *mechanism of speech* is composed of several highly integrated processes (Fig. 8-5)[26,31,122]:

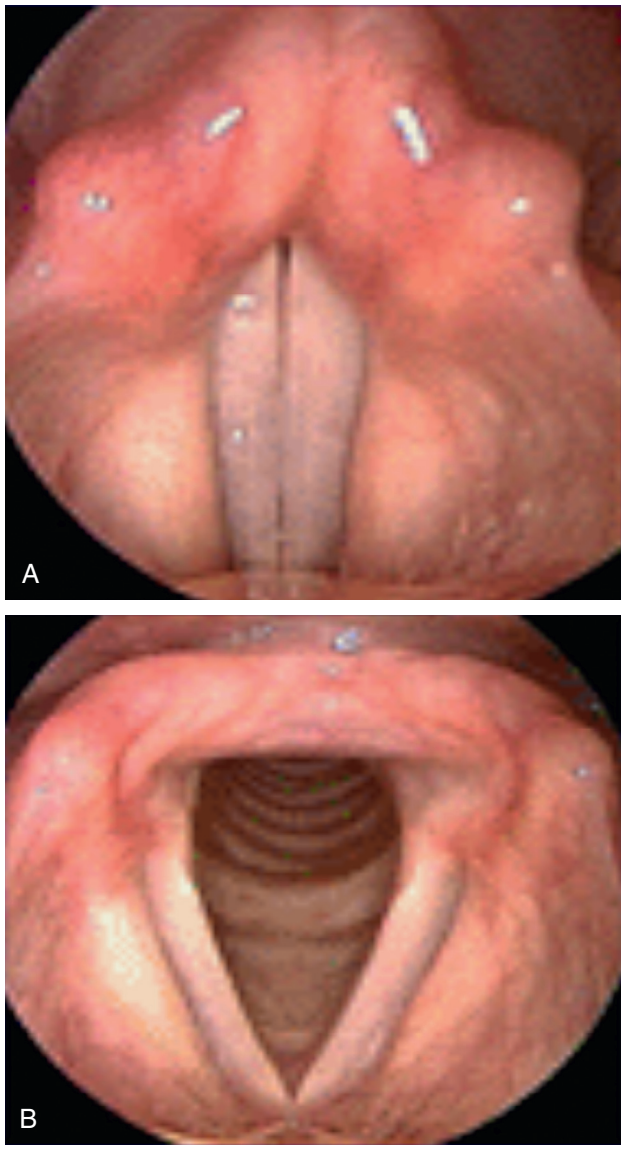
- *Speech initiation,* during which the content of the utterance to be spoken is converted into phonemes in the brain's language center
- The *generation of commands* that go from the brain's motor center to the speech organs
- *Phonation,* during which the emission of air sent from the lungs moves through the vocal tract and causes the formation of the voice as it moves past the vocal folds
- *Articulation,* which involves movement by the oral cavity, the oral pharynx, and the hypopharynx for the production of speech via this specific set of motor commands

During *human speech production,* the airflow that is sent from the lungs with a constant pressure passes between the vocal cords; the vibration of the vocal cords causes the airflow to be converted into cyclic puffs of air that then become sound (Fig. 8-6). The airstream is interrupted by movements of the jaws, the tongue, the soft palate, and the lips. These movements change the shape of the vocal tract, which in turn enables the individual to control the articulated sound and resonance characteristics.[4] The *pitch* of the voice changes in accordance with adjustments that are made in the tension of the vocal cords. *Voiced sounds* incorporate the vibration of the vocal cords in addition to the placement of the articulators. *Unvoiced sounds* are produced by the interruption of the airstream by the articulators. Voiced sounds are produced with vocal fold vibration that accompanies articulator placement. *Vocal tract resonance* characteristics are controlled by the cross-sectional area of various parts of the vocal tract.[36] Examples include the articulation of the upper lip to the lower lip for the bilabial sounds of /p/, /b/, and /m/ and the articulation of the tongue to the alveolar ridge for the production of the lingual-alveolar sounds /t/, /d/, /n/, /l/, /s/, and /z/.[120]



• **Figure 8-5** Illustration of a cross section through the head and neck that shows the pertinent anatomy required to articulate speech.

Individual articulators (i.e., upper jaw, lower jaw, upper lip, lower lip, tongue, soft palate, hard palate, upper teeth, and lower teeth) are used for both the speech mechanism (Figs. 8-7 and 8-8; Table 8-2) and for the mastication and swallowing (deglutition) mechanisms (see Figs. 8-1, 8-2, and 8-3).[123] *Speech sounds* are composed of the approximation of individual articulators within the speech mechanism and the manner in which the airflow from the vocal cords is modulated.[59] This in turn produces what is called a *phoneme,* which is known to most as an isolated speech sound. A significant feature of articulatory movements is the phenomenon of *assimilation* in which the mouth position for an *individual phoneme* in the *utterance*



• **Figure 8-6** A view of vocal cords as seen via nasoendoscopic examination. **A,** Closed vocal cords (adduction position). **B,** Open vocal cords (abduction position).

incorporates the effects of the mouth position for the phonemes uttered immediately before and after. Interestingly, a human can imitate the sound of an utterance that he or she has heard without necessarily being able to speak the language of the utterance.[66] In other words, the individual can estimate which mouth movements are necessary to produce a sound that is similar to the one that he or she has heard. By convention, the classification of speech sounds is categorized in accordance with their manner and place of production (i.e., the location of articulation).

Vocal tract variables include the dynamic voluntary movements of the *vocal organs* (e.g., lower jaw, tongue, soft palate, vocal cords, upper lip, lower lip); these organs and their movements are influenced by the basic positions of the organs in relation to each other. The baseline position of the jaws and teeth vary widely; however, if they are significantly displaced (i.e., if there is malposition of the teeth or jaws), this can negatively affect articulation (Fig. 8-9). When the position of a jaws is abnormal (i.e., dentofacial deformity) and then successfully corrected via orthodontics and orthognathic surgery, including a return of normal morphology, then the tongue may or may not respond instantaneously to achieve a more normal utterance of specific consonant or vowel sounds (see Fig. 8-8).

In general, in the patient with a dentofacial deformity, the articulators will try to adapt first to the genetically malpositioned jaws and teeth and then to the corrected anatomy after successful orthodontics and orthognathic surgery. It is said that the tongue is the single most important articulator, because it is highly mobile and therefore adaptable to the adjacent vocal organs for the production of a spectrum of speech sounds, even when the other articulators are abnormally located (e.g., as a result of the malposition of the teeth and jaws, a palatal fistula, a poorly moving velum, or widely separated lips). The *vocal folds* also have the capacity to adapt when speech pressure leaks out of the nose. The upper and lower lips, the soft palate, and the mandible are also mobile but to a more limited degree; therefore, they are less adaptable components of the vocal tract.

Speech sounds are generally divided into the categories of *consonant sounds* and *vowel sounds.* The production of consonant sounds requires the obstruction or partial closure (i.e., sphincteringe) of the airstream at specific sites along the vocal tract.[121] The classifications of consonant sounds are based on the extent of airstream obstruction, the site of airstream obstruction, and whether the vocal cords vibrate.[16,17] Vowel sounds are generally produced by the position of the tongue and lips without airstream obstruction and with the upper and lower teeth more widely separated. All vowel sounds are voiced, and it is for this reason that vowel sounds are generally less affected by the malpositioning of the teeth or jaws than consonant sounds are.[138]

With regard to the consonant sounds, the different extents of airstream obstruction are generally considered to fall into the following categories:

- *Plosive/stop:* Air pressure is stopped within the oral cavity at a specific location, and it is then suddenly released. Examples are /p/, /b/, /t/, /d/, /k/, and /g/.
- *Fricative:* Air pressure is partially blocked or obstructed within the vocal tract, thereby causing turbulent airflow. Examples are /f/, /v/, /s/, and /z/.
- *Affricative:* There is a sequencing of the airstream initially with complete obstruction (i.e., plosive/stop) followed by fricative (i.e., partial vocal tract obstruction) at the same location. Examples are /ch/ and /j/.
- *Nasal:* Air is allowed to pass freely through the nasal cavity. Examples are /m/, /n/, and /ng/.

The anatomic locations of the vocal organs that block or partially block the airflow (i.e., places of articulation) include the following: bilabial (upper lip to lower lip);

TABLE 8-2 Kinesiologic Positions for the Oral and Pharyngeal Musculature during the Articulation of Speech Sounds

Structural Components of the Articulatory Valve	Closed Valve Voiceless	Closed Valve Voiced	Constricted Valve Voiceless	Constricted Valve Voiced	Voiced Nasals
Labiodental (upper teeth and lower lip)			"f"/f/	"v"/v/	
Bilabial (lips)	"p"/p/	"b"/b/		"w"/w/	"m"/m/
Linguodental (tongue tip and upper teeth)			"th"/θ/	"th"/ð/	
Linguoalveolar (tongue tip and alveolar ridge)	"t"/t/	"d"/d3/	"s"/s/	"z"/z/ "l"/l/	"n"/n/
Linguopalatal (tongue blade and hard palate)	"ch"/tʃ/	"j"/dʒ/	"sh"/ʃ/	"zh"/ʒ/ "r"/r/ "y"/j/	
Linguovelor (tongue back and velum)					"ng"/r/
Linguo-velor-pharyngeal (tongue back, velum, and pharyngeal wall)	"k"/k/	"g"/g/			
Glottal (glottis)			"h"/h/		

• BOX 8-1

Speech Assessment Tests

- Templin-Darley Screening Test of Articulation
- Iowa Pressure Articulation Test
- Bzoch Error Pattern Diagnostic Articulation Test
- Fisher-Logemann Test of Articulation
- Goldman-Fristoe Test of Articulation

labiodental (lower lip to upper teeth); interdental (tongue between the upper and lower teeth); lingual to palate alveolar (tongue to alveolar process of the upper jaw), lingual to palate (tongue to hard palate); and glottal (vocal cords to each other).[124]

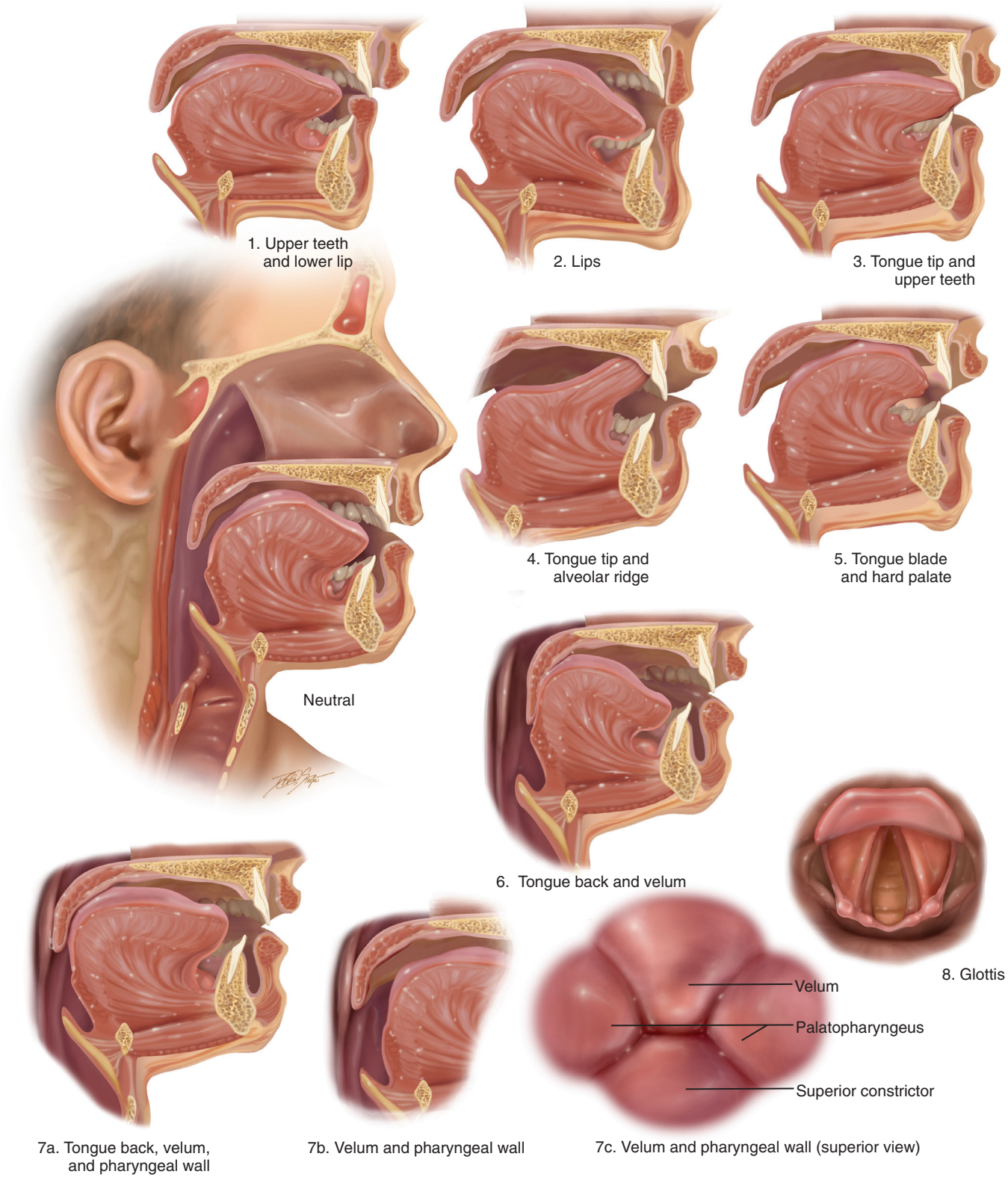
The assessment of speech disorders involves the use of a number of tests and techniques.[23,24,27,32,34,75,109,132,139] Historically, various tests have been described to assess speech and how it compares with recognized normative values. These assessments typically will evaluate sound production in the word position or within sentences. Tests that are frequently used for the assessment of speech are found in Box 8-1.

These tests are based on determining errors when the results are compared with those of normal individuals during single-word utterances. The Bzoch Error Pattern Diagnostic Articulation Test further grades each error type in accordance with its severity. All of these tests fall short for the assessment of the complexities of dynamic speech. Among educators and clinicians, there is often a lack of agreement with regard to a preferred objective method for the analysis of speech. The majority of the published studies were carried out to analyze the effects of orthodontics and orthognathic surgery on speech attempt to document articulation error rates in a traditional manner by noting sound production as simply correct or incorrect and by noting substitutions, distortions, or omissions. Some studies further describe errors as either visual or auditory in an attempt to overcome the usual problems associated with detecting mild sibilant distortions that might not be captured on audiotape.[161]

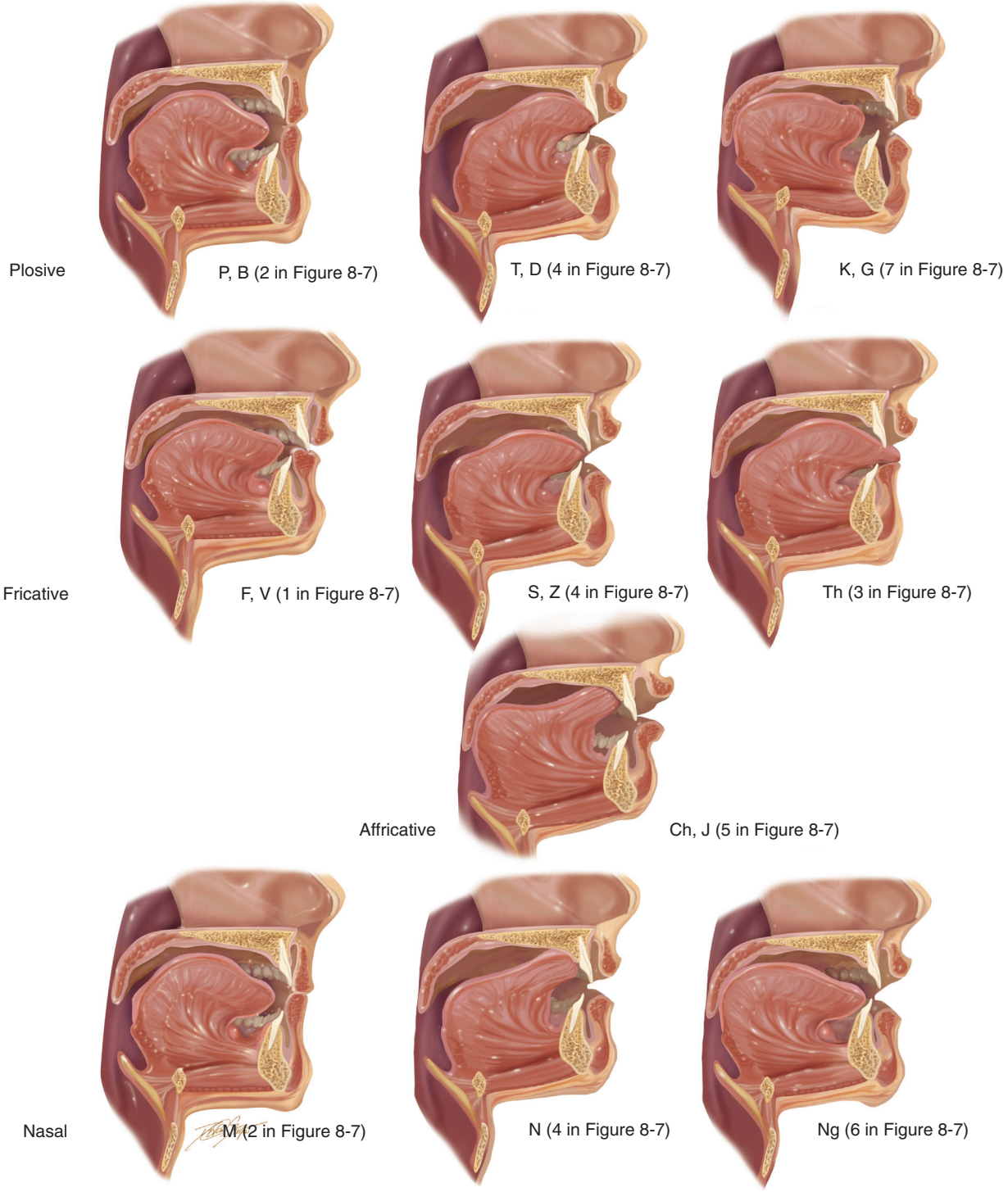
The classic negative effects of a jaw deformity with malocclusion on speech articulation are known.[30,35] The speech benefits of successful orthodontic and surgical correction will have their most notable effects on consonant articulation within the fricative class, which includes /s/, /z/, and /f/. It is known that the /s/ sound in particular is highly sensitive to precise tongue placement and to the accurate direction of airflow across the incisal edges of the upper and lower teeth.

The characterization of speech error type is usually defined as follows:

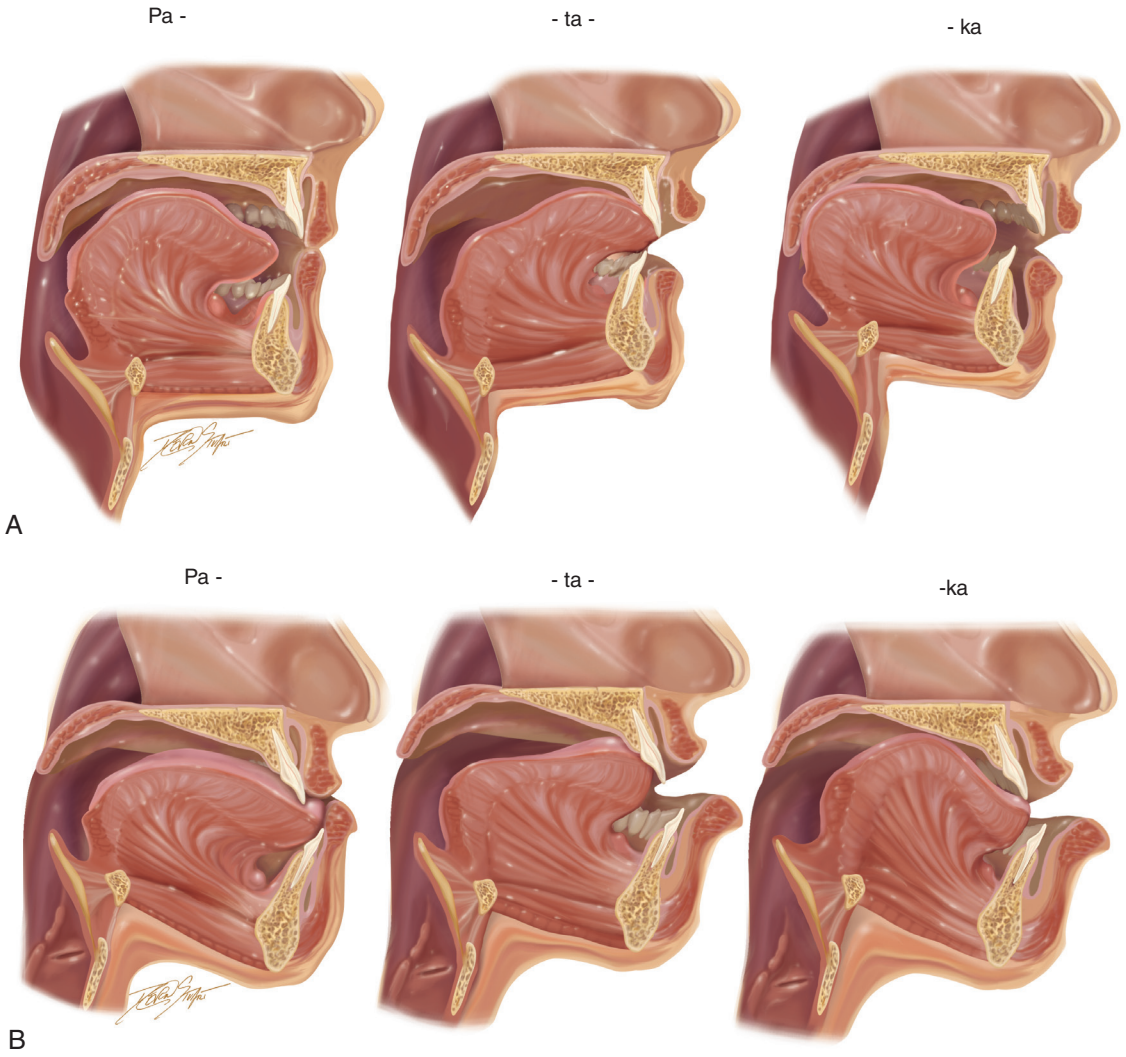
- *Substitution:* when one sound is replaced by another
- *Omission:* when a sound is omitted from a word altogether
- *Distortion:* when sound is not produced appropriately but is still understood
  - *Visual distortion:* when the visual appearance of making the sound is abnormal but the sound itself is correct
  - *Auditory distortion:* when the perception of the sound (what is heard) is abnormal but the appearance (visual appearance) of the positioning of the anatomy is correct



• **Figure 8-7** Diagram showing the kinesiologic positions for the oral and pharyngeal musculature during the articulation of speech sounds.



• **Figure 8-8** Illustrations that demonstrate the articulation of sounds created by specific positions of the lips, tongue, and teeth in the presence of harmonious jaws and occlusion. The numbering allows you to refer back to the chart shown in Figure 8-7, *B*, to correlate the anatomic position of the articulators with the associated speech sounds.



• **Figure 8-9 A,** Drawings that show the representative stages of lip, dental, and tongue positioning during the production of the word *patty-cake.* **B,** In the presence of a jaw deformity with malocclusion, the lips and tongue must position themselves in other locations (i.e., via compensatory mechanisms) to create similar sounds in an attempt to achieve effective communication. This is demonstrated in an individual with a long face Class III negative overjet anterior open-bite malocclusion and similar to what is seen in the patient in Figure 8-11.

- *Combined visual/auditory distortion:* when both the perception of the sound and the appearance of the production of the sound are abnormal

NOTE: Often speech errors become learned rule patterns by individuals on the basis of learned patterns of placement and manners of articulation. After successful orthognathic surgery, relearning may require professional help from a speech pathologist.

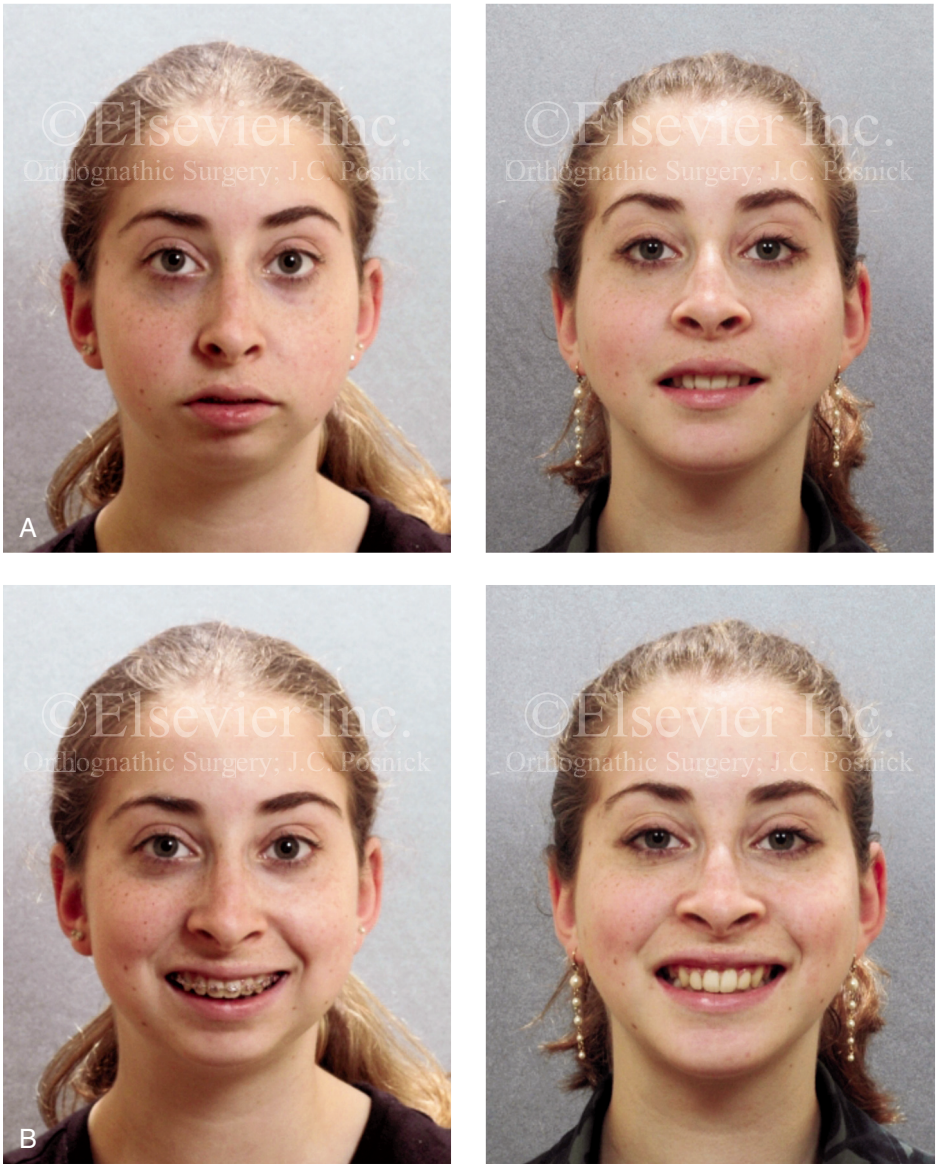
Effects of Jaw Deformities with Malocclusion on Speech

Background

Published studies show a tendency for individuals with Angle Class I occlusions to rate better with regard to speech as compared with those individuals with malocclusions.[37] A review of the literature shows a significant association between misarticulation in speech and Angle Class II malocclusion (Fig. 8-10), Angle Class III malocclusion (Fig. 8-11), and anterior open-bite malocclusion (Fig. 8-12).[39,43,53,54] Studies that evaluate speech articulation in individuals with jaw deformity and malocclusion confirm that as many as 90% of affected individuals have significant misarticulation errors. Several published studies report articulation errors among all jaw deformity patients analyzed.[39,47,53]

The jaw deformity patterns that are most frequently associated with significant articulation errors include those with 1) Angle Class II open-bite malocclusion in association with vertical maxillary excess and mandibular deficiency (i.e., the long face growth pattern; see Chapter 21) and 2) Angle Class III negative overjet malocclusion in association with maxillary deficiency and relative mandibular excess (see

Text continued on p. 252



• **Figure 8-10** A 16-year-old girl arrived with her parents for the surgical evaluation of a developmental jaw deformity with malocclusion. She was diagnosed with a primary mandibular deficiency, a maxillary arch constriction, and a Class II excess overjet growth pattern. Attempts at orthodontic growth modification and camouflage in the past were unsuccessful. There was a lifelong history of obstructed nasal breathing, heavy snoring, and the suggestion of sleep apnea. The patient experienced difficulty with chewing, swallowing, speech articulation, breathing, and lip closure/posture. A formal evaluation by a speech–language pathologist was carried out.

Chewing/swallowing:

- Difficulty with chewing solid, textured food
- Only able to chew on the back teeth
- Frequently swallows foods partially whole, especially raw vegetables and meat

Oral-sensory motor:

- Normal to mild labial weakness during repeated puckering and retraction tasks
- Lingual range of motion within normal limits
- Palatal structures revealed adequate elevation

Articulation:

- Assessed via the Fisher-Logemann Test of Articulation and informally during speech and conversation
- Misarticulations were identified, including /s/, /sh/, /st/, /z/, /dz/, and /sk/

Continued

• **Figure 8-10, cont'd**

Production of sounds:

- Anterior tongue thrusting is required to facilitate speech sounds in conversation and in contextual speech.
- Imprecision in speech makes her difficult to understand.
- She frequently uses a closed-mouth posture to speak, thereby causing her speech to be unintelligible for the listener.

Recommendations:

The patient's presenting jaw deformity and malocclusion interfere with her ability to communicate information precisely to a listener and to be understood. Orthodontic treatment and orthognathic surgery are recommended to correct the maxillofacial deformities. If successful, then improvements in the dysfunctions described should be expected. It is highly doubtful that speech therapy alone could solve the extent of the deficits.

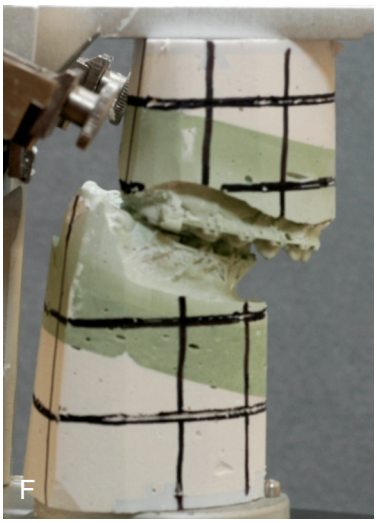
Treatment

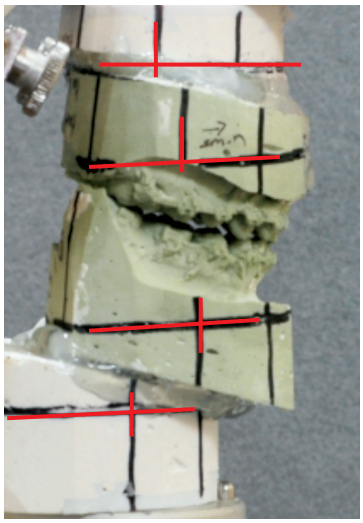
The patient agreed to a combined orthodontic and surgical approach. Mandibular first bicuspid extractions provided space to orthodontically relieve dental compensation. Surgery included a maxillary Le Fort I osteotomy (arch expansion and horizontal advancement); bilateral sagittal split ramus osteotomies (horizontal advancement); osseous genioplasty (horizontal advancement); and septoplasty and inferior turbinate reduction.

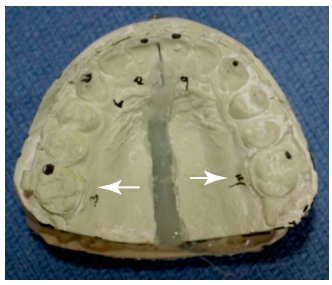
A, Frontal views in repose before and after treatment. **B,** Frontal views with smile before and after treatment. **C,** Oblique facial views before and after treatment. **D,** Profile views before and after treatment.

Pre surgery with lower bicuspid extractions

After treatment



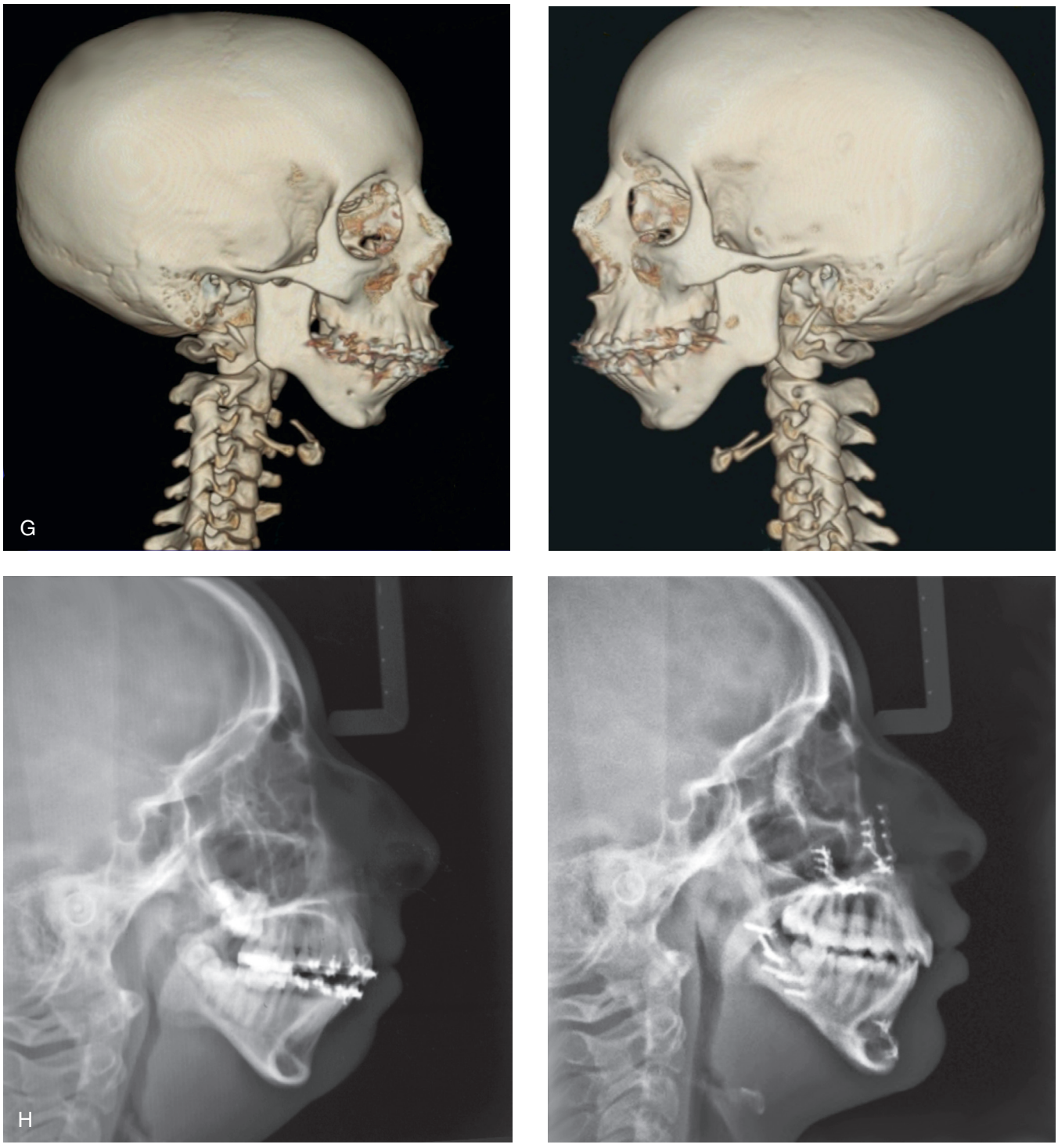




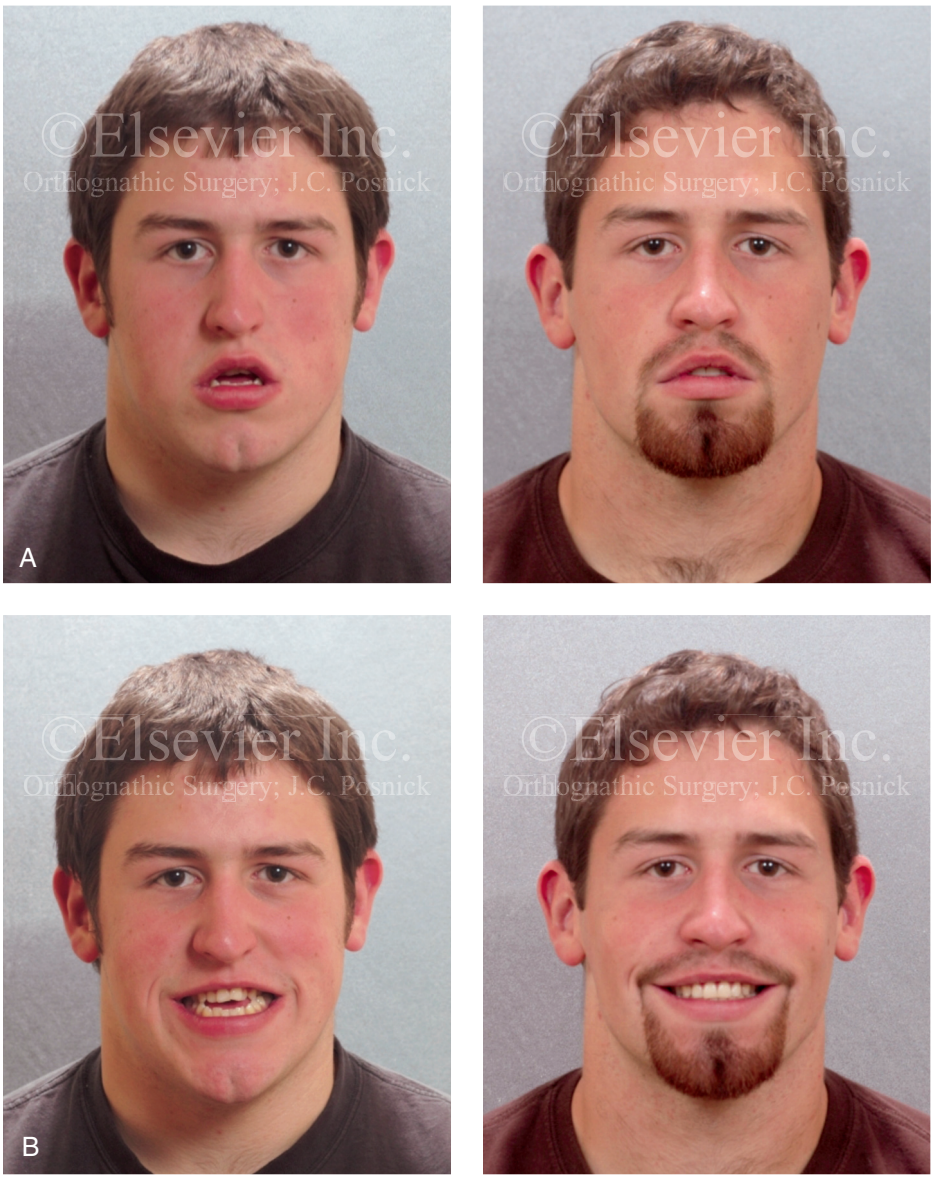
Arch expansion

• **Figure 8-10, cont'd E,** Occlusal views with orthodontics in progress (mandibular bicuspid extractions) and then after treatment. **F,** Articulated dental casts that indicate analytic model planning.

Continued



• **Figure 8-10, cont'd** **G,** Computed tomography scans prior to reconstruction confirming non-progressive glenoid fossa and condyle malformations. **H,** Lateral cephalometric radiographs before and after surgery.



• **Figure 8-11** An 18-year-old man arrived with his parents for the surgical evaluation of a developmental jaw deformity with malocclusion. He was diagnosed with a long face Class III anterior open-bite negative overjet growth pattern. This was due to both a hereditary Class III tendency and a lifelong obstructed nasal breathing open-mouth posture. The patient described difficulty with chewing, swallowing, speech articulation, breathing, and lip closure/posture. A formal speech–language pathologist's evaluation was carried out.

Chewing/swallowing:

- Frequent need to tear food apart before chewing
- Unable to bite into solid, textured food
- Only able to chew on the posterior teeth
- Frequently noted to swallow foods partially whole
- Difficulty with eating meats and breads

Oral-sensory motor:

- Labial motion and strength greatly reduced, especially with puckering and retraction of the tongue
- Soft palate structure and elevation within normal limits

Articulation:

- Assessed via the Fisher-Logemann Test of Articulation and informally during spontaneous conversation
- Misarticulations were identified, including /s/, /sh/, /z/, /dz/, /sk/, /ch/, and /s/ blends

Continued

• **Figure 8-11, cont'd**

Production of sounds:

- Production of sounds required anterior tongue thrusting and lateral lisping
- Exhibited imprecision in coarticulation, which often interfered with speech intelligibility
- Speech rate is rapid, with mumbling used as a compensatory feature for communication
- Frequently uses closed-mouth posturing with lack of enunciation, which greatly interfere with communication
- States that friends, teachers, and parents often comment that they cannot understand his speech
- Unintelligible speech interferes with daily performance at school and in social situations
- Avoids telephone conversations and hesitates to leave voice messages as a method of communicating

Recommendations:

The presenting jaw anomalies and malocclusion are responsible for the described findings. Orthodontics and orthognathic surgery are recommended to correct the maxillofacial anatomy. If successful, it is highly probable that the dysfunction described will resolve.

Treatment

The patient agreed to a combined orthodontic and surgical approach. With the removal of dental compensation, surgical procedures included a maxillary Le Fort I osteotomy (arch expansion, arch form correction, horizontal advancement, clockwise rotation, and vertical shortening); bilateral sagittal split ramus osteotomies (horizontal advancement and counterclockwise rotation); osseous genioplasty (horizontal advancement); and septoplasty, inferior turbinate reduction, and nasal floor recontouring.

A, Frontal views in repose before and after treatment. **B,** Frontal views with smile before and after treatment. **C,** Oblique facial views before and after treatment. **D,** Profile views before and after treatment.

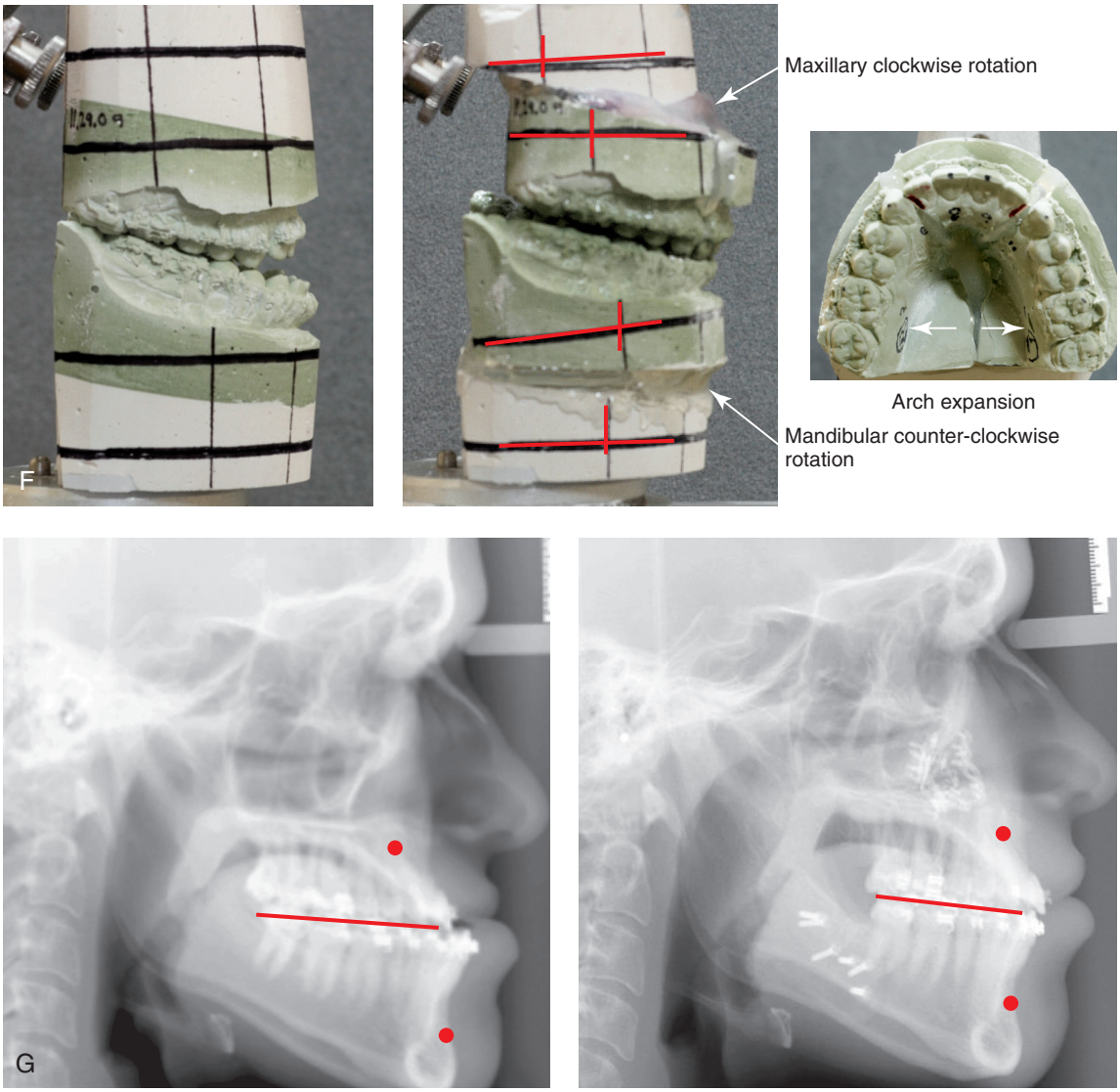
Prior to treatment

Pre surgery

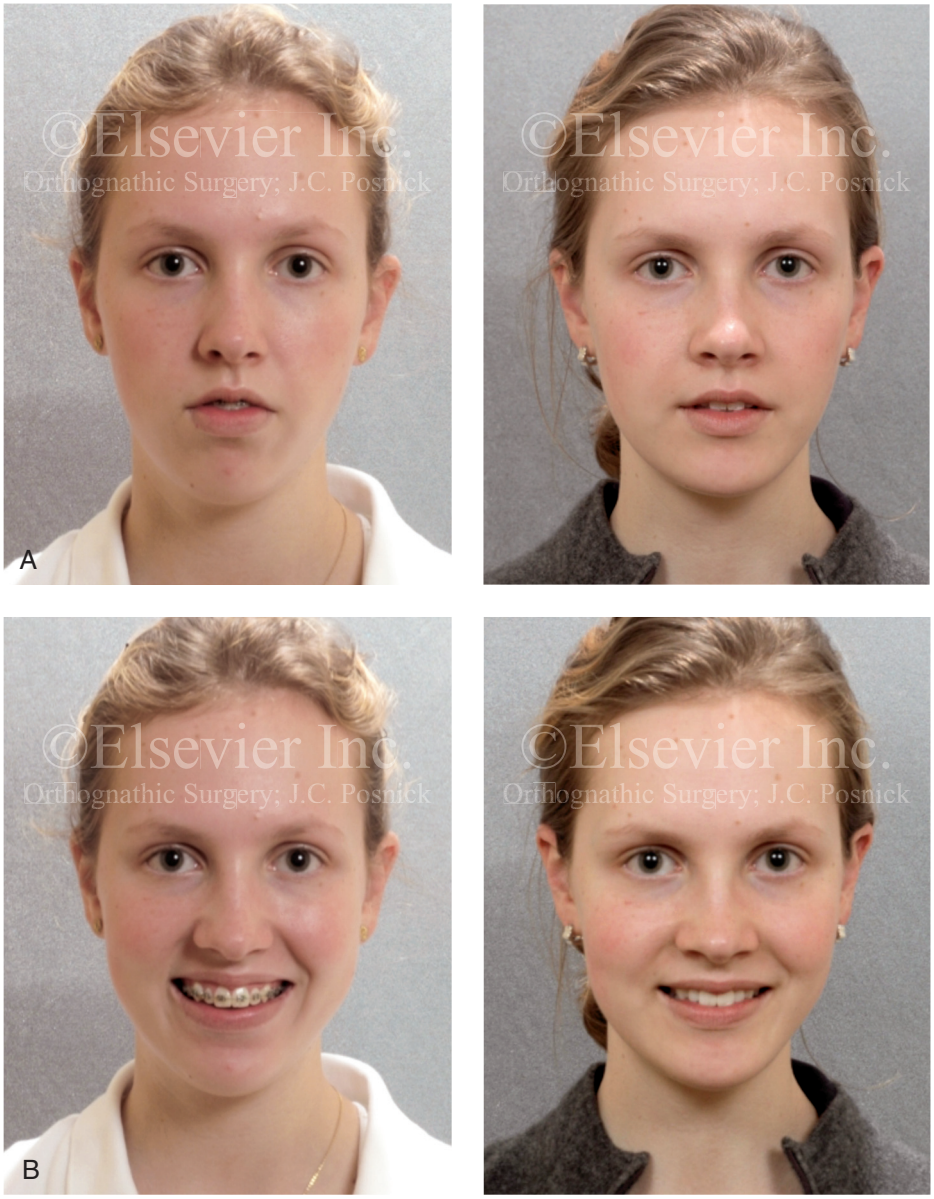
After treatment

• **Figure 8-11, cont'd E,** Occlusal views before treatment, with orthodontics in progress, and after treatment.

Continued



• **Figure 8-11, cont'd** **F,** Articulated dental casts that indicate analytic model planning. **G,** Lateral cephalometric radiographs before and after treatment.



• **Figure 8-12** A 16-year-old girl arrived with her parents for the evaluation of a developmental jaw deformity with malocclusion. She was diagnosed with a long face Class II anterior open-bite growth pattern. She had undergone years of orthodontic growth modification and camouflage treatment, without success. Her dentofacial deformity was found to affect speech, chewing, swallowing, breathing, and lip closure/posture. A formal speech–language pathologist's evaluation was carried out.

Chewing/swallowing:

- Only able to chew on the posterior teeth
- Malocclusion results in frequently swallowing food whole
- Noted to frequently have food falling out of her mouth secondary to malocclusion

Oral-sensory motor:

- Mild labial weakness
- Demonstrates anterior carriage of the tongue at rest
- Tends to hold oral cavity in a closed posture during speech

Articulation:

- Assessed via the Fisher-Logemann Test of Articulation and informally during conversation
- Misarticulations were identified, including /s/, /sh/, /z/, /dz/, /z/, /sk/, and /st/

Continued

• Figure 8-12, cont'd

Production of sounds:

- Sound production is with anterior tongue thrusting and lateral lisping during contextual speech
- Exhibits imprecise coarticulation, which interferes with speech intelligibility
- Speech rate is often fast, which compounds the difficulties with articulatory precision (i.e., perceived mumbling)
- Occasional final consonant deletions were noted

Recommendations:

The correction of the presenting abnormal jaw morphology and dental malposition is recommended, if feasible, through orthodontics and orthognathic surgery.

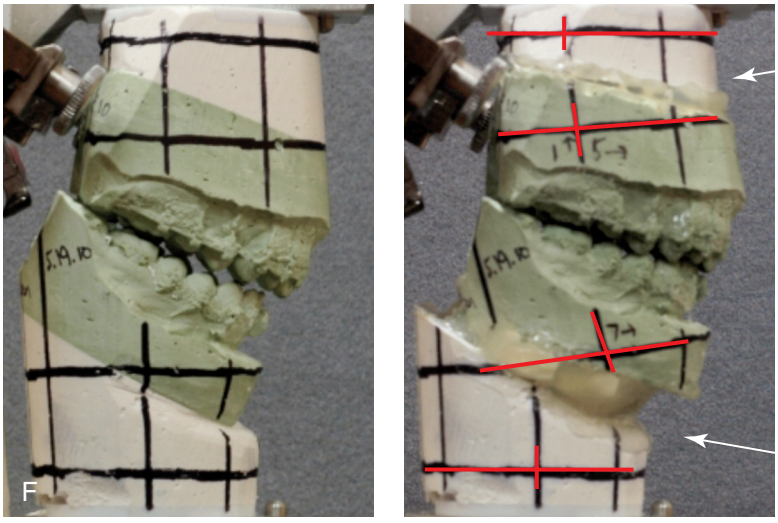
Treatment

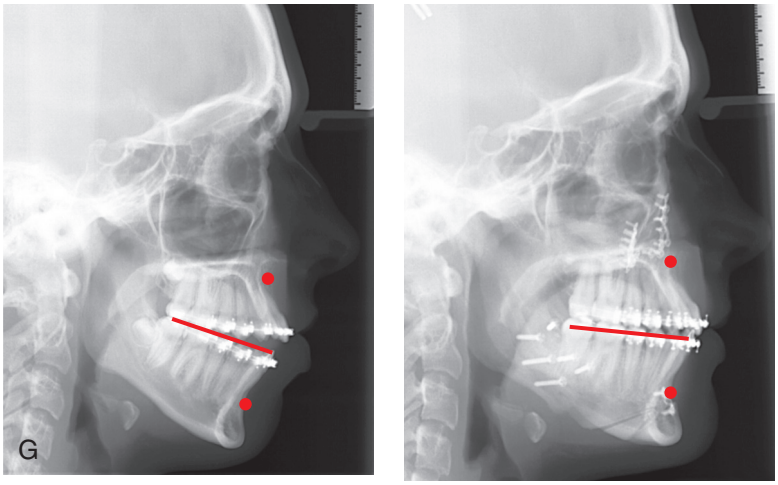
Further orthodontic (dental) decompensation in combination with orthognathic surgery was planned. The procedures included a maxillary Le Fort I osteotomy (vertical intrusion, horizontal advancement, and counterclockwise rotation); bilateral sagittal split ramus osteotomies (horizontal advancement and counterclockwise rotation); osseous genioplasty (vertical shortening and horizontal advancement); and septoplasty, inferior turbinate reduction, and nasal floor recontouring.

A, Frontal views in repose before and after treatment. **B,** Frontal views with smile before and after treatment. **C,** Oblique facial views before and after treatment. **D,** Profile views before and after treatment.

Pre surgery

After treatment





• **Figure 8-12, cont'd** **E,** Occlusal views before surgery and then after treatment. **F,** Articulated dental casts that indicate analytic model planning. **G,** Lateral cephalometric radiographs before and after surgery.

Chapter 20).[46,61,157] There were less articulation errors found in patients with Angle Class II primary mandibular deficiency malocclusions (see Chapter 19) and in patients with conditions that involve jaw asymmetry without significant malocclusion (i.e., mild hemifacial microsomia; see Chapter 28).[67] Voice disorders were more frequently seen with closed-bite malocclusion as compared with open-bite malocclusion. Closed-bite jaw deformities are generally associated with adequate overjet and often with a deep overbite (i.e., the short face growth pattern; see Chapter 23).[50]

Patients with Class III skeletal patterns show distinct differences with regard to the consonant fricatives (/f/ and /s/) as compared with normal controls.[73] Distortion type speech errors are also frequently associated with this type of malocclusion. This is especially true for patients with combined acoustic and visual errors. The speech sounds that are most commonly affected in the presence of malocclusion are sibilant sounds and bilabial (i.e., upper lip to lower lip) sounds. Errors in bilabial sounds are generally found in individuals with wide lip separation, such as those with maxillary deficiency in combination with mandibular excess or long face growth patterns (i.e., vertical maxillary excess and mandibular deficiency).[87]

Class II mandibular retrusion with excess overjet is likely to produce significant bilabial errors (see Fig. 8-10). Patients with Class II mandibular retrognathia also frequently show misarticulation errors (e.g., with the sound /r/). It is known that individuals with mild to moderate Class II mandibular retrusion are frequently able to adapt by posturing their lower jaw forward (i.e., by having a dual bite) to maintain control of their speech. This compensatory jaw position mechanism tends to break down with rapid speech or when the individual is otherwise fatigued.[130] As a result of the inherent anatomic barriers, it is not possible for patients with maxillary deficiencies or mandibular excesses with significant negative overjet to posture their lower jaw posteriorly as a method of speech compensation (see Fig. 8-11).[147] This makes the production of the /s/ sound difficult for most individuals with Class III malocclusions.[133]

Published reports indicate that, for a majority of the individuals studied, the orthodontic and surgical correction of the baseline jaw deformity and malocclusion either eliminates or dramatically reduces articulation errors.[95,97,106,107,113,127,146] A minority of the study patients report little change and occasionally speech deterioration. This lack of success in some patients is most likely attributable to 1) initial errors in dental or jaw diagnosis; 2) the incomplete initial correction of the dental or jaw deformity 3) or long-term skeletal or dental relapse with a return of the abnormal dental or jaw position. The inability of an individual with normal neuromotor ability to favorably adapt to the corrected dental and jaw relationships is not the most common reason for a lack of speech improvement. Nevertheless, some individuals will be in need of speech articulation retraining after corrective surgery and orthodontics.

Unfortunately, many of the published studies that set out to evaluate speech aspects after the orthodontic and surgical correction of the initial jaw deformity or malocclusion include too few subjects, have inadequate controls, mix varied patterns of dental and jaw deformities, and lack a standardization of the sampling times before and after treatment.* Some of the published studies seem to lack an understanding of the expected postoperative convalescence, during which short-term speech disability is to be expected. Definitive speech evaluation should occur after adequate healing (i.e., 3 to 6 months) and with documentation of the level of success of the correction of the jaw deformity and malocclusion. As a result of these study shortcomings, there is a need for the further investigation of the speech effects of jaw and dental corrections with 1) larger samples of homogenous patterns of jaw deformity and malocclusion 2) standardized treatment protocols 3) the confirmation of the actual correction of jaw dysmorphology and malocclusion 4) the setting of agreed-upon speech sampling parameters 5) interobserver and intraobserver error testing of speech evaluations and 6) appropriate statistical analysis.

Cause and Effect Relationships

If the upper and lower lips cannot easily close together—as is frequently observed in patients with Angle Class III negative overjet skeletal patterns (e.g., maxillary deficiency with relative mandibular excess; see Fig. 8-11) or in those with classic long face growth patterns with Class II anterior open-bite malocclusion (e.g., maxillary vertical excess with mandibular deficiency; see Fig. 8-12)—then bilabial sounds (e.g., words such as *pill, baby,* and *man*) cannot be normally formed.[55,95,97,104,136] These sounds will then be attempted through labiodental articulation. If the lower lip cannot contact the upper incisors (e.g., long face growth pattern with Class II anterior open-bite malocclusion or maxillary deficiency with mandibular excess negative overjet), then labiodental sounds (e.g., words such as *five* and *vein*) will be affected. Compensation will be attempted with bilabial sounds. If the tongue has difficulty reaching the lingual alveolar ridge of the maxilla (e.g., with moderate to severe Class II mandibular retrognathia), if the airstream cannot be correctly directed to the edges of the incisors (e.g., with an anterior open bite), or if the tongue is too far from the anterior aspect of the lower jaw (e.g., with Class III mandibular excess), then sibilant sounds (e.g., words such as *sun, zoo, ship, chair, judge,* and *measure*) will be affected (see Fig. 8-10).

Interestingly, from a speech perspective, some individuals are able to adapt to their dental or jaw defects more effectively than others. The individual's ability to alter the airflow over the incisors or through other articulation points is necessary to effectively compensate for anatomic

*References 8, 21, 18, 28, 29, 40, 42, 44, 45, 74, 106, 113, 126, 127, 131, 134, 135, 137, 146, 148, 150

variations in the teeth and jaws. Marked deformities such as severe Class III maxillary deficiency (see Fig. 8-11), severe long face growth pattern (i.e., anterior open bite with mentalis strain; see Fig. 8-12), and severe Class II mandibular retrognathia with the inability to posture the jaw forward (see Fig. 8-10) are examples of situations in which compensation is not possible and speech articulation is noticeably affected. With successful orthodontics and jaw surgery, improved positioning of the teeth, jaws, lips, tongue, and soft palate results in ease of articulation to accomplish effective speech.[28,74,126,131,137]

It is not surprising that vowel sounds are less frequently disturbed by a jaw disproportion or malocclusion. Physiologically, the major difference between vowel sounds and consonant sounds is that consonant sounds require significant constriction of the airflow within the vocal tract, whereas vowel sounds are formed by the tongue being positioned to only partially constrict the airflow.[143] Therefore, *consonant sounds* are more dependent on an exact positioning of the teeth with respect to each other and the lips with respect to each other (i.e., complete valve closure). Consonant sounds are more affected by jaw deformities such as Class III maxillary deficiency; long face anterior open bite; and severe Class II mandibular retrusion. These abnormal skeletal and occlusal patterns significantly interfere with the production of speech sounds. However, each individual's speech system has variable adaptive ability. The adaptability primarily comes from the stretching of the lips (i.e., mentalis strain); the stretching of the tongue to extended positions; the posturing of the lower jaw forward (i.e., centric relation to the centric occlusal slide); and the ability to alter the airflow force across the valves and sphincters. The ability of an individual to control these factors is also dependent on the speed of speech and the amount and length of continuous speech required.

Effects of Successful Orthodontics and Orthognathic Surgery on Speech

Effects on Speech Articulation: Review of the Literature

A study by **Witzel and colleagues** examined the articulation of 41 individuals before and after orthognathic surgery that was carried out to improve facial aesthetics and to correct the occlusion.[150] The investigators found a direct relationship between the pretreatment degree of mandibular retrognathia and the confirmed speech articulation errors. Twenty-two of 29 (76%) of the study patients had documented articulation errors before surgery; these included errors in sibilant production, labiodental sounds, and bilabial sounds. After surgery, significant improvement in the mean total articulation score for each patient was documented. There was also significant improvement in the sibilant sounds for all groups and in the mean bilabial scores of patients with mandibular retrognathia. Individuals with a Class III skeletal pattern and significant negative overjet (e.g., maxillary deficiency with relative mandibular excess) achieved the maximum improvement in labiodental scores after successful orthodontic and surgical correction.

Ruscello and colleagues studied the speech characteristics of 20 individuals who were scheduled to undergo orthognathic surgery for the correction of a variety of dentofacial deformities.[106,107] They underwent speech evaluation before and at intervals for up to 6 months after surgery. Speech assessment at each interval included the Templin-Darley Screening Test of Articulation, a 60-item nonsense syllable task, the Deep Test of Articulation, and the Rainbow Passage. Sixty percent of the patients with jaw deformities demonstrated preoperative articulation errors. The majority of those who were exhibiting preoperative articulation errors had documented improvements after surgery. None of the patients experienced a deterioration in their articulation after surgery at the close of the study.

Kummer and colleagues studied 16 skeletal Class III patients before and after Le Fort I maxillary advancement.[71] Seven of the 16 study patients were adolescents with repaired cleft lip and palate. Each patient underwent preoperative and postoperative speech evaluations and a multi-view videofluoroscopic speech study. Patients were evaluated before surgery as well as 3 and 6 months after surgery. The Templin-Darley Screening Test of Articulation was given, and articulation, resonance, and nasal emission were judged at each time interval. Eleven of 16 patients (69%) demonstrated articulation errors before surgery. After surgery, 7 of these 11 (64%) patients (4 without clefts and 3 with clefts) showed a decrease in the number of misarticulation speech sounds. None of the patients showed deteriorations in articulation after surgery.

Vallino studied the articulation, voice, resonance, hearing sensitivity, and middle-ear function of 34 individuals before and at intervals (i.e., at 3, 6, 9, and 12 months) after they underwent orthognathic surgery.[129] Thirty of the 34 patients (88%) exhibited articulation errors before surgery. The most frequent sibilant errors were /s/ and /z/, followed by /j/, /zh/, /ch/, and /sh/. The errors were predominantly distortions of both the visual and acoustic types. After surgery, articulation generally improved spontaneously, without the need for speech training or intervention. Most of the preoperative articulation errors were eliminated by 3 months after surgery. In a minority of the study patients, a gradual decline in some of these improvements was measured at 12 months after surgery. This could be explained by either a relapse of the initial satisfactory occlusion and jaw correction or a limited sustained adaptation of speech ability in some individuals. Interestingly, voice, resonance, and hearing sensitivity were not altered by the surgeries that were carried out.

Xue and colleagues carried out a study to compare vocal tract configuration in males with skeletal Class III malocclusion (n = 8) with that of their normally developed Class I counterparts (n = 8).[157] The goal of these researchers was to investigate the concomitant acoustic changes caused by any alterations in the vocal tract configuration that were

identified. The findings of the study confirmed that young adolescents with Class III malocclusion had different vocal tract configurations than their counterparts with normal occlusion. The differences occurred in the oral cavity only. The individuals with Class III malocclusion had significantly longer oral length and larger oral volume than their counterparts in the control group. They surmised that the Class III dentofacial deformity restricted lip protrusion during the production of /u/, thereby contributing to the significantly higher resonating frequency (first formant or F1) of /u/ as compared with that of normal controls.

A review of these studies indicate that a majority of individuals who present with moderate or severe jaw deformity and malocclusion will demonstrate articulation errors during speech. The pattern of jaw deformity (i.e., Class II, Class III, open bite, or long face) will predict the pattern of specific articulation errors. Some individuals will have a greater ability to adapt to their dentofacial deformity to achieve relatively normal articulation.

Distortions are the most frequently reported errors, which occur in association with malpositioning of the jaws and teeth. They are of more concern than substitutions or omissions. Individuals will attempt to adapt, but their speech production will not always be within acceptable phonetic boundaries. The most frequent speech sound errors are of the /s/ speech sounds, which are sibilants produced in the alveolar part of the palate with the tongue resting in this region. This type of sibilant distortion error can occur with a spectrum of jaw deformities (e.g., mandibular retrognathic Class II).

NOTE: Residual misarticulations may become learned behavior patterns. If this occurs, a few speech treatment sessions may be required for complete correction during contextural speech production. We have found that adults more frequently than adolescents benefit from speech therapy sessions after successful surgery and orthodontics.

In general, individuals who present with articulation errors caused by dentofacial deformity with malocclusion are expected to achieve improved speech function after successful jaw surgery and orthodontics. This assumes that the individual articulators are placed in sufficient proximity to each other. Published studies indicate that 80% to 90% of individuals will find positive improvement in their articulation after the vocal organs are correctly positioned. Interestingly, approximately 10% of individuals are found to be either unchanged or to show some negative effects in speech articulation after jaw surgery. Possible explanations for this small failure rate include errors in jaw or dental diagnoses; unsatisfactory surgical or dental execution; skeletal or dental relapse; effects of neurosensory deterioration; or the inability of the individual to adapt over the long term.

Effects on Velopharyngeal Function: Review of the Literature

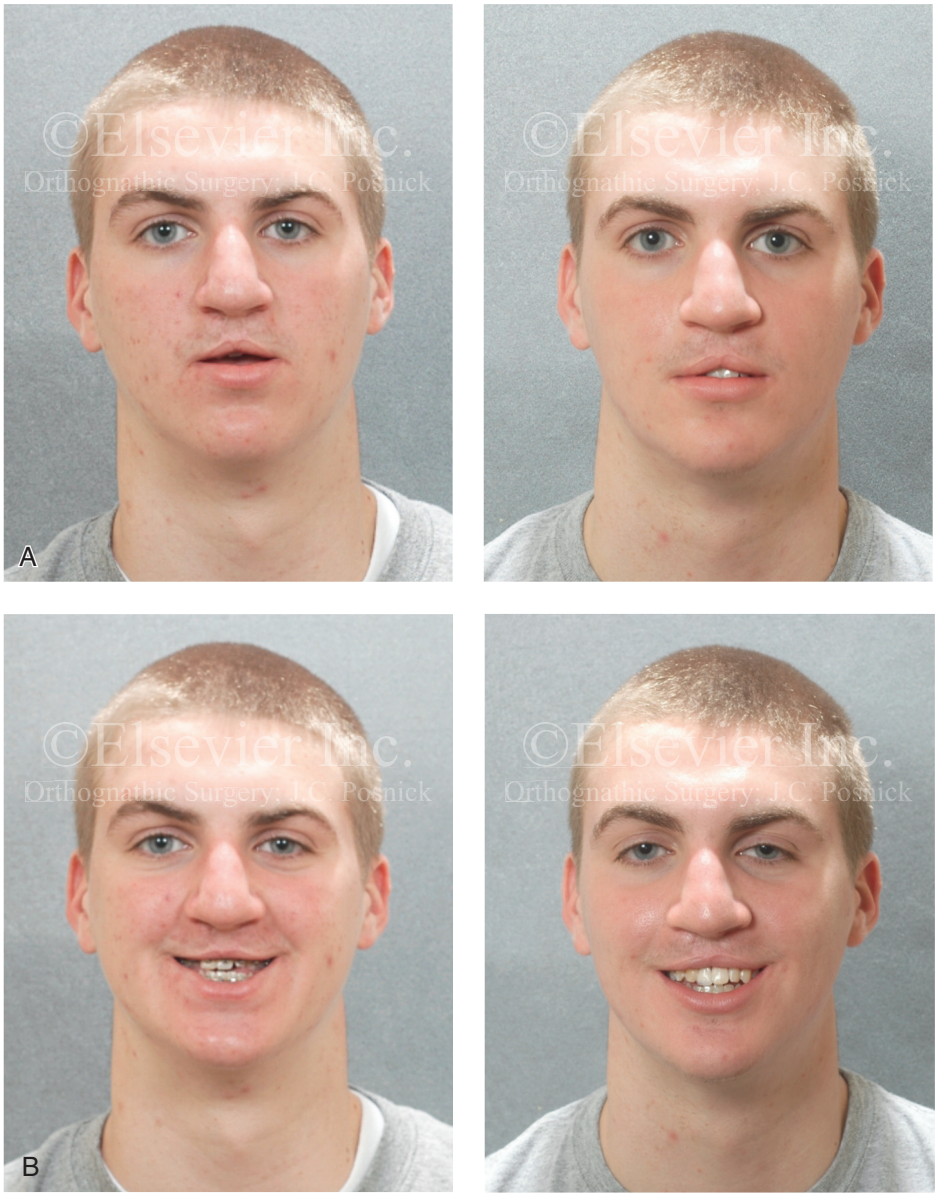
Velopharyngeal insufficiency (VPI) is the hallmark sign of the negative speech effects among individuals who are born with cleft palates.[7,12,22,52,76,91] The basic etiology of VPI in the repaired or unrepaired cleft palate is dysfunction of the soft palate (Fig. 8-13).[13,19,20,51,101,111] The muscular anatomy that is affected by clefting includes both the elevators of the soft palate (i.e., the musculus uvulus and the levator veli palatini) and the antagonists to the elevators (i.e., the palatoglossus and the palatopharyngeus).[56,69,70,116-118,145,160] VPI involves the inadequate closure of the velum to the pharyngeal walls while speaking.[78,108,141,142] VPI results in increased nasal air escape with increased nasal resonance that can be heard.[72,88,159] Resonance disorders include both hypernasality and hyponasality.[77] When there is improper control of airflow across the velopharyngeal (VP) sphincter, hypernasality can occur, with increased nasal resonance occurring during the production of vowel sounds.[84,153,154] Hyponasality is defined as a reduction in normal nasal resonance as a result of the blockage of airflow within the nasal cavity.[92,104] A review of the literature is helpful to better understand the effects of Le Fort I osteotomy with advancement on VP function.*

In 1969, **Jabaley and Edgerton** were the first to report about the potential for changes in the spatial relationship of the soft palate to the pharyngeal wall as a result of maxillary Le Fort I advancement.[63] They described an 18-year-old patient without clefting who underwent a Le Fort I osteotomy with advancement. They stated that there was "no adverse alteration of velopharyngeal function accompanying the maxillary advancement."[63]

In 1976, **Schwarz and Gruner** reported about 40 patients who were undergoing Le Fort I osteotomy with repositioning (31 out of 40 with repaired cleft palate).[113] The authors concluded that the "subjective evaluation of velopharyngeal function and nasality in the cleft palate patients showed no relationship between the degree of hypernasality before or after the Le Fort I advancement."[113]

Witzel and Munro discussed a 16-year-old patient with a repaired unilateral cleft lip and palate who presented to them with maxillary hypoplasia and malocclusion.[149] Before surgery, this patient's VP valving mechanism showed only touch closure of the soft palate against the posterior pharyngeal wall. Two months after Le Fort I osteotomy (10 mm of advancement and 4 mm of vertical lengthening), his speech evaluation revealed marked hypernasality throughout connected discourse. The authors concluded that "maxillary advancement may have a disastrous effect on the velopharyngeal valving mechanism."[149] They recommended that "all patients undergoing this procedure should have detailed clinical investigation of speech and velopharyngeal function pre- and postoperatively."[149]

*References 9, 14, 15, 38, 45, 47-49, 64, 68, 71, 82, 83, 85, 98, 102, 110, 125, 128, 129, 144, 149, 152, 155



• **Figure 8-13** A 15-year-old boy was born with bilateral cleft lip and palate. Previous cleft surgery included bilateral cleft lip repair (3 months of age); cleft palate repair (12 months of age); and alveolar bone grafting (8 years of age). The patient has maxillary deficiency and secondary deformities of the mandible, and there is chronic nasal obstruction (i.e., septal deviation, inferior turbinate hypertrophy, and nasal floor irregularities). There is a severe Class III negative overjet malocclusion. Orthodontic (dental) decompensation has been carried out in preparation for jaw and intranasal surgery. The patient's current maxillofacial findings affect speech, chewing, swallowing, breathing, and lip closure/posture. A formal speech–language pathologist's evaluation was carried out.

Chewing/swallowing:

- Able to chew on posterior teeth only
- Occasional nasopharyngeal leakage (into the nose) when drinking milk

Oral-sensory motor:

- Maxillary hypoplasia with Class III malocclusion
- Exhibits mild lip weakness with protrusion and retraction
- Lingual movements appear adequate
- Ability to elevate the soft palate is suspect

Articulation:

- Assessed via the Fisher-Logemann Test of Articulation and during informal conversation
- Misarticulations were identified, including /s/, /sh/, /z/, /dz/, /sk/, and /st/

Production of sounds:

- Sibilant sounds were noted to involve anterior tongue thrusting and lateral lisping during contextual speech
- Exhibits imprecision in coarticulation of speech, which interferes with speech intelligibility
- Unintelligible speech is frequently noted, with mumbling

Continued

• **Figure 8-13, cont'd**

Nasopharyngeal examination:

- Nasal emission under right nares observed during non-nasal speech sounds
- Soft palate motion weak
- Shortened palate during non-nasal speech production of phrases and single words
- Good lateral wall movements bilaterally

Recommendations:

- Exhibits sound misarticulations associated with jaw anomaly and malocclusion
- Misarticulations affect speech function during routine conversation
- Forced to use anterior tongue thrusting and lateral lisping, which distort speech and affect perception of intelligence
- Rated as having inadequate velopharyngeal closure before jaw reconstruction
- Reassess 3 to 6 months after jaw surgery, with anticipated need for a pharyngeal flap

Treatment

Further orthodontic (dental) decompensation in combination with orthognathic surgery was planned. The procedures included maxillary Le Fort I osteotomy (vertical intrusion, horizontal advancement, and counterclockwise rotation); bilateral sagittal split ramus osteotomies (horizontal advancement and counterclockwise rotation); osseous genioplasty (vertical shortening and horizontal advancement); and septoplasty, inferior turbinate reduction, and nasal floor recontouring.

A, Frontal views in repose before and after treatment. **B,** Frontal views with smile before and after treatment. **C,** Oblique facial views before and after treatment. **D,** Profile views before and after treatment.

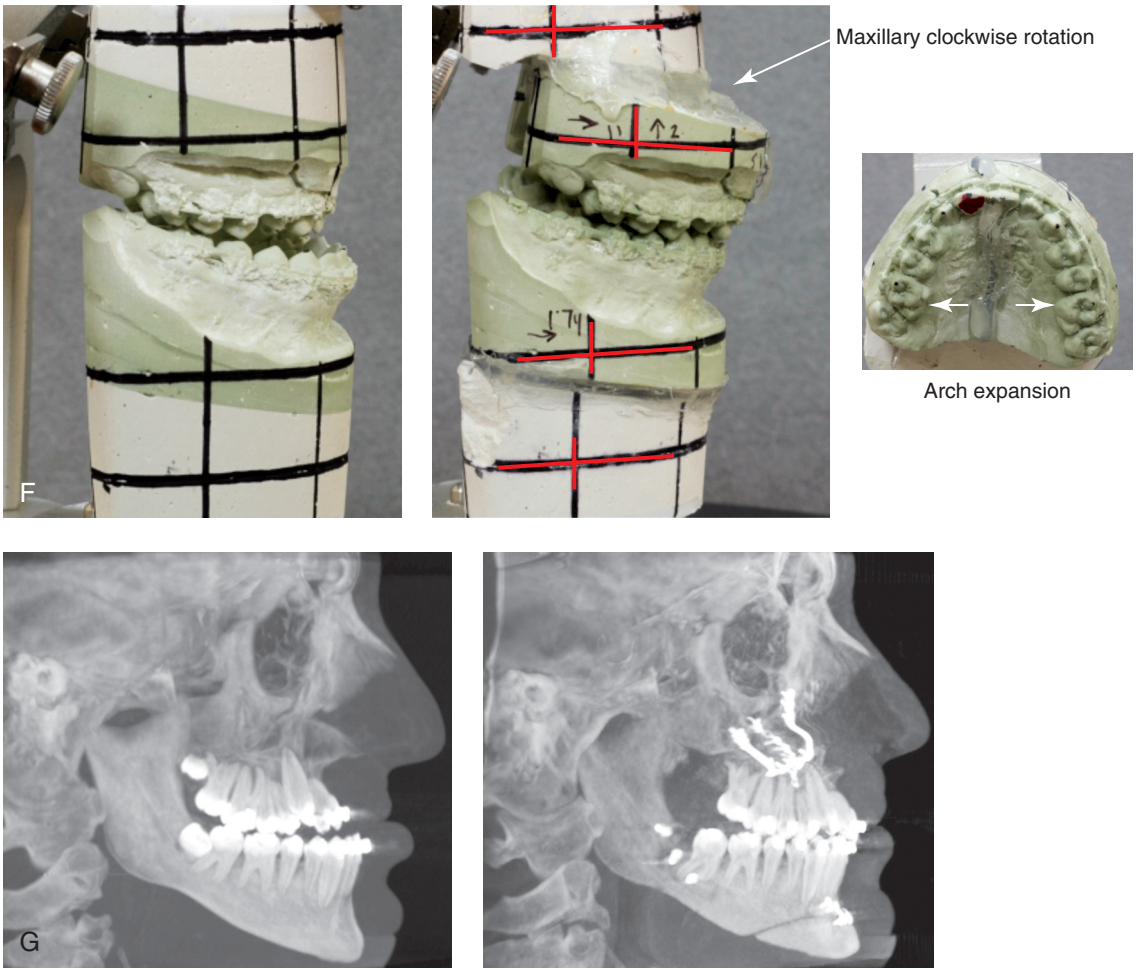
After bone grafting in mixed dentition

Pre orthognathic surgery

After treatment

• **Figure 8-13, cont'd E,** Occlusal views before surgery and then after treatment.

Continued



• **Figure 8-13, cont'd** **F,** Articulated dental casts that indicate analytic model planning. **G,** Lateral cephalometric radiographs before and after surgery. Six months after successful jaw and intranasal reconstruction; a nasoendoscopic speech assessment confirmed the need for a tailor-designed pharyngeal flap.

Epker and Wolford observed that adolescent patients with repaired clefts, maxillary deficiency, and preoperative VPI became worse after Le Fort I advancement.[29] The authors stated that individuals with only borderline VP closure may demonstrate notable VPI after maxillary osteotomy, particularly if the advancement exceeds 10 mm. **Bralley and Schoeny** reported about a 19-year-old patient with a submucous cleft palate and maxillary deficiency who showed no change in nasality after Le Fort I advancement.[9] Schendel and colleagues evaluated 21 patients without clefts before and after maxillary Le Fort I osteotomy to assess VP closure.[110] They did this through speech evaluation, lateral cephalometric radiography, and nasopharyngoscopy, and they found no change in VP competence after Le Fort I advancement.

Witzel described a sample of 41 patients without cleft palate and 50 patients with repaired cleft palate (bilateral, unilateral, or isolated) who underwent Le Fort I maxillary advancement.[152] The results indicated that patients without cleft palate have a very low risk of deterioration of VP function. Patients with a repaired cleft palate who had adequate VP function before Le Fort I advancement were also at lower risk. However, 11 of 15 of the study patients with a repaired cleft palate and a preoperative rating of borderline VP closure acquired surgically induced symptomatic VPI after Le Fort I advancement. Patients who were considered to have inadequate VP closure before surgery remained so after surgery. For most patients, the extent of preoperative competence of the VP sphincter before Le Fort I advancement predicted the competence of the VP sphincter after surgery (as documented by videofluoroscopy and nasoendoscopy).

Kummer and colleagues reported on VP function in two different patient groups: those with repaired clefts and those without clefts who underwent maxillary Le Fort I advancement with or without vertical change.[71] The measured change in VP function was not clinically significant. They concluded that "Le Fort I maxillary osteotomy has a negligible effect on velopharyngeal function in both cleft and non-cleft cases."[71]

Vallino discussed 34 patients without clefts before and at intervals after orthognathic surgery that included Le Fort I osteotomy.[129] The VP port area was measured in the study patients before and at intervals (i.e., 3, 6, 9, and 12 months) after Le Fort I osteotomy. The estimation of the size of the VP port area was obtained with the pressure-flow technique

described by Warren and Dubois.[139] All study patients demonstrated VP port areas that measured between 0 and 0.49 cm^2 both before and after surgery across all speech tasks, which indicated adequate VP competence. The author concluded that the Le Fort I osteotomies carried out in patients without clefts did not alter the VP valve or cause hypernasal speech.

Watzke and colleagues evaluated VP function with the use of aerodynamic testing before and at least 1 year after Le Fort I maxillary advancement in 24 adolescents with repaired cleft palates.[144] Five of these patients (23%) demonstrated VP deterioration, whereas another five (23%) showed improvement after Le Fort I advancement. Interestingly, those patients with a pharyngeal flap in place had a higher incidence of improved VP function after surgery. Of the five (23%) that showed VP deterioration, four had adequate VP function and one had borderline VP function preoperatively. Fourteen (46%) of the patients with clefts showed no significant change in VP function as a result of Le Fort I advancement.

Janulewicz and colleagues completed a retrospective study that evaluated VP function in patients with repaired cleft lips and palates (N = 54) who underwent maxillary Le Fort I advancement.[64] The authors documented a significant deterioration of VP function in many of the patients with clefts after Le Fort I advancement. There was also an increase in hypernasality, which further supported the findings of VP deterioration in a subgroup of the patients with repaired clefts after Le Fort I advancement. The authors also found improvements in hyponasality as compared with in preoperative values, which was attributed to a decrease in nasal airflow obstruction after maxillary advancement.

Velopharyngeal Evaluation Among Individuals with Repaired Cleft Palates and Maxillary Deficiencies Undergoing Le Fort I Advancement

A variety of methods for the assessment of VP function have been described.[57,62,86,89,90,93,94,99,100,105,115,119,140,152] A protocol for the evaluation of patients undergoing orthognathic surgery has been advanced by the American Cleft Palate-Craniofacial Association in *Parameters for Evaluation and Treatment of Patients with Cleft Lip/Palate or Other Craniofacial Anomalies* (American Cleft Palate-Craniofacial Association, Revised edition 2009 www.acpa-cpf.org).

Witzel also developed a protocol for the evaluation of VP function in adolescents and adults after palate repair to anticipate alterations in the VP sphincter after maxillary Le Fort I osteotomy.[154] She recommends a routine speech assessment that includes instrumentation evaluation of VP function, preferably with nasoendoscopy or, as a second choice, videofluoroscopy. After surgery and sufficient time for healing (i.e., 6 to 12 months), a final assessment of articulation and VP function can be carried out. At that time, if VP function is inadequate, definitive management can go forward (e.g., pharyngeal flap surgery or the revision of a flap that is already in place).

Both before and after Le Fort I osteotomy, VP function is rated as adequate, borderline, or inadequate:

- *Adequate function* is defined as normal nasal resonance, normal nasal air emission, and adequate VP closure during speech.
- *Borderline function* is defined as normal nasal resonance or clinically insignificant hypernasality, inaudible nasal air emission (via mirror test), and borderline or marginal VP closure during speech. Patients with this level of functioning usually have small pinhole gaps in the VP valve through which bubbles, barium, or mucus is observed during videofluoroscopy, nasoendoscopy, or both.
- *Inadequate function* is defined as clinically significant hypernasal resonance, nasal air emission, and VP insufficiency during speech.

Patients without clefting and those with repaired cleft palates who present with maxillary deficiency and adequate preoperative VP closure seem to have enough adaptability in the pharyngeal and soft palate muscles to maintain VP closure after Le Fort I advancement. In general, patients with either a repaired cleft palate or those who have undergone uvulopalatopharyngoplasty and who have only borderline VP closure do not have the same adaptability of the soft palate musculature to achieve valve closure after Le Fort I advancement. Thus, they are at higher risk for the development of clinical VPI. Patients with repaired cleft palates and maxillary deficiency or those with obstructive sleep apnea who have undergone uvulopalatopharyngoplasty and who are considered to have inadequate VP closure before Le Fort I advancement are expected to have the same degree of poor closure after surgery.

Conclusions

When managing a dentofacial deformity with malocclusion, the achievement of a successful outcome and a high level of patient satisfaction requires a thorough understanding of issues related to speech, mastication, and swallowing.

A review of the literature shows significant associations between misarticulated speech and Angle Class II, Angle Class III, and anterior open-bite malocclusions. As long as the other aspects of the speech mechanism are not at fault, the correction of misarticulations through successful orthodontics and jaw surgery is the rule.

A competent VP valve is also an important aspect for the production of intelligible speech. The individual with a repaired cleft palate and maxillary deficiency who is only able to achieve borderline VP closure is at higher risk for the development of VP insufficiency after Le Fort I advancement.

With interdisciplinary evaluation that takes into account speech, mastication and swallowing, the most effective care can be rendered to the individual with a jaw deformity and malocclusion.

References

1. Ahlgren J: Mechanism of mastication. *Acta Odontol Scand* 24:44–45, 1966.
2. Anderson DJ, Picton DCA: Tooth contact during chewing. *J Dent Res* 36:21–26, 1957.
3. Anderson DM: *Dorland's illustrated medical dictionary*, ed 28, Philadelphia, 1988, Saunders, p 1104.
4. Aronson AE: Nasal resonatory disorders. In Aronson AE, editor: *Clinical voice disorders*, New York, 1990, Thieme.
5. Beyron HL: Occlusal changes in the adult dentition. *J Am Dent Assoc* 48:674–686, 1954.
6. Beyron HL: Occlusal relations and mastication in Australian aborigines. *Acta Odontol Scand* 22:597–678, 1964.
7. Blakeley RW: The rationale for a temporary speech prosthesis in palatal insufficiency. *Br J Disord Commun* 4:134–139, 1969.
8. Bowers J, Tobey EA, Shaye R: An acoustic-speech study of patients who received orthognathic surgery. *Am J Orthod* 88:373–379, 1985.
9. Brally RC, Schoeny ZG: Effect of maxillary advancement on the speech of a sub-mucosal cleft palate patient. *Cleft Palate J* 14:98, 1977.
10. Brekhus PH: Stimulation of the muscles of mastication. *J Dent Res* 20:87–92, 1941.
11. Bruce FA, Hanson ML: Speech and swallowing changes associated with sagittal osteotomy: a report of four subjects. *Int J Orofac Myol* 13:1, 1987.
12. Brunner M, Stellzig-Eisenhauer A, Proschel U, et al: The effect of nasopharyngoscopic biofeedback in patients with cleft palate and velopharyngeal dysfunction. *Cleft Palate Craniofac J* 42:649–657, 2005.
13. Cable BB, Canady JW, Karnell MP, et al: Pharyngeal flap surgery: long-term outcomes at the University of Iowa. *Plast Reconstr Surg* 113:475, 2004.
14. Chanchareonsook N, Samman N, Whitehill TL: The effect of cranio-maxillofacial osteotomies and distraction osteogenesis on speech and velopharyngeal status: a critical review. *Cleft Palate Craniofac J* 43:477–487, 2006.
15. Chanchareonsook N, Whitehill TL, Samman N: Speech outcome and velopharyngeal function in cleft palate: comparison of Le Fort I maxillary osteotomy and distraction osteogenesis–early results. *Cleft Palate Craniofac J* 44:23–32, 2007.
16. Clark HM: Neuromuscular treatments for speech and swallowing: a tutorial. *Am J Speech Lang Pathol* 12:400–415, 2003.
17. Clark HM: The role of strength training in speech sound disorders. *Semin Speech Lang* 29:276–283, 2008.
18. Cleall JF: Deglutition: a study of form and function. *Am J Orthod* 51:161–182; 566–695, 1965.
19. Costello BJ, Ruiz RL, Turvey T: Surgical management of velopharyngeal insufficiency in the cleft patient. *Oral Maxillofac Surg Clin North Am* 14:539, 2002.
20. Croft CB, Shprintzen RJ, Daniller A, et al: The occult submucous cleft palate and the musculus uvulae. *Cleft Palate J* 15:150, 1978.
21. Dalston RM, Vig PS: Effect of orthognathic surgery on speech: a prospective study. *Am J Orthod* 86:291–298, 1984.
22. Dalston RM: Velopharyngeal impairment in the orthodontic population. *Semin Orthodon* 2:220–227, 1996.
23. Daniloff RG, Wilcox K, Stephens MI: An acoustic-articulatory description of children's defective /s/ productions. *J Commun Dis* 13:347, 1980.
24. De Bodt M, Wuyts F, van de Heyning P, Croux C: Test-retest study of the GRBAS Scale: the influence of experience and professional background on perceptual rating of voice quality. *J Voice* 11:74–80, 1997.
25. Dellow PG, Lund JP: Evidence for central timing of rhythmical mastication. *J Physiol* 215(1):1–13, 1971.
26. Denes PB, Pinson EN: *The speech chain: the physics and biology of spoken language*, Garden City, NY, 1973, Anchor Books, Doubleday & Co, pp 75–83.
27. DeRuyter F, Diefendorf AO: Hearing sensitivity and measurements of middle ear and eustachian tube function after maxillary osteotomy with advancement surgery. *J Oral Surg* 38:343, 1980.
28. Epker BN, Wolford LM: Middle-third facial osteotomies: their use in the correction of acquired and developmental dentofacial and craniofacial deformities. *J Oral Surg* 33:491, 1975.
29. Epker BN, Wolford LM: Middle-third facial osteotomies: their use in the correction of congenital dentofacial and craniofacial deformities. *J Oral Surg* 34:324–342, 1976.
30. Fairbanks G, Lintner MVH: A study of minor organic deviations in 'functional' disorders of articulation. 4. The teeth and hard palate. *J Speech Hearing Dis* 16:273, 1951.
31. Fant G: *Acoustic theory of speech production*, ed 2, The Hague, 1970, Mouton & Co NV.
32. Fisher MA, Logemann JA: *The Fisher-Logemann test of articulation competence*, Boston, 1971, Houghton-Mifflin.
33. Flanagan JB: The 24-hour pattern of swallowing in man. *J Dent Res* 35:109–114, 1956.
34. Flanagan JL: *Speech analysis synthesis and perception*, ed 2, Berlin, Germany, 1972, Springer-Verlag, pp 280–281.
35. Fletcher SG, Casteel RL, Bradley DP: Tongue-thrust swallow, speech articulation and age. *J Speech Hear Dis* 26:201, 1961.
36. Fletcher SG: Contingencies for bioelectronic modification of nasality. *J Speech Hear Disord* 37:329–346, 1972.
37. Forssell H, Finne K, Forssell K, et al: Expectations and perceptions regarding treatment: a prospective study of patients undergoing orthognathic surgery. *Int J Adult Orthodon Orthognath Surg* 13:107–113, 1998.
38. Garber SR, Speidel TM, Marse G: The effects on speech of surgical premaxillary osteotomy. *Am J Orthod* 79:54, 1981.
39. Gardner AF: Dental, oral, and general causes of speech pathology. *Oral Surg Oral Med Oral Pathol* 2:742, 1949.
40. Geffen D: The effects of mandibular osteotomy on articulation and resonance. *South African J Comm Disord* 25:54, 1978.
41. Gibbs CH, Messerman T, Reswick JB, Derda HJ: Functional movements of the mandible. *J Prosthet Dent* 26:604–620, 1971.
42. Glass L, Knapp J, Bloomer HH: Speech and lingual behavior before and after mandibular osteotomy. *J Oral Surg* 35:104–109, 1977.
43. Glickman I, Pameijer JH, Roeber FW, Brian MA: Functional occlusion as revealed by miniaturized radio transmitters. *Dent Clin North Am* 13:667–679, 1969.
44. Goodstein DB, Cooper D, Wallaca L: The effect on speech of surgery for correction of mandibular prognathism. *Oral Surg* 37:846, 1974.
45. Gotzfried HF, Thumfart WE: Pre- and postoperative middle ear function and muscle activity of the soft palate after total maxillary osteotomy in cleft patients. *J Craniomaxillofac Surg* 16:64, 1988.
46. Guay AH, Maxwell DL, Beecher R: Radiographic study of tongue posture at rest and during the phonation of /s/ in class III malocclusion. *Angle Orthod* 48:10, 1978.
47. Guenther TA, Sather AH, Kern EB: The effect of Le Fort I maxillary impaction on nasal airway resistance. *Am J Orthod* 85:388, 1984.
48. Guyette TW, Polley JW, Figueroa AA, Cohen M: Mandibular distraction osteogenesis: effects on articulation and velopharyngeal function. *J Craniofac Surg* 7:186, 1996.
49. Guyette TW, Polley JW, Figueroa A, Smith BE: Changes in speech following maxillary distraction osteogenesis. *Cleft Palate Craniofac J* 38:199–205, 2001.
50. Hamlet S, Cullison BL, Stone ML: Physiological control of sibilant duration: insights afforded by speech compensation to dental prosthesis. *J Acoust Soc Am* 65:1276, 1979.
51. Hardin MA, Van Demark DR, Morris HL, Payne MM: Correspondence between nasalance scores and listener judgments of hypernasality and hyponasality. *Cleft Palate Craniofac J* 29:346–351, 1992.
52. Hardin-Jones M, Chapman K: The impact of early intervention on speech and lexical development for toddlers with cleft palate: a

retrospective look at outcome. *Lang Speech Hear Serv Sch* 39:89–96, 2008.
53. Harvold EP, Vargervik K, Chierici G: Primate experiments on oral sensation and dental malocclusion. *Am J Orthod Dentofacial Orthop* 63:494, 1973.
54. Haryett RD, Hansen FC, Davidson PO: Chronic thumb sucking: the psychological effects and relative effectiveness of various methods of treatment. *Am J Orthod Dentofacial Orthop* 53:569, 1967.
55. Hassan T, Naini F, Gill D: The effects of orthognathic surgery on speech: a review. *J Oral Maxillofac Surg* 65:8, 2007.
56. Henningsson GE, Isberg AM: Velopharyngeal movements in patients alternating between oral and glottal articulation: a clinical and cineradiographical study. *Cleft Palate J* 23:1–9, 1986.
57. Hirschberg J, Van Demark DR: A proposal for standardization of speech and hearing evaluations to assess velopharyngeal function. *Folia Phoniatr Logop* 49:158–167, 1997.
58. Horio T, Kawamura Y: Effects of texture of food on chewing patterns in the human subject. *J Oral Rehabil* 16:177–183, 1989.
59. Houde JF, Jordan MI: Sensorimotor adaptation in speech production. *Science* 279:1213, 1998.
60. Howell AH, Brudevold F: Vertical forces used during chewing of food. *J Dent Res* 29:133–136, 1950.
61. Hu W, Zhou Y, Fu M: Effect of skeletal class III malocclusion on speech articulation. *Zhonghua Kou Qiang Yi Xue Za Zhi* 32:3, 1997.
62. Iglesias A, Kuehn DP, Morris HL: Simultaneous assessment of pharyngeal wall and velar displacement for selected speech sounds. *J Speech Hear Res* 23:429–446, 1980.
63. Jabaley MF, Edgerton MT: Surgical correction of congenital midface retrusion in the presence of mandibular prognathism. *Plast Reconstr Surg* 44:1–8, 1969.
64. Janulewicz J, Costello BJ, Buckley MJ, et al: The effects of Le Fort I osteotomies on velopharyngeal and speech functions in cleft patients. *J Oral Maxillofac Surg* 62:308–314, 2004.
65. Jenkins GN: *The physiology and biochemistry of the mouth*, ed 4, Oxford, UK, 1974, Blackwell Scientific Publications, pp 34–74.
66. Kavanagh M, Fee E, Kalinowski J, et al: Nasometric values for three dialectical groups within the Atlantic provinces of Canada. *J Speech Language Pathol Audiol* 18:7–13, 1994.
67. Kent K: The effects of dental abnormalities on speech production. *Quintessc Int Dent Dig* 12:1353, 1982.
68. Ko EW, Figueroa AA, Guyette TW, et al: Velopharyngeal changes after maxillary advancement in cleft patients with distraction osteogenesis using a rigid external distraction device: 1-year cephalometric follow-up. *J Craniofac Surg* 10:312–320, 1999.
69. Kuehn DP: Velopharyngeal anatomy and physiology. *Ear Nose Throat J* 58:59–136, 1979.
70. Kuehn DP, Moon JB: Levator veli palatini muscle activity in relation to intraoral air pressure variation. *J Speech Hear Res* 37:1260–1270, 1994.
71. Kummer AW, Strife JL, Grau WH, et al: The effects of Le Fort I osteotomy with maxillary movement on articulation, resonance, and velopharyngeal function. *Cleft Palate J* 26:193–199, 1989.
72. Kummer AW, Briggs M, Lee L: The relationship between the characteristics of speech and velopharyngeal gap size. *Cleft Palate Craniofac J* 40:590–596, 2003.
73. Laine T: Malocclusion traits and articulatory components of speech. *Eur J Orthod* 14:302, 1992.
74. Lee SY, Whitehill TL, Ciocca V, et al: Acoustic and perceptual analysis of the sibilant sound /s/ before and after orthognathic surgery. *J Oral Maxillofac Surg* 60:364, 2002.
75. Lof GL, Watson MM: A nationwide survey of nonspeech oral motor exercise use: implications for evidence-based practice. *Lang Speech Hear Serv Sch* 39:392–407, 2008.
76. Lohmander A, Olsson M: Methodology for perceptual assessment of speech in patients with cleft palate: a critical review of the literature. *Cleft Palate Craniofac J* 41:64, 2004.
77. Lubker JF: Normal velopharyngeal function in speech. *Clin Plast Surg* 2:249, 1975.
78. Luce EA, McGibbon B, Hoopes JE: Velopharyngeal insufficiency in hemifacial microsomia. *Plast Reconstr Surg* 30(4):602–606, 1977.
79. Lund JP, Dellow PG: The influence of interactive stimuli on rhythmical masticatory movements in rabbits. *Arch Oral Biol* 16(2):215–223, 1971.
80. Lund JP: Mastication and its control by the brain stem. *Crit Rev Oral Biol Med* 2(1):33–64, 1991.
81. Lundeen HC, Gibbs CH: *Advances in occlusion*, Boston, 1982, John Weight, pp 99–127.
82. Maegawa J, Sells RK, David DJ: Speech changes after maxillary advancement in 40 cleft lip and palate patients. *J Craniofac Surg* 9:177–182, 1998.
83. Mason RM, Turvey TA, Warren DW: Speech considerations with maxillary advancement procedures. *J Oral Surg* 38:752–758, 1980.
84. Massengill R, Quinn GW: Adenoidal atrophy, velopharyngeal incompetence and sucking exercises: a 2-year follow-up case report. *Cleft Palate J* 11:196–199, 1974.
85. McCarthy JG, Coccaro JP, Schwartz MD: Velopharyngeal function following maxillary advancement. *Plast Reconstr Surg* 64:180, 1979.
86. McComb R, Marrinan E, Nuss RC, et al: Predictors of velopharyngeal insufficiency after Le Fort I maxillary advancement in patients with cleft palate. *J Oral Maxillofac Surg* 69:2226–2232, 2011.
87. McFarland DH, Baum SR: Incomplete compensation to articulatory perturbation. *J Acoust Soc Am* 97:1865, 1995.
88. Marrinan EM, LaBrie RA, Mulliken JB: Velopharyngeal function in nonsyndromic cleft palate: relevance of surgical technique, age at repair, and cleft type. *Cleft Palate Craniofac J* 35:95–100, 1998.
89. Miyazaki T, Matsuya T, Yamaoka M: Fiberscopic methods for assessment of velopharyngeal closure during various activities. *Cleft Palate J* 12:107–114, 1975.
90. Moll KL: A cinefluorographic study of velopharyngeal function in normals during various activities. *Cleft Palate J* 2:112–122, 1965.
91. Moon JB, Kuehn DP, Chan G, Zhao L: Induced velopharyngeal fatigue effects in speakers with repaired palatal clefts. *Cleft Palate Craniofac J* 44:251–260, 2007.
92. Morris HL, Spreistersbach DC, Darley FL: An articulation for assessing competency of velopharyngeal closure. *J Speech Hearing Res* 4:48, 1961.
93. Morris HL: Some questions and answers about velopharyngeal dysfunction during speech. *Am J Speech Lang Pathol* 1:26–28, 1992.
94. Niemi M, Laaksonen J, Peltomaki T, et al: Acoustic comparison of vowel sounds produced before and after orthognathic surgery for mandibular advancement. *J Oral Maxillofac Surg* 64:910, 2006.
95. Niemi M, Laaksonen J, Peltomaki T, Aaltonen O: Acoustic comparison of vowel sounds produced before and after orthognathic surgery for mandibular advancement. *J Oral Maxillofac Surg* 64:7, 2006.
96. Nozaki S, Iriki A, Nakamura Y: Localization of central rhythm generator involved in cortically induced rhythmical masticatory jaw-opening movement in the guinea pig. *Neurophysiology* 55:806–825, 1986.
97. O'Gara M, Wilson K: The effects of maxillofacial surgery on speech and velopharyngeal function. *Clin Plast Surg* 34:8, 2007
98. Okazaki K, Satoh K, Kato M, et al: Speech and velopharyngeal function following maxillary advancement in patients with cleft lip and palate. *Ann Plast Surg* 30:304–311, 1993.
99. Pannbacker M, Lass NJ, Stout BM: Speech language pathologists: opinions on the management of velopharyngeal insufficiency. *Cleft Palate J* 27:68–71, 1990.
100. Pannbacker M: Velopharyngeal incompetence: the need for speech standards. *Am J Speech Lang Pathol* 13:195–201, 2004.
101. Peterson-Falzone SJ, Hardin-Jones MA, Karnell MP: *Cleft palate speech*, St. Louis, 2001, Mosby.
102. Phillips JH, Klaiman P, Delorey R, MacDonald DB: Predictors of

velopharyngeal insufficiency in cleft palate orthognathic surgery. *Plast Reconstr Surg* 115:681–686, 2005.
103. Pond LH, Barghi N, Barnwell GM: Occlusion and chewing side preference. *J Prosthet Dent* 55(4):498–500, 1986.
104. Proffit WR, Mason RM: Myofunctional therapy for tongue thrusting: background and recommendations. *J Am Dent Assoc* 90:403, 1975.
105. Roll DJ: Modification of nasal resonance in cleft palate children by informative feedback. *J Appl Behav Anal* 6:397–403, 1973.
106. Ruscello DM, Tekieli ME, Van Sickels JE: Speech production before and after orthognathic surgery: a review. *Oral Surg* 59:10, 1985.
107. Ruscello DM, Tekieli ME, Jakomis T, et al: The effects of orthognathic surgery on speech production. *Am J Orthod* 89:237–241, 1986.
108. Ruscello DM: Treatment of velopharyngeal closure for speech: discussion and implications for management. *J Speech Lang Pathol Appl Behav Anal* 1:62–82, 2007.
109. Ruscello DM: Oral motor treatment issues related to children with developmental speech sound disorders. *Lang Speech Hear Serv Sch* 39:380–391, 2008.
110. Schendel SA, Oeschlaeger M, Wolford LM, Epker BN: Velopharyngeal anatomy and maxillary advancement. *J Maxillofac Surg* 7:116–124, 1979.
111. Schneider E, Shprintzen RJ: A survey of speech pathologists: current trends in the diagnosis and management of velopharyngeal insufficiency. *Cleft Palate J* 17:249–253, 1980.
112. Schneyer LH, Pigman W, Hanahan L, Gilmore RW: Rate of flow of human parotid, sublingual and submaxillary secretions during sleep. *J Dent Res* 35:109–114, 1956.
113. Schwarz C, Gruner E: Logopaedic findings following advancement of the maxilla. *J Maxillofac Surg* 4:40–55, 1976.
114. Schweitzer JM: Masticatory function in man. *J Prosthet Dent* 11:625–647, 1961.
115. Seaver E, Dalston R, Leeper H, Adams L: A study of nasometric values for normal nasal resonance. *J Speech Hearing Res* 34:715–721, 1991.
116. Shelton RL, Lindquist AF, Arndt WB, et al: Effect of speech bulb reduction on movement of the posterior wall of the pharynx and posture of the tongue. *Cleft Palate J* 8:10–17, 1971.
117. Shelton RL, Paesani A, McClelland KD, Bradfield SS: Panendoscopic feedback in the study of voluntary velopharyngeal movements. *J Speech Hear Disord* 40:232–244, 1975.
118. Shprintzen RJ, McCall GN, Skolnick ML: The effect of pharyngeal flap surgery on the movements of the lateral pharyngeal walls. *Plast Reconstr Surg* 66:570, 1980.
119. Shprintzen RJ, Golding-Kushner KJ: Evaluation of velopharyngeal insufficiency. *Otolaryngol Clin North Am* 22:519, 1989.
120. Stevens K: On the quantal nature of speech. *J Phon* 17:3, 1989.
121. Subtelny JD, Mestre JC, Subtelny JD: Comparative study of normal and defective articulation of /s/ as related to malocclusion and deglutition. *J Speech Hearing Dis* 29:264, 1964.
122. Thompson AE, Hixon TJ: Nasal airflow during normal speech production. *Cleft Palate J* 16:412–420, 1979.
123. Thompson EC, Murdoch BE, Stokes PD: Tongue function in subjects with upper motor neuron type dysarthria following cerebrovascular accident. *J Med Speech Lang Pathol* 3:27–40, 1995.
124. Thompson EC, Murdoch BE, Stokes PD: Interlabial contact pressures during performance of speech and nonspeech tasks in young adults. *J Med Speech Lang Pathol* 5:191–199, 1997.
125. Trindale IE, Yamashita RP, Suguimoto RM, et al: Effects of orthognathic surgery on speech and breathing of subjects with cleft lip and palate: acoustic and aerodynamic assessment. *Cleft Palate Craniofac J* 40:54–64, 2003.
126. Turvey TA, Joumot V, Epker BN: Correction of anterior open bite deformity: a study of tongue function, speech changes, and stability. *J Maxillofac Surg* 4:93, 1976.
127. Turvey TA, Hall DJ, Warren DW: Alterations in nasal airway resistance following superior repositioning of the maxilla. *Am J Orthod* 85:109, 1984.
128. Vallino L: *The effects of orthognathic surgery on speech, velopharyngeal function, and hearing*. Doctoral dissertation. University of Pittsburgh, 1987.
129. Vallino L: Speech, velopharyngeal function, and hearing before and after orthognathic surgery. *J Oral Maxillofac Surg* 48:1274–1281, 1990.
130. Vallino LD, Tompson B: Perceptual characteristics of consonant errors associated with malocclusion. *J Oral Maxillofac Surg* 51:850–856, 1993.
131. Van de Weijer J, Slis I: Nasaliteitsmeting met de nasometer. *Logopedie en Foniatrie* 63:97–101, 1991.
132. Van Lierde KM, Wuyts FL, De Bodt M, Van Cauwenberge P: Nasometric values for normal nasal resonance in the speech of young Flemish adults. *Cleft Palate Craniofac J* 38:112–118, 2001.
133. Van Lierde KM, De Bodt M, Baetens I, et al: Outcome of treatment regarding articulation, resonance and voice in Flemish adults with unilateral and bilateral cleft palate. *Folia Phoniatrica et Logopaedica* 55:80–90, 2003.
134. Van Lierde KM, Schepers S, Timmermans L, et al: The impact of mandibular advancement on articulation, resonance, and voice characteristics in Flemish speaking adults: a pilot study. *Int J Oral Maxillofac Surg* 35:1, 2006.
135. Wakumoto M, Isaacson KG, Friel S, et al: Preliminary study of articulatory reorganization of fricative consonants following osteotomy. *Folia Phoniatr Logop* 48:275, 1996.
136. Wang LL, Yang JF, Chen J, et al: Correlation analysis on the malocclusion and articulation of skeletal angle III malocclusion in mixed dentition. *Hua Xi Kou Qiang Yi Xue Za Zhi* 24:3, 2006.
137. Ward EC, McAuliffe M, Holmes SK, et al: Impact of malocclusion and orthognathic reconstruction surgery on resonance and articulatory function: an examination of variability of five cases. *Br J Oral Maxillofac Surg* 40:410, 2002.
138. Warren DW: Velopharyngeal orifice size and upper pharyngeal pressure flow patterns in normal speech. *Plast Reconstr Surg* 2:148, 1964.
139. Warren DW, DuBois AB: A pressure-flow technique for measuring velopharyngeal orifice area during continuous speech. *Cleft Palate J* 1:52, 1964.
140. Warren DW: Perci: a method for rating palatal efficiency. *Cleft Palate J* 16:279, 1979.
141. Warren DW: Compensatory speech behaviors in cleft palate: a regulation/control phenomenon. *Cleft Palate J* 23:251, 1986.
142. Warren DW, Dalston RM, Mayo R: Hypernasality in the presence of "adequate velopharyngeal closure." *Cleft Palate Craniofac J* 30:150–154, 1993.
143. Warren DW: Perceptual characteristics of consonant errors associated with malocclusion [discussion]. *J Oral Maxillofac Surg* 51:856, 1993.
144. Watzke I, Turvey TA, Warren DW, Dalston R: Alterations in velopharyngeal function after maxillary advancement in cleft palate patients. *J Oral Maxillofac Surg* 48:685–689, 1990.
145. Weber J, Jobe RP, Chase RA: Evaluation of muscle stimulation in the rehabilitation of patients with hypernasal speech. *Plast Reconstr Surg* 46:173–174, 1970.
146. Weimer K, Astrand P: Effect on speech of mandibular prognathism before and after surgical treatment. *Swed Dental J* 1:173–176, 1977.
147. Whitehill TL, Samman N, Wong LLN, et al: Speech errors associated with dentofacial abnormalities in Cantonese speakers. *J Med Speech-Lang Pathol* 9:177, 2001.
148. Wickwire NA, White RP, Proffit WR: The effect of mandibular osteotomy on tongue position. *J Oral Surg* 30:184–190, 1972.
149. Witzel MA, Munro IR: Velopharyngeal insufficiency after maxillary advancement. *Cleft Palate J* 14:176–180, 1977.
150. Witzel MA, Ross RB, Munro IR: Articulation before and after facial osteotomy. *J Maxillofac Surg* 8:195–202, 1980.
151. Witzel MA, Ross RB, Munro K: Articulation before and after facial osteotomy. *J Maxillofac Surg* 8:8, 1980.
152. Witzel MA: *Orthognathic defects and surgical correction: The effects on speech and*

velopharyngeal function. Doctoral dissertation. University of Pittsburgh, 1981.
153. Witzel MA, Tobe J, Sayler KE: The use of nasopharyngoscopy biofeedback therapy in the correction of inconsistent velopharyngeal closure. *Int J Pediatr Otorhinolaryngol* 15:137–142, 1988.
154. Witzel MA, Tobe J, Sayler KE: The use of videonasopharyngoscopy after pharyngeal flap surgery. *Cleft Palate J* 26:129–135, 1989.
155. Witzel MA: The effects of Le Fort I osteotomy with maxillary movement on articulation, resonance, and velopharyngeal function [commentary]. *Cleft Palate J* 26:199–200, 1989.
156. Worner HK, Anderson MN: Biting force measurements in children. *Aust Dent J* 48:1–5, 1944.
157. Xue SA, Lam CW, Whitehill TL, Samman N: Effects of Class III malocclusion on young male adults' vocal tract development: a pilot study. *J Oral Maxillofac Surg* 69:845–852, 2011.
158. Yamashita S, Hatch JP, Rugh JD: Does chewing performance depend upon a specific masticatory pattern? *J Oral Rehabil* 26(7):547–553, 1999.
159. Ysunza A, Pamplona C, Toledo E: Change in velopharyngeal valving after speech therapy in cleft palate patients: a videonasopharyngoscopic and multi-view videofluoroscopic study. *Int J Pediatr Otorhinolaryngol* 24:45–54, 1992.
160. Ysunza A, Pamplona C, Ramirez E, et al: Velopharyngeal surgery: a prospective randomized study of pharyngeal flaps and sphincter pharyngoplasties. *Plast Reconstr Surg* 110:1401, 2000.
161. Yules RB, Chase RA: A training method for reduction of hypernasality in speech. *Plast Reconstr Surg* 43:180–185, 1969.

9

Temporomandibular Disorders: Effects of Occlusion, Orthodontic Treatment, and Orthognathic Surgery

JEFFREY C. POSNICK, DMD, MD

- Incidence of Temporomandibular Disorders in the General Population and the Basic Treatment Approach
- Mandibular Range of Motion
- Review of Occlusal Factors and Association with Temporomandibular Disorders
- Occlusal Conditions More Frequently Associated with Temporomandibular Disorders
- Orthodontic Treatment and Its Effects on Temporomandibular Disorders
- Association of Jaw Deformities, Orthognathic Surgery, and Temporomandibular Disorders
- Brief Overview of Okeson's Thinking About Temporomandibular Disorders and the Effects of Occlusion
- Personal Thoughts About the Role of Open Joint Procedures for Temporomandibular Disorders
- Conclusions

Incidence of Temporomandibular Disorders in the General Population and the Basic Treatment Approach

The prevalence of temporomandibular disorders (TMDs) in the general population as reported in the literature varies widely. It is estimated that an average of 32% of the population reports at least one symptom of TMD, whereas an average of 55% demonstrates at least one clinical sign.* Schiffman and colleagues completed a cross-sectional epidemiologic study that indicated that, at any given time, between 40% and 75% of individuals who were not currently seeking treatment had at least one sign of TMD, and about one third of individuals reported at least one symptom of TMD.[296] Mongini and colleagues documented a prevalence of TMD symptoms in children and teenagers of 12% and 20%, respectively.[210] Wanman and colleagues showed an incidence of joint noise in young adults in their late teens to be as high as 17.5% over a 2-year period.[345] TMDs remain a frequent cause of visits to primary care physicians, otolaryngologists, internists, and pediatricians. Despite the high incidence of TMDs, their natural history suggests that, in up to 40% of patients, symptoms resolve spontaneously.[296]

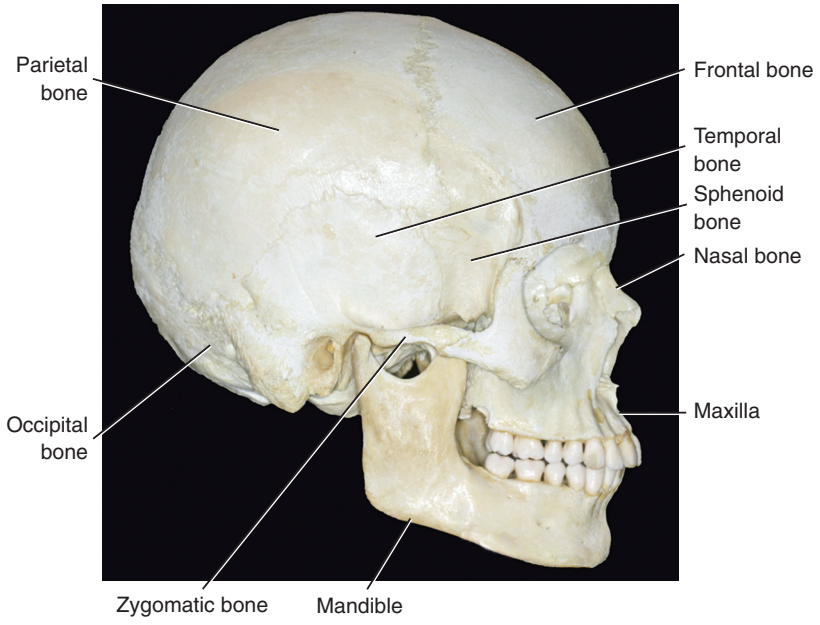
TMD includes various signs and symptoms of the temporomandibular joint (TMJ), the masticatory muscles, and related structures (Figs. 9-1 through 9-8). These may include a spectrum of referred head and neck pain; joint noise (e.g., popping, clicking, crepitus); reduced or altered mandibular range of movement as a result of muscle spasm or disc displacement; condylar head erosion; and direct pain on palpation of either the TMJ or the masticatory muscles. The signs and symptoms associated with TMDs can also lead to misdiagnosis.[16,112,171,216,342]

A wide variety of often contradictory "conservative" treatment modalities (i.e., non-invasive or potentially

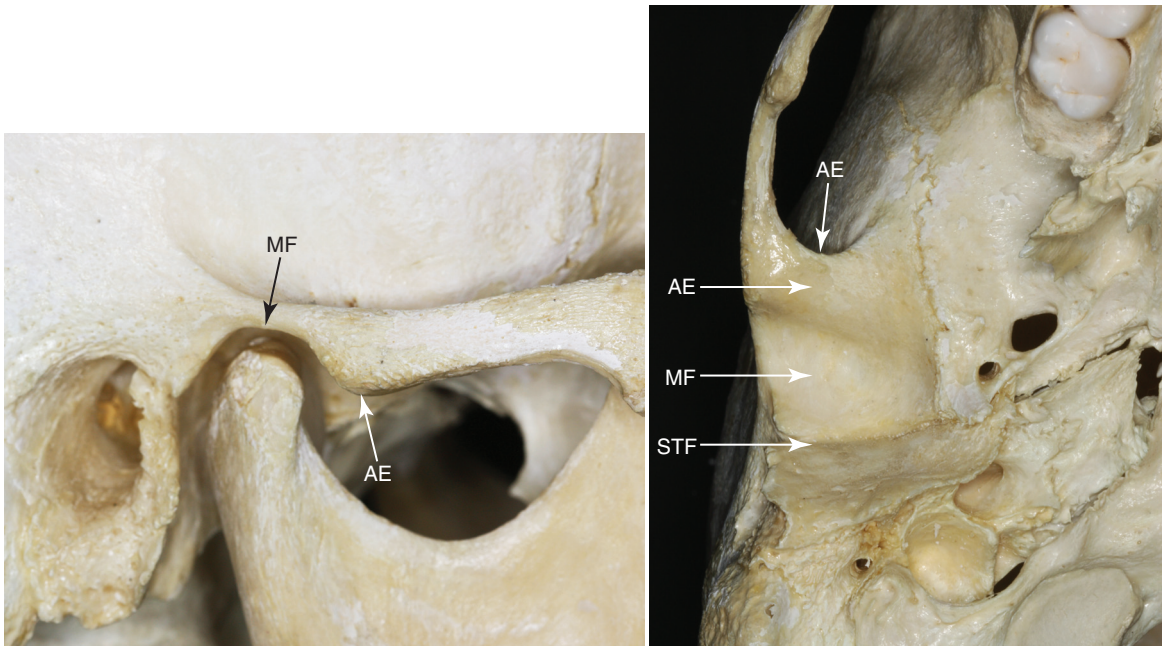
Text continued on p. 269

*References 22, 24, 39, 40, 50, 80, 81, 83, 94, 95, 116-118, 120, 122, 126, 139, 147, 148, 151, 161, 170, 173, 175-177, 181, 183, 190-195, 199, 207, 214, 218, 220-221, 223, 244, 251, 252, 262, 264, 270, 277, 300, 301, 319, 326, 343, 345, 348

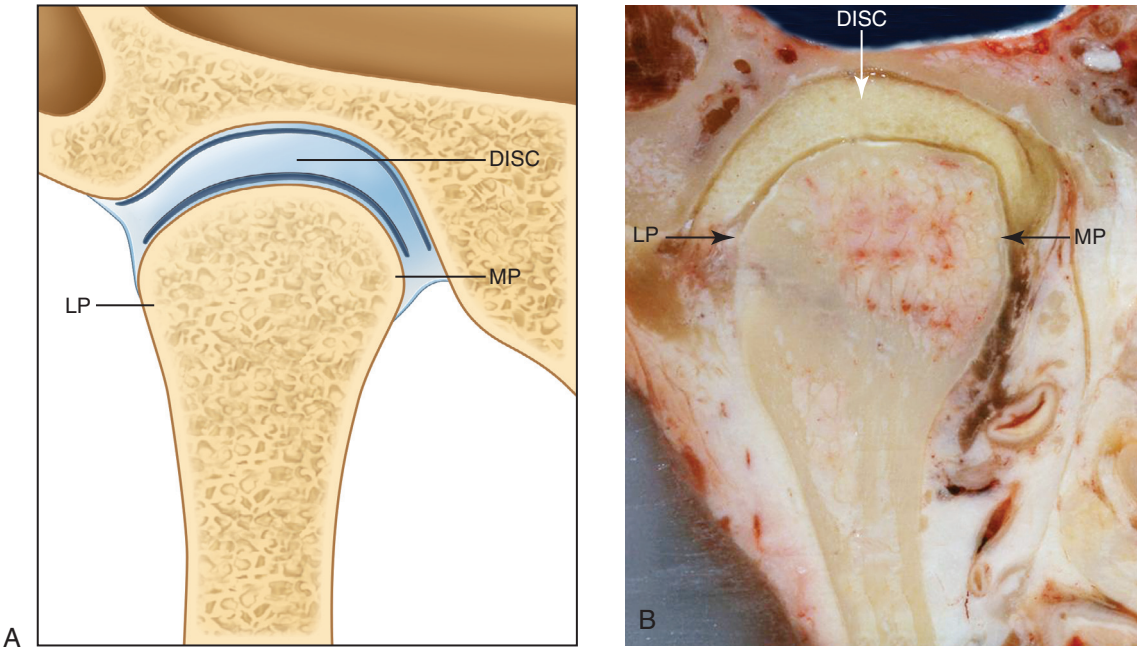
• **Figure 9-1 A,** Intraoral frontal view of dentition juxtaposed with the underlying alveolar bone. **B,** Intraoral lateral view of dentition juxtaposed with the underlying alveolar bone. *From Okeson JP:* Management of temporomandibular disorders and occlusion, *St. Louis, 2012, Elsevier, p 3, Figures 1-1,* A *and* B, *1-3, and 1-4.*



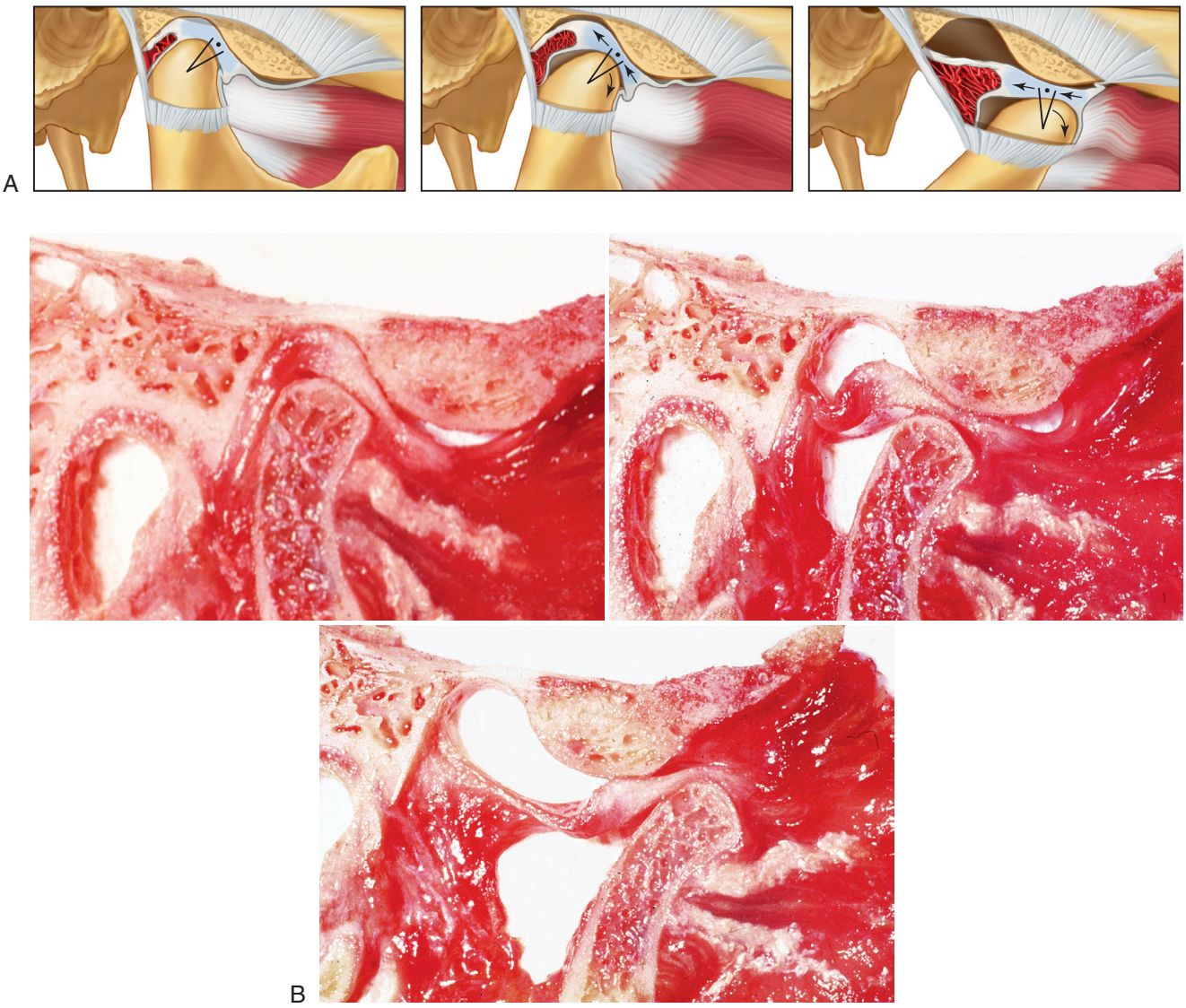
• **Figure 9-2** Lateral view of the craniofacial skeleton. *From Okeson JP:* Management of temporomandibular disorders and occlusion, *St. Louis, 2012, Elsevier, p 4, Figure 1-5.*



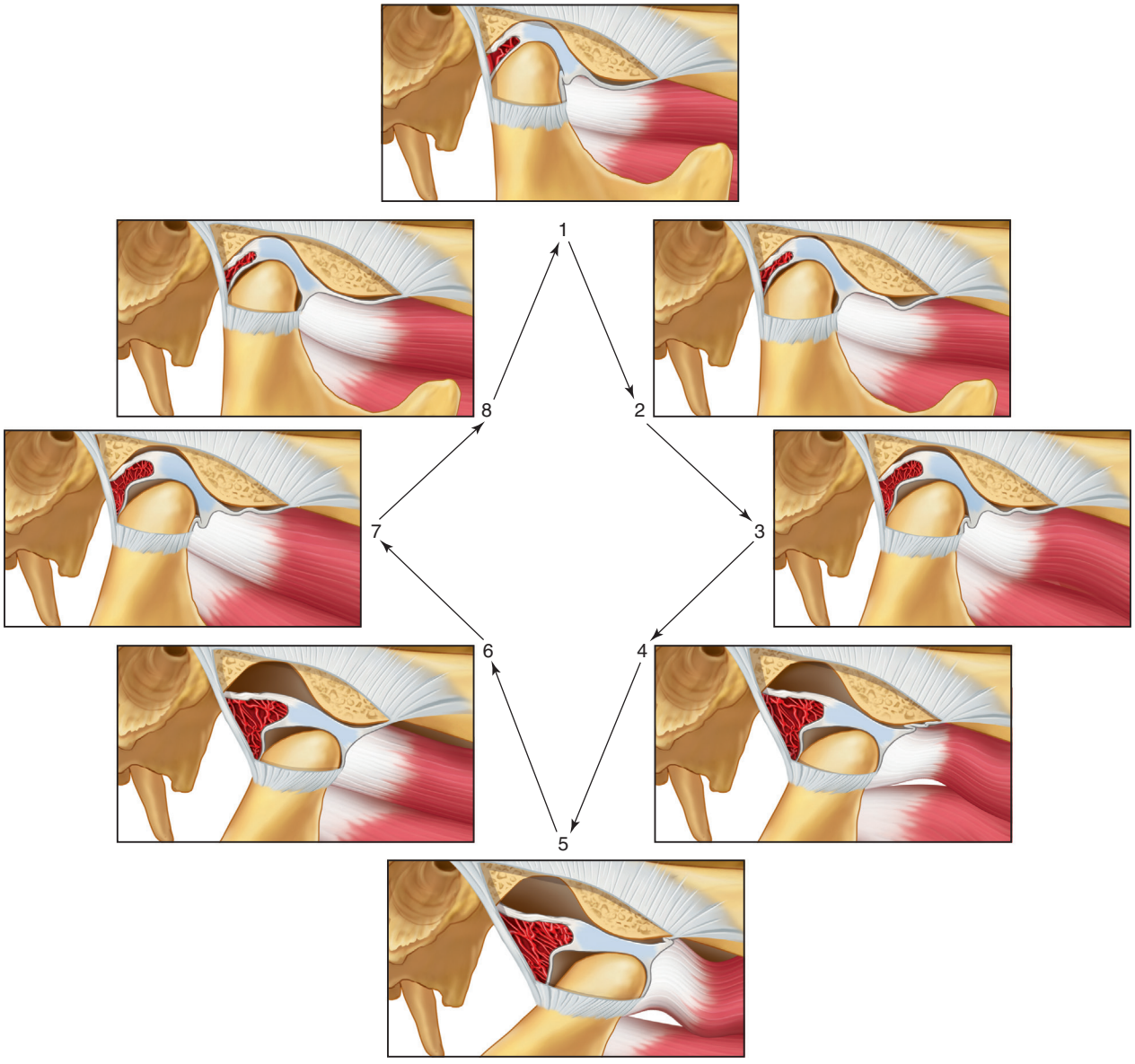
• **Figure 9-3** Bony structures (components) of the temporomandibular joint. *MF,* Mandibular fossa; *AE,* articular eminence; *STF,* squamotympanic fissure. *From Okeson JP:* Management of temporomandibular disorders and occlusion, *St. Louis, 2012, Elsevier, p 4, Figure 1-12.*



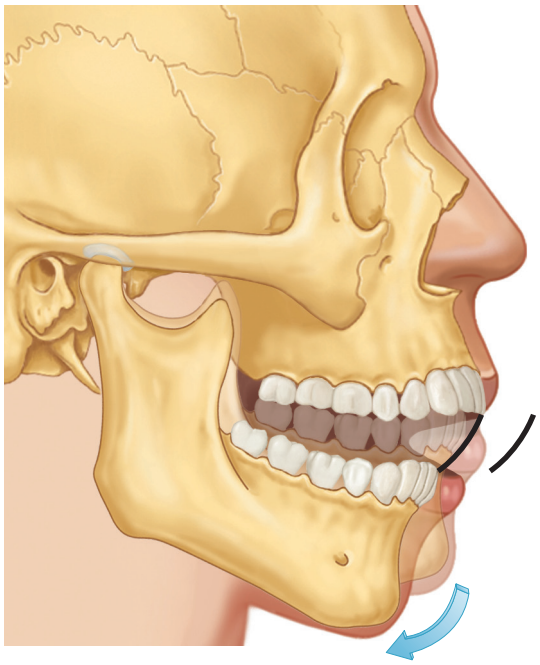
• **Figure 9-4** Anterior cross-sectional view of the articular disc, the fossa, and the condyle. **A,** Illustration. **B,** Cadaver specimen. The disc adapts to the morphology of the fossa and the condyle. *LP,* Lateral pole; *MP,* medial pole. *From Okeson JP:* Management of temporomandibular disorders and occlusion, *St. Louis, 2012, Elsevier, p 8, Figure 1-14.*



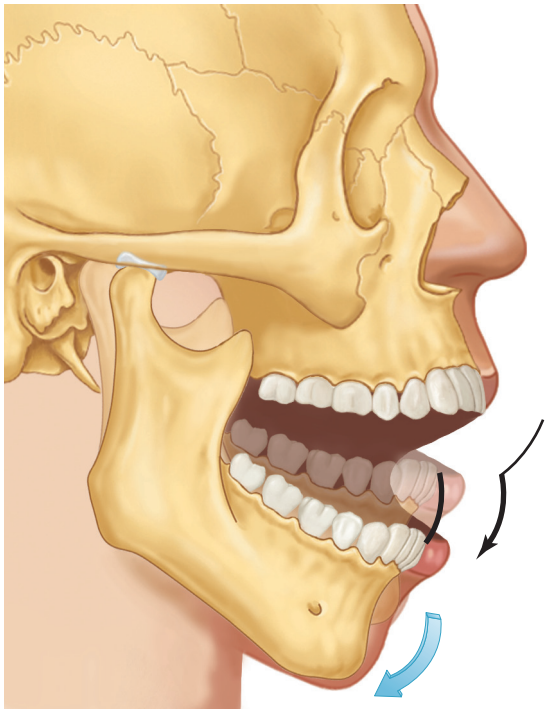
• **Figure 9-5 A,** Lateral view illustrations and **B,** cadaver specimen views of normal movement of the condyle and disc during vertical mouth opening. As the disc moves out of the fossa, it rotates posteriorly on the condyle. First, rotational movement occurs (predominantly in the lower joint space). After that, translation takes place (predominantly in the superior joint space) as mouth opening continues. **A** *from Okeson JP:* Management of temporomandibular disorders and occlusion, *St. Louis, 2012, Elsevier, p 17, Figure 1-30.* **B** *courtesy Terry Tanaka, MD, San Diego, Calif.*



• **Figure 9-6** Illustrations of the normal functional movement of the condyle and disc during the full range of opening and closing. The disc is rotated posteriorly on the condyle as the condyle is translated anteriorly out of the fossa. The closing movement is the exact opposite of the opening movement. *From Okeson JP:* Management of temporomandibular disorders and occlusion, *St. Louis, 2012, Elsevier, p 19, Figure 1-31.*



• **Figure 9-7** Illustration of rotational movement of the mandible. This movement occurs with the condyles in the terminal hinge position. This pretranslation opening will occur until the anterior teeth are approximately 20 to 25 mm apart. *From Okeson JP:* Management of temporomandibular disorders and occlusion, *St. Louis, 2012, Elsevier, p 64, Figure 4-7.*



• **Figure 9-8** The second stage of movement during mouth opening is illustrated. Note that the condyle is translated down the articular eminence as the mouth rotates open to its maximum limit. *From Okeson JP:* Management of temporomandibular disorders and occlusion, *St. Louis, 2012, Elsevier, p 64, Figure 4-8.*

reversible) have been recommended to alleviate the symptoms of TMD and to prevent recurrence.* Enthusiasm by clinicians for the direct surgical alteration of the structures that comprise the TMJ to treat TMD have waxed and waned over the past half century.[60,71,141,143,200,222,241,344] Occlusal factors (i.e., degrees of malocclusion) are often claimed to be associated with TMD. Although it is sometimes stated that orthodontic treatment increases the prevalence of TMD, it is also claimed that orthodontic maneuvers (for at least some types of malocclusion) may reduce the signs and symptoms of TMD. Studies document that the correction of a baseline jaw deformity with malocclusion through orthodontics and orthognathic surgery improves the presenting TMD in the majority of the treated patients, whereas a minority get worse.[6,29,31,67,102,131,152,232,236,347,349,356,359] This is described in greater detail later in this chapter.

Mandibular Range of Motion

The expected normal mandibular range of motion and inter-incisor mouth opening is discussed periodically in the literature.[9,70,93,115,146,179,197,234,235,249,308,309,311-313,315,316,318] Even a 6-year-old child can often open the mouth to a maximum 40 mm or more. Measurements of maximum vertical mouth opening with and without discomfort are considered useful parameters of TMJ health. A restricted mouth opening is considered by many to be a distance of less than 40 mm. Only 1.2% of young adults open their mouths less than 40 mm. However, 15% of the healthy elderly population open their mouths less than 40 mm. Any lateral or protrusive movement of less than 8 mm is considered restricted, although the significance of this measurement is unclear.

Review of Occlusal Factors and Association with Temporomandibular Disorders

Clinicians from a spectrum of medical and dental specialties often consider malocclusion to be the major etiologic factor of TMD.† An assumed strong association between TMD and the malpositioning of the teeth serves as one reason why the diagnosis and treatment of TMD is generally considered within the purview of dentistry. Many of the therapeutic options for the treatment of TMD are based on the idea of "normalizing" or "neutralizing" the

*References 10, 13, 20, 30, 53, 65, 72, 82, 91, 97, 113, 119, 142, 204-206, 213, 219, 238, 266, 297, 324, 328, 336, 338, 339, 360

†References 1, 4, 5, 8, 11, 12, 14, 15, 17, 21, 23, 26-28, 37, 38, 41, 43, 44, 51, 56-58, 63, 64, 78, 79, 85-88, 92, 99-101, 106, 114, 125, 129, 130, 132, 133, 136, 140, 149, 153, 162, 163, 178, 186, 188, 196, 198, 201, 202, 208-210, 215, 217, 233, 239, 242, 243, 246, 248, 250, 254-261, 263, 265, 269, 278, 280, 283-287, 298, 299, 302-307, 310, 314, 317, 321, 327, 333, 337, 341, 346, 351, 357

occlusion.[135,355] Common treatment approaches to do so include the reversible use of an occlusal appliance or definitive techniques such as occlusal adjustment (equilibration), restorative dentistry, orthodontics, and orthognathic surgery. Concurrently, it is also true that either the onset of TMD or the worsening of current symptoms is frequently blamed on any one of the previously mentioned treatment regimens.

The dental literature contains clinical studies and reports that claim there are causal relationships between occlusal factors and the symptoms and signs associated with TMD. Egermark-Eriksson and colleagues examined 402 children and reported an association between signs and symptoms of TMD, specific malocclusions (i.e., Class II, Class III, anterior crossbite, and anterior open bite), and occlusal supra-contacts.[87] Brandt studied 1342 children and found a correlation between TMD and degrees of overbite (deep and open) and overjet.[33] Nilner examined 749 teenagers and young adults and reported an association with centric relation (CR)–centric occlusion (CO) discrepancies (i.e., slides), balancing-side contact slides, and symptoms and signs of TMD.[220] Confusion in the literature arises as independent research studies have drawn contrary conclusions. DeBoever and colleagues examined 135 individuals with signs and symptoms of TMD and found no specific associations with malocclusion.[59] Gunn and colleagues similarly found no association between TMD and occlusion in 151 migrant children that they studied.[121] Dworkin and colleagues examined 592 individuals within a specific health maintenance organization and found no association between TMD signs and symptoms and specific occlusions.[80]

In their review article, Seligman and Pullinger pointed out that the majority of existing clinical research (up to that point in time) did not support a clear relationship between occlusion and TMD symptoms.[302] They further clarified that the majority of published research relied on symptom-based data rather than discrete disease classifications. Seligman pointed out that symptoms like joint sounds (e.g., popping, clicking, crepitus) and pain (e.g., in the TMJ region and the masticatory system) lack specificity.[303] In addition, most published studies did not differentiate between TMD diagnostic categories (e.g., disc displacement, myalgia, TMJ osteoarthritis). Most published studies also suffered from a lack of or an inadequate control group. Seligman and colleagues attempted to avoid these shortcomings by completing a multifactorial analysis of the risks and relative odds of an individual having a TMD as a function of 1 of 11 common occlusal features.[259,304] They looked for correlations between each of these 11 specific occlusal factors (Box 9-1) and 1 of 6 TMJ diagnostic categories (Box 9-2), including asymptomatic normal controls without TMD.[259]

The asymptomatic control subjects (N = 147) were considered the reference standard because they were without current symptoms or signs of TMD and because they had no history of TMD. Occlusal findings in the symptomatic group showed wide variations, including the following:

- Overjets ranging from –1 to +6 mm
- Overbites ranging from –2 to +10 mm
- Midline discrepancies of up to 5 mm
- Anteroposterior molar relationships from –6 to +6 mm
- Molar asymmetries of 0 to 6 mm
- CR–CO slides of up to 2 mm

There were also a wide variety of crossbites, asymmetrical slides, posterior contacts, and severe attrition facets in the control group. Interestingly, no anterior open bites were found in the control group.

The authors concluded that what have been traditionally considered malocclusions cannot be used to define or

• BOX 9-1

Occlusal Features and Factors to Consider in TMD*

1. Anterior open bite
2. Maxillary lingual posterior crossbite
3. Retruded centric position–inter-contact position slide length
4. Retruded centric position–inter-contact position slide asymmetry
5. Unilateral retruded centric position contact
6. Overbite
7. Overjet
8. Dental midline discrepancy
9. Number of missing posterior teeth
10. First molar relationships (i.e., the greater of the mesiodistal maxillary discrepancies at the first molar location)
11. Right versus left first molar position asymmetry

*Data from Pullinger AG, Seligman DA, Gornbein JA: A multiple logistic regression analysis of the risk and relative odds of temporomandibular disorders as a function of common occlusal features. *J Dent Res* 72:968–979, 1993.

• BOX 9-2

Temporomandibular Joint Diagnostic Categories to Consider in TMD*

1. Disc displacement with reduction (N = 81)
2. Disc displacement without reduction (N = 48)
3. Temporomandibular joint osteoarthrosis with disc displacement history (N = 75)
4. Primary osteoarthrosis (N = 85)
5. Myalgia only (N = 124)
6. Asymptomatic normals (N = 147)

*Data from Pullinger AG, Seligman DA, Gornbein JA: A multiple logistic regression analysis of the risk and relative odds of temporomandibular disorders as a function of common occlusal features. *J Dent Res* 72:968–979, 1993.

predict either individuals with TMD or those who are or will become asymptomatic. No single occlusal factor was able to differentiate a TMD patient from a healthy subject. The authors did identify four specific occlusal features that occurred mainly in individuals who were symptomatic with TMD (see diagnostic categories 1 through 5) and that were rarely found in those who were asymptomatic for TMD (see diagnostic category 6). The occlusal features that primarily occurred in individuals with symptomatic TMD included the following:

1. The presence of a skeletal anterior open bite
2. CR-CO slides of more than 2 mm
3. Overjets of more than 4 mm
4. Five or more missing and unreplaced posterior teeth

Pullinger and colleagues concluded that occlusion cannot be considered the most important factor in the definition of TMD but that specific occlusal factors do make biologic contributions to TMD and thus cannot be ignored.[254-261] In most individuals, they observed that the human biologic system will adapt to a variety of malocclusions in an attempt to achieve TMJ stability. The authors surmised that some occlusal features will place greater adaptive demands on the individual's biologic system than others. Although most individuals are able to compensate for their occlusal disharmony without problems, adaptation for others may lead to a greater risk for TMD. On the basis of this data, the authors stressed the importance of five occlusal conditions, which in isolation or combination are more likely to reach the threshold that exceeds adaptation for an individual and therefore results in TMJ dysfunction. The high-risk malocclusions are discussed later in this chapter.

Occlusal Conditions More Frequently Associated with Temporomandibular Disorders

Anterior Open Bite

Research by Pullinger and colleagues showed a strong relationship between osteoarthritis of the condylar head with a loss of condylar height and an anterior open-bite malocclusion.[254-261] Interestingly, anterior open bite was not commonly associated with disc displacement disorders with or without reduction. Myalgia (i.e., masticatory muscle discomfort) was also a frequent symptom among patients with an anterior open bite. It is not stated in what percentage of individuals the anterior open-bite malocclusion occurred only secondarily as a result of osteoarthritis followed by a loss of condylar height with clockwise rotation of the mandible. Therefore, it is unclear what percentage of the examined individuals with anterior open bites had these finding as part of a developmental jaw deformity.

Overjet (>6 to 7 mm)

In the patients that were studied, an overjet of more than 4 mm was more likely to be associated with osteoarthritis. Only when the overjet was more than 6 mm was there an association with disc displacement. The occurrence of a progressive overjet in adults would be suggestive of active condylysis. It was not stated in what percentage of those individuals with significant overjet (i.e., >6 to 7 mm) the occlusal feature was part of a developmental jaw deformity rather than occurring secondarily only after condylysis with a loss of condylar height.

Centric Relation–Centric Occlusion Slides

Limited occlusal slides (i.e., 1 mm) were common in all patient groups, including the normal controls. CR–CO slides of more than 2 mm were only found in the patients with TMDs. Pullinger found that larger slides were frequently associated with osteoarthritis of the condylar head. He questioned the advisability of the clinicians' automatic attempt to "relieve" the occlusal slides in all individuals—whether by irreversible (e.g., occlusal equilibration, orthodontics, surgery, restorative dentistry) or reversible (e.g., occlusal splints) means—because the observed slide may be either adaptive or inconsequential.[260]

NOTE: The studies by Pullinger and colleagues seem to confirm that the occlusal features of anterior open bites; overjets of more than 6 mm; and CR–CO slides of more than 2 mm are frequently seen in conjunction with osteoarthritis (i.e., loss of condylar height) and less commonly seen with disc displacement disease. Unfortunately, it is not clearly stated which came first: a developmental deformity with malocclusion and then osteoarthritis or condylysis with the loss of posterior facial height that results in a secondary jaw deformity with malocclusion.

Unilateral Maxillary (Lingual) Crossbite

The occlusal feature of unilateral maxillary crossbite occurs in about 10% of the adult population, and it is frequently found in association with TMJ disc displacement with or without disc reduction. Approximately 25% of the patients with non-reducing disc displacements were found to have unilateral maxillary lingual crossbites. This indicated to Pullinger and colleagues that disc displacement may be a biologic adaptive response of the individual to a unilateral maxillary lingual crossbite.[260] The investigators theorized that these individuals may later be prone to osteoarthritis. They suggested that the orthodontic treatment of a unilateral crossbite discovered during childhood should be considered as a preventative measure to reduce the adaptive demands on the masticatory system. They did not come to the same conclusion for the correction of a longstanding

unilateral posterior crossbite in an adult, because skeletal adaptation would have already occurred. Unfortunately, the investigators did not clarify if the crossbites in their study patients were the result of developmental jaw deformities (e.g., asymmetrical mandibular excess) or simply due to the malpositioning of the teeth. They did not specifically comment on whether orthognathic surgery should be carried out in conjunction with orthodontics (if needed) in young adults to correct the presenting malocclusion as a way to prevent the progression of TMD.

Missing Posterior Teeth (≥5)

The investigators found that, when five or more posterior teeth were missing, the incidence of TMD increased. This includes TMD categories of both osteoarthritis and disc displacement with or without osteoarthritis. Pullinger and colleagues further claimed that, when two or four of the "posterior teeth" (bicuspids) were removed as part of an orthodontic treatment protocol, there was a negligible effect on TMD.[260] Other studies carried out by Kayser[156] and Witter[354] have also documented acceptable masticatory function after bicuspid extractions and orthodontic treatment without increased signs and symptoms of TMD.

NOTE: The multifactorial analysis of occlusal factors and TMD disease categories carried out by Pullinger and colleagues showed that, except for the few clearly defined occlusal features described previously, there was a low probability of a malocclusion being associated with TMD. Seligman and colleagues estimated that the overall contribution of occlusal factors for the defining of patients with TMD was only in the range of 10% to 20%.[302] The corollary is that 80% to 90% of the causative factors cannot be explained by patients' presenting malocclusions.

In the Pullinger and Seligman study, it must be assumed that many of the significant overjets observed in the adult subjects were secondary deformities after condylar resorption (i.e., condylysis).[260] The authors did not present evidence to suggest that the presence of significant overbite or overjet plays a role in the pathophysiology of non-arthritic disorders of the TMJ, and they did not show that it was a causative factor in the observed osteoarthritis. They do believe that a combination of unilateral retruded centric position in the presence of a clinically apparent retruded centric position–inter-contact position slide may encourage TMJ disc displacement but that unilateral retruded centric position per se is not associated with TMD. They found that lost molar support (i.e., 4 or 5 posterior teeth) may be associated with osteoarthrosis. They concluded that a dental midline discrepancy is not a predictor of TMD development during adulthood. Anterior open bite is frequent in the presence of condylysis or osteoarthritic changes in the TMJ, but it is probably the result of (rather than the cause of) the osteoarthritic changes. Interestingly, when open bites are observed in children, they are rarely associated with TMJ symptoms. In the pediatric population, the majority of observed open bites represent a developmental jaw deformity rather than a result of osteoarthritis. Nevertheless, it is recommended that, when either a child or an adult is found to have an anterior open bite, radiographic analysis of the condylar head should be undertaken to check for osteoarthritic changes.

Orthodontic Treatment and Its Effects on Temporomandibular Disorders

Review of the Literature

The relationships among malocclusion, orthodontic treatment to improve the occlusion, and the effects on TMD is discussed frequently in the literature.* A spectrum of orthodontic treatment philosophies has been championed by individual practitioners on the basis of presumed effects on TMD. The longitudinal nature of orthodontic treatment, which is often carried out over several years, means that a changing TMD history would also be expected in a control group that is not simultaneously undergoing orthodontic treatment. Therefore, the occurrence of joint noise (e.g., popping, clicking) or myalgia (i.e., masticatory muscle discomfort) during orthodontic treatment may either be the result of the treatment or consistent with the natural history of the untreated individual.

Sadowsky and colleagues reported on 75 adult study patients who had undergone orthodontic treatment with full appliances during adolescence at least 10 years earlier.[290] The treated group was compared with a group of 75 similar adults with untreated malocclusions. The authors also went on to increase both their control and subject groups and to report these additional findings several years later.[291] In the two sequenced studies, respectively, 15% to 21% of the treated subjects presented with one or more signs of TMD (e.g., joint noise), and 29% to 42% had at least one or more symptoms of TMD. There were no statistically significant differences between the treated and untreated (control) groups.

Larsson and colleagues evaluated 23 individuals who had undergone orthodontic treatment during adolescence at least 10 years earlier.[172] Mild TMD was recorded in 8 out of 23 (35%) of the patients, whereas only 1 (4%) had severe TMD. The authors compared this with published epidemiologic controls and stated that there was no difference in the incidence of TMD in the treated group versus the control group.

Dahl and colleagues examined 51 subjects 5 years after the completion of orthodontic treatment and compared them with an untreated control group.[54] The authors found

*References 2, 3, 19, 34, 35, 42, 45-49, 52, 68, 69, 84, 89, 90, 104, 107-109, 127, 134, 137, 138, 150, 155, 159, 164, 168, 169, 182, 184, 185, 203, 224, 237, 240, 245, 253, 268, 271-276, 279, 281, 282, 288-291, 323, 325, 329-332, 334, 352, 358

no statistical differences in TMD between the two groups. The number of subjects in both groups who had at least one mild TMD symptom was relatively high (70% in the treated group versus 90% in the untreated group). Mild symptoms (e.g., joint noise), masticatory muscle fatigue, and stiffness in the lower jaw were frequently observed in both groups.

A study by **Smith and Freer** examined 87 individuals who were treated with full orthodontic bracketing during adolescence.[320] The treatment group (N = 87) was compared with an untreated control group (N = 28). Four years after treatment ended, TMD symptoms were found in 21% of the treated group and 14% of the control group; this was not found to be statistically significant. The one symptom that was statistically more common in the treatment group was a "soft click" (64% of the treated group versus 36% of the untreated group).

Rendell and colleagues examined 462 individuals (90% adolescents and 10% adults) who were undergoing orthodontic treatment in a graduate orthodontic dental school clinic.[267] With the use of a modified Helkimo Index, only 11 of the patients presented with TMD signs or symptoms before treatment. Of the other 451 patients, during the 18-month study, none developed TMD signs or symptoms. For those with preexisting TMD signs or symptoms (N = 11), there was no consistent change in the level of pain or dysfunction reported during treatment.

Kremenak and colleagues reported pretreatment and posttreatment TMD examination data from 109 patients who were treated with full orthodontics.[166,167] The Helkimo Index scores were also compared. No statistically significant differences were noted between the subjects' pretreatment and posttreatment scores. Ninety percent of the patients had Helkimo Index scores that either remained the same or improved, and 10% of the treated patients had scores that worsened. The authors concluded that the orthodontic treatment provided to their patients was not an important etiologic factor for the development of TMD.

Hirata and colleagues examined 102 individuals before and after orthodontic treatment for signs and symptoms of TMD.[128] They compared their treatment group with 41 untreated controls who were matched for age. The incidence of TMD for the treatment and control groups was not statistically significantly different.

Studies have also been carried out to evaluate specific orthodontic treatment maneuvers and their relationship to TMD symptoms and signs. The studied orthodontic maneuvers include the type of orthodontic mechanics used; extraction versus non-extraction therapy; orthodontic treatment and posterior condylar displacement; and orthodontic treatment focused on classic gnathologic principles.

NOTE: In the studies that were reviewed, no consistent benefits or causative factors of TMD were identified on the basis of any specific orthodontic treatment maneuvers carried out in the treatment group as compared with controls.

Orthodontic Treatment Undertaken Specifically to Relieve Temporomandibular Disorders

In everyday dental practice, the establishment of a preferred intra-arch morphologic relationship of the teeth and then the achievement of a satisfactory inter-arch relationship of the teeth in one jaw to those in the other jaw (i.e., occlusion) serves as the basis of treatment objectives. According to a review of the literature, the establishment of a "normal" occlusion through orthodontic mechanics or via other dental means carried out for the purpose of eliminating or preventing the worsening of TMD does not seem warranted. Pullinger and Seligman have stated five potential exceptions to this, which were described previously in this chapter.[255-261] McNamara and colleagues also stated that prudent basic dental treatment objectives will generally include the establishment of the six keys to ideal occlusion as advocated by Andrews, when feasible; the establishment of a limited CR–CO slide (i.e., <2 mm); the limiting of an anterior open bite; the alleviation of significant overjet (i.e., >6 to 7 mm); the limiting of unilateral maxillary lingual crossbites in children; and the establishment of a stable posterior occlusion.

Orthodontic Treatment Undertaken Specifically to Prevent Temporomandibular Disorders

The literature also contains studies that suggest that specific orthodontic maneuvers are effective for the prevention of TMD. The studies by Magnusson and colleagues[187-192] and the study by Egermark and colleagues[89] suggest preventative TMD advantages related to orthodontic occlusal corrections. At 5 and 10 years, these investigators reevaluated a group of 402 children and adolescents who had originally undergone a full course of orthodontic treatment. The Helkimo Index was used to measure clinical signs of TMD in the now older age group (i.e., ≥25 years old). Interestingly, the Helkimo Index outcome was lower in those who had undergone orthodontic treatment as compared with the control group. The studies by Dahl and colleagues and Sadowsky and colleagues also demonstrated a trend (although not a statistically significant one) toward the decreased prevalence of TMD signs and symptoms in orthodontically treated patients.[53,290,291] These studies would require further confirmation before accepting them as credible indications of a cause-and-effect relationship.

Summary of Effects of Orthodontic Treatment on Temporomandibular Disorders

McNamara, Seligman, and Okeson summarize their understanding of the relationship between orthodontic treatment and TMDs as follows[203]:

1. Signs and symptoms of TMD do occur in healthy individuals.

2. Signs and symptoms of TMD increase with age, particularly during adolescence.
3. Orthodontic treatment performed during adolescence generally does not increase or decrease the odds of developing TMD later in life.
4. The extraction of teeth as part of an orthodontic treatment plan does not increase the risk of TMD.
5. There is no elevated risk for TMD associated with any particular type of conventional orthodontic mechanics.
6. Although a stable occlusion is a reasonable orthodontic treatment goal, the inability to achieve a specific gnathologic ideal occlusion does not result in TMD signs or symptoms.
7. No specific method of TMD prevention has been demonstrated to be effective.
8. When more severe TMD signs and symptoms are present, simple forms of treatment can alleviate them in most patients.

NOTE: McNamara, Seligman, and Okeson suggest that until reliable criteria are developed for the treatment of TMD, the dental and medical professions should be encouraged to manage presenting TMJ symptoms with reversible therapies and to only consider permanent alterations in the occlusion (for the specific purpose of treating or preventing TMD) in patients with very unique circumstances. This is in agreement with the National Institute of Health conference guidelines which states that reversible therapies should be used in the primary treatment of TMD[219]

A summary of evidence-based systematic reviews of TMDs was carried out by Rinchuse and McMinn.[283] Their systematic review resulted in the following conclusions:

1. Occlusion, which was once considered the primary and sole cause of TMD, now at best is recognized as having a secondary role in causing it.
2. Conventional orthodontic treatment that improves the occlusion does not cause TMD.
3. The use of occlusal adjustments in orthodontic patients has no evidence-based support for the treatment of TMD.
4. There is evidence-based support for the use of occlusal splints and biofeedback for the treatment of TMD.

Association of Jaw Deformities, Orthognathic Surgery, and Temporomandibular Disorders

Review of the Literature

The association of a jaw deformity with malocclusion and the effects of orthodontic and surgical correction on TMD signs and symptoms have been examined in clinical studies for the past several decades.* The quality of the research and therefore the value of the conclusions vary. There are just three longitudinal prospective controlled clinical trial studies that have been carried out to assess the effects of orthognathic surgery on TMD: the studies by Onizawa and colleagues,[232] Panula and colleagues,[236] and Dervis and colleagues.[67]

The study by **Onizawa and colleagues** sought to evaluate changes in the symptoms of TMD after orthognathic surgery.[232] TMJ symptoms in preoperative orthognathic patients (N = 30) were compared with those of healthy control volunteers (N = 30). Changes in TMD symptoms were evaluated in both groups at 3- and 6-month postoperative intervals. The authors found no significant differences in the incidence of TMJ sounds, deviation of mouth opening, or tenderness on direct palpation of the TMJ and masticatory muscles between patients who were scheduled to undergo orthognathic surgery and control volunteers. The treatment group did not report TMJ symptoms significantly more often than the control subjects. At both the 3- and 6-month postoperative intervals, half or more of the treatment group showed no change in TMJ sounds, deviation of mouth opening, or tenderness on palpation of the TMJ or masticatory muscles. At each evaluation time frame, some individuals in both the control group and the study group exhibited improvement in TMJ symptoms, whereas others showed a change for the worse. Findings of postoperative TMJ sounds were observed to be associated with a reduction of mandibular mobility (i.e., mouth opening). The authors concluded that TMD symptoms did not always show improvement after surgical correction and that, for some patients, the symptoms changed for the worse.

Panula and colleagues carried out research to study the effects of orthognathic surgery on TMJ dysfunction.[236] They completed a prospective follow-up study to examine the influence of orthognathic procedures for the correction of dentofacial deformities on signs and symptoms of TMJ dysfunction. Sixty consecutive patients with dentofacial deformities were examined preoperatively and then twice after orthognathic surgery. The Helkimo Anamnestic and Dysfunction Indices were also given as a questionnaire, and the results were used as part of the determination. The prevalence of headache was also assessed. The average follow-up was 4 years after the initial preoperative examination. A group (N = 20) with a similar type and grade of dentofacial deformity who elected not to undergo surgical correction or any other occlusal therapy served as the control group. The majority of the treatment group (73%) was found to have signs and symptoms of TMJ dysfunction or TMD during the preoperative examination. At the final

*References 6, 7, 25, 32, 33, 36, 55, 61, 62, 66, 67, 96, 98, 103, 105, 110, 111, 123, 124, 131, 144, 145, 154, 157, 158, 160, 174, 180, 187, 189, 211, 225, 226, 232, 236, 247, 292-295, 322, 335, 340, 347, 349, 350, 353, 356, 359

postsurgery examination (i.e., 4 years after surgery), the prevalence of TMD had been reduced to 60%. There was also a dramatic improvement in the prevalence of headache, which was initially experienced by 63% of patients and reduced to 25% of patients at the final visit. The authors concluded that signs and symptoms of TMD, including pain (headache) levels, could be reduced with orthognathic treatment. The risk of an orthognathic patient experiencing new TMD was extremely low. No association could be shown between TMD and any specific type or magnitude of dentofacial deformity.

A study was conducted by **Dervis and colleagues** with the objective of looking at the long-term prevalence of TMD in individuals with a dentofacial deformity who then underwent orthognathic surgery as compared with a control group.[67] Signs and symptoms of TMD in both the dentofacial deformity patients and the control group were evaluated before surgery, 1 week after release of intermaxillary fixation, and 1 and 2 years after surgery with the use of the Helkimo Anamnestic and Dysfunction Indices. At both the initial preoperative examination and the final examination (i.e., 2 years after surgery), signs and symptoms of TMD in the patients who were undergoing orthognathic surgery were compared with those of healthy control subjects. The lengths of condylar pathways during opening and with lateral and protrusive movements were also evaluated with the use of axiography. A statistically significant reduction of TMD symptoms and signs 2 years after surgery as compared with before surgery was found. Interestingly, at the initial examination, the patients with dentofacial deformities who were scheduled to undergo orthognathic surgery did not report TMD signs and symptoms with a greater frequency than other healthy control subjects. At the final examination (i.e., 2 years after surgery), greater improvements in TMD symptoms in the patients who had undergone orthognathic surgery were found as compared with healthy control subjects. The authors also found a statistically significant decrease in masticatory muscle tenderness after surgery. The authors concluded that the "functional status" of the TMJ can be improved with orthognathic surgery. There was no clear association between TMD symptoms and a specific type or pattern of dentofacial deformity.

When comparing and contrasting the three published longitudinal prospective controlled studies that were conducted to determine the effects of orthognathic procedures on TMD symptoms and signs (i.e., Onizawa and colleagues,[232] Panula and colleagues,[236] and Dervis and colleagues[67]), several observations can be made:

1. Patients with dentofacial deformities who were scheduled to undergo orthognathic surgery were not found to have significant differences in their TMD signs and symptoms as compared with patients in the control groups.
2. After orthognathic surgery and orthodontics, signs and symptoms related to TMD were frequently improved.
3. Despite the trend for diminished signs and symptoms of TMD after orthognathic surgery and orthodontics, individuals did not always show improvement; for some, the symptoms changed for the worse.
4. After orthognathic surgery and orthodontic treatment, a decrease in masticatory muscle tenderness was frequent. A decrease in joint tenderness on palpation was also a common occurrence.
5. In one of the three studies reviewed, a decrease in maximum mouth opening was observed, but this was not found to be of clinical importance.

There are other less rigorous reports and studies that can be found in the literature that also analyze the effects of orthodontics and orthognathic surgery carried out for the correction of dentofacial deformities on TMD, including the following:

Karabouta and colleagues reviewed 280 individuals with different patterns of mandibular deformities (e.g., prognathism, retrognathia, open bite, asymmetry) who underwent orthognathic surgery that included sagittal split ramus osteotomies of the mandible. Before surgery, the patients were found to present subjective or objective TMJ dysfunction symptoms with an incidence of 41%.[154] After surgery, the incidence of such symptoms in the same patients was reduced to 11%. Individuals with no preoperative TMJ dysfunction symptoms developed such symptoms with an incidence of 4% after surgery.

White and colleagues set out to review the prevalence of TMD in patients with dentofacial deformities who were undergoing orthognathic surgery.[349] Preoperatively, 49% of their study group presented with TMD. After orthognathic surgery, in the symptomatic patients, 89% had improved temporomandibular function, 3% were unchanged, and 8% developed increased symptoms. Of the preoperative asymptomatic patients, 8% developed TMD after surgery. In the study group, TMD was significantly greater in patients with Class II skeletal deformities as compared with those with Class III tendencies. Overall, temporomandibular function generally improved in both treatment groups (i.e., Class II and Class III) after successful surgery.

Hori and colleagues reported three individual (nonconsecutive) cases in which there was a worsening of preexisting TMJ dysfunction after sagittal split ramus osteotomy of the mandible and 6 weeks of intermaxillary fixation.[131] All three of their patients initially presented specifically seeking treatment of malocclusion for the relief of TMD. All three patients had active TMD before and after their orthognathic procedures.

Aghabeigi and colleagues studied the effect of orthognathic surgery on the TMJ of patients who presented with an anterior open bite.[6] This was a retrospective survey study of patients (N = 83) who presented with an anterior open bite who then underwent orthognathic surgery. The preoperative prevalence of head and neck pain was 32%, TMJ dysfunction was found in 40% of patients, and limited mouth

opening was found in 7%. The overall prevalence of TMJ signs and symptoms was not found to be significantly different after orthognathic surgery. In the study group, an abnormal psychological profile was the most significant factor associated with the presence and persistence of TMJ pain. The authors concluded that orthognathic surgery does not significantly influence TMD in patients with anterior open bites. Female patients—particularly those with abnormal psychological profile—were at higher risk for persistent postoperative TMJ pain.

Westermark and colleagues studied a consecutive series of individuals to understand the effects of orthognathic surgery on the signs and symptoms of TMD.[347] The authors sent a survey questionnaire to a consecutive series of patients (N = 1516) who initially presented with dentofacial deformities and who then underwent orthognathic surgeries that were performed by a group of experienced surgeons at a single institution. The survey was given at least 2 years after surgery. The questions asked in the survey related to joint pain, chewing pain, joint noise, grinding, daytime headache, and morning headache. Preoperatively, 43% of the patients reported subjective symptoms of TMD; postoperatively, 28% of the patients did. This difference suggested an overall beneficial effect of orthognathic surgery on TMD signs and symptoms. Patients with Class II skeletal deformities did not improve as much as with those with Class III skeletal prognathism.

Yamada and colleagues studied the incidence of condylar morphologic change, disc displacement, and signs and symptoms of TMD in individuals set to undergo orthognathic surgery.[359] The study showed preoperative condylar morphologic change (unilateral or bilateral) in 36% of the subjects and in 24% of each joint. Preoperative disc displacement (unilateral or bilateral) was seen in 41% of the subjects and in 30% of each joint. The percentage of condylar morphologic change and disc displacement was higher for individuals with anterior open-bite skeletal Class II growth patterns and lower for patients with skeletal Class III deformities.

Wolford and colleagues studied changes in TMD after orthognathic surgery in a series of 25 patients who were scheduled to undergo bimaxillary surgery.[356] They used magnetic resonance imaging and clinical verification of TMJ articular disc displacement. This was a retrospective evaluation with an average follow up of 2.2 years. Presurgically, 16% of the patients had only TMJ pain, 64% had only TMJ sounds, and 20% had both TMJ pain and sounds. Postoperatively, 24% of the patients had only TMJ pain, 16% had only TMJ sounds, and 60% had both TMJ pain and sounds. Thirty-six percent of the patients had TMJ pain preoperatively; this jumped to 84% after surgery. In addition, the authors claimed that 6 out of 25 of their patients (24%) developed condylar resorption postoperatively, thereby explaining the high incidence of observed Class II open-bite malocclusion. The authors demonstrated that patients with preexisting TMJ dysfunction who undergo bimaxillary osteotomies via the techniques that they used have a high probability of the significant worsening of TMJ dysfunction after surgery. This reported high incidence of TMD and condylar resorption after orthognathic surgery is contrary to all other studies reported in the literature.

Borstlap and colleagues reported about a consecutive series of individuals who were scheduled to undergo sagittal split ramus osteotomies for both Class II mandibular retrognathism and Class III mandibular prognathism.[31] Patients were reviewed preoperatively, immediately postoperatively, and at 3, 6, and 24 months postoperatively. Any signs and symptoms of TMD were documented, including clicking, preauricular pain, maximum mouth opening, protrusive movements, and lateral excursions. The authors documented that 56% of the patients with preexisting TMD had relief of their signs and symptoms. Interestingly, 22% of individuals without preexisting TMD went on to develop clinical signs or symptoms after surgery.

Bock and colleagues studied the importance of temporomandibular function as a factor in the achievement of patient satisfaction after orthognathic surgery.[29] The authors' orthognathic surgery patients (N = 102) were examined at an average of 47 months after surgery. The average age of their patients was 24 years. A patient satisfaction questionnaire and other modalities were reviewed, including the Helkimo Index, clinical findings, nerve function parameters, and the incidence of intraoperative and postoperative complications. The results of their study showed that 91% of patients were satisfied or very satisfied with their overall orthognathic results. Answers to the questionnaires revealed that only 79% of patients would undergo treatment again (i.e., 21% of the patients would not). Statistically significant differences were found in the Helkimo Index and in the pain reported both in the TMJs and with jaw movements between the satisfied and dissatisfied postoperative patients. The authors surmised that the 21% of patients who would not undergo treatment again were more likely to have a greater degree of TMD and limitations with regard to mandibular range of movement. Statistically significant differences were found between those who were satisfied and those who were dissatisfied with surgery on the basis of their Helkimo Indices. Those with pain on palpation in the TMJ region and those with pain experienced during motion of the mandible were more likely to be overall dissatisfied with the treatment. The persistence of postoperative symptoms of TMD, including pain, was the single most important correlation with low patient satisfaction.

Frey and colleagues studied the effects of mandibular advancement procedures and counterclockwise rotation on signs and symptoms of TMD.[102] The study patients were followed for a minimum of 2 years after orthognathic surgery. A series of patients with Angle Class II malocclusions (N = 127) who were undergoing sagittal split ramus osteotomies with advancement and counterclockwise rotation of the mandible were studied to evaluate signs and symptoms of TMD at specific intervals after surgery. The results of the study indicate that counterclockwise rotation of the mandible was associated with more masticatory

muscle tenderness early after surgery, especially in those who were undergoing more than 7 mm of advancement. The combination of extensive mandibular advancement with counterclockwise rotation was also associated with increased TMD symptoms early after surgery. Interestingly, all TMJ symptoms declined by 2 years after surgery. The authors conclude that surgical advancement and counterclockwise rotation of the mandible (i.e., sagittal split ramus osteotomy) is associated with a slight increase in masticatory muscle discomfort during initial recovery. All symptomatology tended to decline over time, which suggests that the amount of advancement and the amount of mandibular counterclockwise rotation should not be considered as risk factors for the long-term development of TMD in patients without preexisting symptoms.

Jung and colleagues retrospectively evaluated the effects of bilateral intraoral vertical ramus osteotomies with setback for the management of Class III skeletal patterns on symptoms that are associated with TMD.[152] Patients were evaluated preoperatively and at intervals (i.e., 1, 3, 6, 12, 18, and 24 months) after surgery for specific parameters, including mouth opening, clicking, and TMJ pain. The authors reported that 94% of those with preoperative joint noise achieved resolution. After surgery, 8% of individuals who were free of noise before surgery developed joint noise afterward. On average, mouth opening was 50 mm before surgery and 49 mm 6 months after surgery. Joints that were free of pain before surgery remained so. Those individuals with preoperative TMJ pain remained free of pain at 18 months. The authors concluded that bilateral intraoral vertical ramus osteotomy with setback as a method of managing Class III skeletal deformities was also effective for the relief of undesirable TMJ symptoms (e.g., joint noise, pain) without negatively affecting mouth opening.

Additional thoughts Concerning Effects of Orthodontics and Orthognathic Surgery on Temporomandibular Disorders

It may not be correct to conclude that orthodontic treatment and orthognathic surgery carried out for the correction of a presenting jaw deformity with malocclusion are the responsible factors for any observed reduction or resolution of TMD signs and symptoms.[18] During the decades that have passed since McCarty and Farrar first defined the diagnoses of internal derangement of the TMJ and then described a disc repositioning procedure to treat it, quality research to evaluate their recommended treatment has been scant.[200] The experience of orthopedic surgeons with regard to the treatment of derangements of the knee should remind us of the need for clinical practice to be based on sound supporting evidence. Moseley and colleagues carried out a controlled clinical trial of arthroscopic surgery (i.e., lysis and lavage or arthroplasty) for osteoarthritis of the knee that should give us pause when considering irreversible TMD treatment.[212] Clinician opinions regarding the effectiveness of lysis and lavage or arthroplasty to manage knee pathology in anticipation of improved function and relief of pain have varied but are currently without consensus. Therefore, a randomized clinical trial that included a placebo group was designed by Moseley and colleagues to address this controversy. To blind the placebo group, a sham surgical procedure was designed as part of the study. The placebo subjects were given a general or regional anesthetic, and incisions were made; however, the arthroscope was not inserted into the joint, and no therapeutic procedure was carried out. The results of the study were that neither treatment group (i.e., lysis and lavage or arthroplasty) offered improved clinical outcomes as compared with the placebo group for osteoarthritis. As a consequence of this evidence-based study, many orthopedic surgeons have changed treatment recommendations for their patients. In any case, Medicare (i.e., the U.S. government medical insurance plan for the elderly) made a decision that it would no longer cover arthroscopy for osteoarthritis of the knee. *Restorative dentists, orthodontists, and orthognathic surgeons should remain circumspect when advising patients regarding irreversible treatment specifically for the purpose of relieving TMD.*

Brief Overview of Okeson's Thinking About Temporomandibular Disorders and the Effects of Occlusion

Okeson believes that, during the normal use of the masticatory system, either local or systemic events can occur that may influence function.[165,227-231] Each individual has a varied ability to tolerate these events without any adverse head and neck effects. It is likely that an individual's tolerance can be influenced by these local or systemic factors. When an event exceeds the individual's physiologic tolerance, the system begins to reveal changes. Each component of the masticatory system can tolerate a certain amount of change. When functional change exceeds a critical level, alteration of the tissues begins. The tissue breakdown site is generally seen in the structure with the least tolerance. Therefore, the breakdown site varies from individual to individual. This is influenced by factors such as the individual's unique maxillofacial morphology, previous head and neck trauma, and local tissue conditions. The weakest link in the masticatory system will break first (i.e. masticatory muscles, TMJs, supporting structures of the teeth, the teeth themselves). Typically, the weakest structures in the system are the masticatory muscles; this is evidenced by muscle tenderness and pain during mandibular movements.

When the TMJs are the weakened structures in the system, the individual may experience joint tenderness and pain. The TMJ can also produce sounds such as clicking or grating. When the individual has increased muscle activity (e.g., bruxism, grinding), then the next weakest link is frequently either the supporting structures of the teeth (i.e., the periodontium) or the teeth themselves. The teeth may

show mobility or wear (e.g., loss of enamel, breakdown of restorations, fracture). Emotional stress seems to be a major influence on bruxing activity.

According to Okeson, the occlusal condition can influence TMDs by way of two mechanisms. One mechanism relates to the introduction of acute changes in the occlusal condition (e.g., recent restorative dentistry with an uneven alteration of the occlusal surfaces). Acute changes can lead to a muscle co-contraction response that leads to a condition of muscle pain. Typically, in these circumstances, new muscle engrams are developed, and the patient adapts with little consequence. A second mechanism relates to a more longstanding variation in the occlusal condition. In these conditions, maximum intercuspation (i.e., centric occlusion) results in the instability of the masticatory system (e.g., a CR–CO discrepancy). The orthopedic instability is not likely to withstand significant compressive loading forces on the TMJs. Dental therapy (i.e., the alteration of the occlusion) will only be helpful for the relief of TMDs when it effectively relieves one of these conditions (i.e., the muscle co-contraction response or the orthopedic instability accompanied by loading forces).

Dr. Okeson concludes that, in general, conventional orthodontic treatment is not an effective method for the prevention of TMD. As a corollary, a review of the literature suggests that patients who receive conventional orthodontic therapy are at no greater risk for the development of TMD than those who do not. According to Dr. Okeson's work, any dental or surgical procedure that produces an occlusal condition that is not in harmony with a musculoskeletal stable position of the joint can—but does not necessarily—predispose the individual to these problems. Alternatively, dental or surgical procedures that reduce one of these occlusal conditions and restore harmony may improve the masticatory system.

Personal Thoughts About the Role of Open Joint Procedures for Temporomandibular Disorders

Unanswered questions remain: What is the appropriate treatment for an individual with a persistent symptomatic intracapsular TMJ disorder that has either not resolved spontaneously or not responded to conservative therapy? In these cases, what are the best studies to use to confirm the diagnosis and then select the appropriate treatment intervention?

A critical review of the literature suggests that outcomes for open TMJ procedures are often unpredictable and may result in further TMD.[73-77] The success rate for "redo" open joint surgery is even less favorable. Among the criteria for an open joint procedure are the following: that the clinician must be confident that the source of the pain or dysfunction is arising from within the joint; that the appropriate use of conservative measures has failed; that the current symptoms are intolerable to a patient who is psychosocially stable; and that a significant long-term success rate for the selected open joint procedure in the specific patient can be assured.

Although an intracapsular lavage procedure with or without limited debridement and lysis of adhesions to relieve inflammation and improve function may be relatively benign in experienced hands, disc repositioning and discectomy have generally proven to be of little statistical value as compared with conservative measures. The technical simplicity of disc repositioning and discectomy procedures has lured many clinicians into offering them as standard options; however, a significant long-term success rate has not been documented.

The overall approach to TMD should aim to avoid further harm to the joint and err on the side of appropriate conservative management to relieve symptoms and to achieve or maintain reasonable function for the long term.

Conclusions

It is estimated that an average of 32% of the population will report at least one symptom of TMD and that an average of 55% will demonstrate at least one clinical sign. Historically, clinicians from a spectrum of medical and dental specialties have considered occlusion to be the major etiologic factor for TMD. A review of the literature suggests that the overall contribution of occlusal factors to identifying patients with TMD to only be in the range of 10% to 20%. The corollary is that 80% to 90% of the causative factors among patients with TMD cannot be explained by those patients' occlusions.

The National Institute of Health conference guidelines state that reversible therapies should be used for the primary treatment of TMD. Despite these words of caution, studies document that, for whatever combination of reasons, the correction of an individual's developmental jaw deformity and malocclusion through successful orthodontics and orthognathic surgery tends to have a positive effect on preexisting TMD in the majority of patients, whereas a minority of patients will get worse.

References

1. Academy of Denture Prosthetics: Glossary of prosthodontic terms (appendix). *J Prosthet Dent* 692:5–34, 1956.
2. Ackerman M: The myth of Janus: orthodontic progress faces orthodontic history. *Am J Orthod Dentofacial Orthop* 123:594–596, 2003.
3. Ackerman M: Evidence-based orthodontics for the 21st century. *J Am Dent Assoc* 135:162–167, 2004.
4. Adams S, Zander HA: Functional tooth contacts in lateral and in centric relation. *J Am Dent Assoc* 69:465–473, 1964.
5. Agerberg A: Occlusal and temporomandibular joint relations: a comparative study. *Cranio* 5:233–238, 1987.
6. Aghabeigi B, Hiranaka D, Keith DA, et al: Effect of orthognathic surgery on the temporomandibular joint in patients with

anterior open bite. *Int J Adult Orthodon Orthognath Surg* 16:153, 2001.
7. Ahlborg G, Finne K: Changes in temporomandibular joint pain dysfunction after surgical correction of dentofacial anomalies. *Int J Oral Maxillofac Surg* 15:707–714, 1986.
8. Ahlgren J, Posselt U: Need of functional analysis and selective grinding in orthodontics: a clinical and electromyographic study. *Acta Odontol Scand* 21:187–217, 1963.
9. Ahlgren J: Mechanism of mastication. *Acta Odontol Scand* 24(Suppl 44):1–109, 1966.
10. Al-Ani Z, Gray RJ, Davies SJ, et al: Stabilization splint therapy for the treatment of temporomandibular myofascial pain: a systematic review. *J Dent Educ* 69:1242–1250, 2005.
11. Alexander PC: Analysis of the cuspid protected occlusion. *J Prosthet Dent* 13:309–317, 1963.
12. Alexander SR, Moore RN, DuBois LM: Mandibular condyle position: comparison of articulator mountings and magnetic resonance imaging. *Am J Orthod Dentofacial Orthop* 104:230–239, 1993.
13. American Academy of Pediatric Dentistry, University of Texas Health Science Center at San Antonio Dental School: Treatment of temporomandibular disorders in children: summary statements and recommendations. *J Am Dent Assoc* 120:265–269, 1990.
14. Andrews LF: The six keys to normal occlusion. *Am J Orthod* 62:296–309, 1972.
15. Angle EH: *Malocclusion of teeth*, ed 7, Philadelphia, 1907, SS White Dental Mfg Co.
16. Ash MM: Current concepts in the etiology, diagnosis and treatment of TMJ and muscle dysfunction. *J Oral Rehabil* 13:1, 1986.
17. Ash MM, Ramjford S: *Occlusion*, ed 4, Philadelphia, 1996, Saunders, p 28.
18. Assael LA: Temporomandibular disorders in surgical practice: does science support treatment decisions? *J Oral Maxillofac Surg* 67:935–936, 2009.
19. Aubrey RB: Occlusal objectives in orthodontic treatment. *Am J Orthod* 74:162–175, 1978.
20. Auerbach SM, Laskin DM, Frantsve LM, Orr T: Depression, pain, exposure to stressful life events, and long-term outcomes in temporomandibular disorder patients. *J Oral Maxillofac Surg* 59:628–634, 2001.
21. Barghi N, Agular T, Martinez C, et al: Prevalence of types of temporomandibular joint clicking in subjects missing posterior teeth. *J Prosthet Dent* 57:617–620, 1987.
22. Bean LR, Thomas CA: Significance of condylar positions in patients with temporomandibular disorders. *J Am Dent Assoc* 114(1):76–77, 1987.
23. Begg PR: Stone Age man's dentition. *Am J Orthod* 40:298–312, 1954.
24. Bell WE: Clinical diagnosis of the pain-dysfunction syndrome. *J Am Dent Assoc* 79:154, 1969.
25. Bell WH, Gonyea W, Finn RA, et al: Muscular rehabilitation after orthognathic surgery. *Oral Surg Oral Med Oral Pathol* 56:229, 1983.
26. Belser UC, Hannam AG: The influence of altered working-side occlusal guidance on masticatory muscles and related jaw movements. *J Prosthet Dent* 53:406–413, 1985.
27. Berry DC, Watkinson AC: Mandibular dysfunction and incisor relationship. *Br Dent J* 144:74–77, 1978.
28. Berry DC, Singh BP: Daily variation in occlusal contacts. *J Prosthet Dent* 50:386–391, 1983.
29. Bock JJ, Fuhrmann RAW: The importance of temporomandibular function for patient satisfaction following orthognathic surgery. *J Orofac Orthop* 68:299–307, 2007.
30. Boero RP: The physiology of splint therapy: a literature review. *Angle Orthod* 59:165–180, 1989.
31. Borstlap WA, Stoelinga PJW, Hoppenreijs TJM, van't Hof MA: Stabilisation of sagittal split advancement osteotomies with miniplates: a prospective, multicentre study with two-year follow-up Part I. Clinical parameters. *Int J Oral Maxillofac Surg* 33:433–441, 2004.
32. Boyd SB, Karas ND, Sinn DP: Recovery of mandibular mobility following orthognathic surgery. *J Oral Maxillofac Surg* 49:924, 1991.
33. Brandt D: Temporomandibular disorders and their association with morphologic malocclusion in children. In Carlson DS, McNamara JA, Ribbens KA, editors: *Developmental aspects of temporomandibular joint disorders. Monograph 16, Craniofacial Growth Series*, Ann Arbor, Mich, 1985, Center for Human Growth and Development, The University of Michigan, pp 296–297.
34. Brodie AG: Differential diagnosis of joint conditions in orthodontia. *Angle Orthod* 4:160–170, 1934.
35. Brodie AG: The temporomandibular joint. *Ill Dent J* 8:2–12, 1939.
36. Buckley MJ, Tulloch JFC, White RP, et al: Complications of orthognathic surgery: a comparison between wire and rigid fixation. *Int J Adult Orthodon Orthognath Surg* 4:69, 1989.
37. Budtz-Jorgensen E, Luan W, Holm-Pedersen P, Fejerskov O: Mandibular dysfunction related to dental, occlusal and prosthetic conditions in a selected elderly population. *Gerodontics* 1:28–33, 1985.
38. Bush FM: Malocclusion, masticatory muscle, and temporomandibular joint tenderness. *J Dent Res* 64:129–133, 1985.
39. Carlsson GE, Egermark I, Magnusson T: Predictors of bruxism, other oral parafunctions, and tooth wear over a 20-year follow-up period. *J Orofac Pain* 17:50–57, 2003.
40. Carlsson GE, Magnusson T, Egermark I: Prediction of demand for treatment of temporomandibular disorders based on a 20-year follow-up study. *J Oral Rehabil* 31:511–517, 2004.
41. Celenza FV: The centric position: replacement and character. *J Prosthet Dent* 30:591–598, 1973.
42. Chiappone RC: A gnathologic approach to orthodontic finishing. *J Clin Orthod* 9:405–417, 1975.
43. Christensen GJ: Is occlusion becoming more confusing? A plea for simplicity. *J Am Dent Assoc* 135:767–770, 2004.
44. Clark JR, Evans RD: Functional occlusion: I. A review. *J Orthod* 28:76–81, 2001.
45. Cohen WE: A study of occlusal interferences in orthodontically treated occlusions and untreated normal occlusions. *Am J Orthod* 51:647, 1965.
46. Cordray FE: A crisis in orthodontists? It's time to look within. *Am J Orthod Dentofacial Orthop* 101:472–476, 1992.
47. Cordray FE: Centric relation treatment and articulator mountings in orthodontics. *Angle Orthod* 66(2):153–158, 1996.
48. Cordray FE: Most condylar discrepancies not detectable clinically. *Am J Orthod Dentofacial Orthop* 120:15A, 2001.
49. Cordray FE: Three-dimensional analysis of models articulated in the seated condylar position from a deprogrammed asymptomatic population: a prospective study. Part 1. *Am J Orthod Dentofacial Orthop* 129:619–630, 2006.
50. Costen JB: Neuralgia and ear symptoms associated with disturbed function of the temporomandibular joint. *JAMA* 107:252–258, 1936.
51. Crawford SD: Condylar axis position, as determined by the occlusion and measured by the CPI instrument, and signs and symptoms of temporomandibular dysfunction. *Angle Orthod* 69:103–115; discussion 115-116, 1999.
52. Creekmore DC, Cetlin NM, Ricketts RM, et al: JCO roundtable: diagnosis and treatment planning. *J Clin Orthod* 26:585–606, 1992.
53. Crider A, Glaros AG, Gevirtz RN: Efficacy of biofeedback-based treatments for temporomandibular disorders. *Appl Psychophysiol Biofeedback* 30:333–345, 2005.
54. Dahl BL, Krogstad BO, Ogaard B, Eckersberg T: Signs and symptoms of craniomandibular disorders in two groups of 19-year-old individuals, one treated orthodontically and the other not. *Acta Scand* 46:89–93, 1988.
55. Dahlberg G, Petersson A, Westesson P-L, Eriksson L: Disk displacement and temporomandibular joint symptoms in orthognathic surgery patients. *Oral Surg Oral Med Oral Pathol* 79:273–277, 1995.
56. Dawson PE: Optimum TMJ condyle position in clinical practice. *Int J Periodontics Restorative Dent* 3:11–31, 1985.
57. Dawson, PE: *Evaluation, diagnosis and treatment of occlusal problems*, ed 2, St. Louis, 1989, C.V. Mosby.

58. Dawson PE: Evidence-based versus experience-based views on occlusion and TMD. *Am J Orthod Dentofacial Orthop* 128:150–151; author reply 151-152, 2005.
59. De Boever JA, Adriaens PA: Occlusal relationship in patients with pain-dysfunction symptoms in the temporomandibular joint. *J Oral Rehabil* 10:1–7, 1983.
60. De Bont LG, Stegenga B: Pathology of temporomandibular joint internal derangement and osteoarthrosis. *Int J Oral Maxillofac Surg* 22:71, 1993.
61. De Clercq CA, Neyt LF, Mommaerts MY, et al: Condylar resorption in orthognathic surgery: A retrospective study. *Int J Adult Orthodon Orthognath Surg* 9:233, 1994.
62. De Clercq CA, Abeloos J, Mommaerts MY: Temporomandibular joint symptoms in an orthognathic surgery population. *J Craniomaxillofac Surg* 23:195–199, 1995.
63. De Laat A, van Steenberghe D: Occlusal relationships and temporomandibular joint dysfunction: Part I. Epidemiologic findings. *J Prosthet Dent* 54:835–842, 1985.
64. De Laat A, van Steenberghe D, Lesaffe E: Occlusal relationship and temporomandibular joint dysfunction: Part II. Correlation between occlusal and articular parameters and symptoms of TMJ dysfunction by means of stepwise logistic regression. *J Prosthet Dent* 55:116–121, 1986.
65. De Laat A, Stappaerts K, Papy S: Counseling and physical therapy as treatment for myofascial pain of the masticatory system. *J Orofac Pain* 17:42–49, 2003.
66. Dechow PC, Carlson DS: Occlusal force after mandibular advancement in adult rhesus monkey. *J Oral Maxillofac Surg* 44:887, 1986.
67. Dervis E, Tuncer E: Long-term evaluations of temporomandibular disorders in patients undergoing orthognathic surgery compared with a control group. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 94:554–560, 2002.
68. Dibbets JMH, van deer Weele L: Orthodontic treatment in relation to symptoms attributed to dysfunction of the temporomandibular joint: a 10-year report of the University of Groningen study. *Am J Orthod Dentofacial Orthop* 91:193–199, 1987.
69. Dibbets JMH, van deer Weele L: Orthodontic treatment modalities and TMJ dysfunction. In Michigan T, editor: *Craniofacial growth theory and orthodontic concept*, Ann Arbor, Mich, 1990, Center for Human Growth and Development, The University of Michigan, pp 153–167.
70. Dijkstra P, Kropmans T, Stegenga B: The association between generalized joint hypermobility and temporomandibular joint disorders: a systematic review. *J Dent Res* 81:158–163, 2002.
71. Dingman RO: Diagnosis and treatment of lesions of the TMJ. *Am J Orthod Oral Surg* 26:374–390, 1940.
72. Dionne RA: Pharmacologic treatments for temporomandibular disorders. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 83:134–142, 1997.
73. Dolwick MF, Katzberg RW, Helms CA: Internal derangement of the temporomandibular joint: fact or fiction? *J Prosthet Dent* 49:415–418, 1983.
74. Dolwick MF, Nitzan DW: TMJ disk surgery: 8-year follow-up evaluation. *Fortschr Kiefer Gesichtschir* 35:162–167, 1990.
75. Dolwick MF, Dimitroulis G: Is there a role for temporomandibular joint surgery? *Br J Oral Maxillofac Surg* 32:307–313, 1994.
76. Dolwick MF, Dimitroulis G: A re-evaluation of the importance of disc position in temporomandibular disorders. *Aust Dent J* 41:184–187, 1996.
77. Dolwick MF: Temporomandibular joint surgery for internal derangement. *Dent Clin North Am* 51:195–208, 2007.
78. Droukas B, Lindee C, Carlsson GE: Occlusion and mandibular dysfunction: a clinical study of patients referred for functional disturbances of the masticatory system. *J Prosthet Dent* 53:402–406, 1985.
79. Du S, Rinchuse DJ, Zullo T, Rinchuse DJ: Reliability of three occlusal classification systems. *Am J Orthod Dentofacial Orthop* 113:463–470, 1998.
80. Dworkin SF, Huggins KH, LeResche L, et al: Epidemiology of signs and symptoms of temporomandibular disorders: clinical signs in cases and controls. *J Am Dent Assoc* 120:273–281, 1990.
81. Dworkin SF, LeResce L: Research diagnostic criteria for temporomandibular disorders: review, criteria, examinations and specifications, critique. *J Craniomandib Disord* 6:301–355, 1992.
82. Dworkin SF: The case for incorporating biobehavioral treatment into TMD management. *J Am Dent Assoc* 127:1607–1610, 1996.
83. Dworkin SF, Sherman J, Mancl L, et al: Reliability, validity, and clinical utility of the research diagnostic criteria for Temporomandibular Disorders Axis II Scales: depression, non-specific physical symptoms, and graded chronic pain. *J Orofac Pain* 16:207–220, 2002.
84. Egermark I, Carlsson GE, Magnusson T: A prospective long-term study of signs and symptoms of temporomandibular disorders in patients who received orthodontic treatment in childhood. *Angle Orthod* 75:645–650, 2005.
85. Egermark-Eriksson I, Ingervall B: Anomalies of occlusion predisposing to occlusal interferences in children. *Angle Orthod* 52:293–299, 1982.
86. Egermark-Eriksson I: Mandibular dysfunction in children and in individuals with dual bite. *Swed Dent J* 6:9–20, 1982.
87. Egermark-Eriksson I: The dependence of mandibular dysfunction in children on functional and morphologic malocclusion. *Am J Orthod* 83:187–194, 1983.
88. Egermark-Eriksson I, Carlsson GE, Magnusson T, Thilander B: A longitudinal study on malocclusion in relation to signs and symptoms of cranio-mandibular disorders in children and adolescents. *Eur J Orthod* 12:399–407, 1990.
89. Egermark I, Thilander B: Craniomandibular disorders with special reference to orthodontic treatment: an evaluation from childhood to adulthood. *Am J Orthod Dentofacial Orthop* 101:28–34, 1992.
90. Egermark I, Blomqvist JE, Cromvik U, Isaksson S: Temporomandibular dysfunction in patients treated with orthodontics in combination with orthognathic surgery. *Eur J Orthod* 22:537–544, 2000.
91. Ekberg E, Nilner M: Treatment outcome of appliance therapy in temporomandibular disorder patients with myofascial pain after 6 and 12 months. *Acta Odontol Scand* 62:343–349, 2004.
92. Ellis PE, Benson PE: Does articulating study casts make a difference to treatment planning? *J Orthod* 30:45–49, 2003.
93. Farrar WB: Characteristics of the condylar path in internal derangements of the TMJ. *J Prosthet Dent* 39:319, 1978.
94. Farrar WB, McCarty WL, Jr: The TMJ dilemma. *J Ala Dent Assoc* 63:19–26, 1979.
95. Farrar WB: Craniomandibular practice: the state of the art; definition and diagnosis. *J Cranio Pract* 1:4–12, 1982-1983.
96. Feinerman DM, Piecuch JF: Long-term effects of orthognatic surgery on the temporomandibular joint: comparison of rigid and nonrigid fixation methods. *Int J Oral Maxillofac Surg* 24:268–272, 1995.
97. Ferrando M, Andreu Y, Galdón MJ, et al: Psychological variables and temporomandibular disorders: distress, coping, and personality. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 98:153–160, 2004.
98. Flynn B, Brown DT, Lapp TH, et al: A comparative study of temporomandibular symptoms following mandibular advancement by bilateral sagittal split osteotomies: rigid versus nonrigid fixation. *Oral Surg Oral Med Oral Pathol* 70:372, 1990.
99. Forssel H, Kirveskari T, Kangasniemi P: Response to occlusal treatment in headache patients previously treated to mock occlusal adjustment. *Acta Odontol Scand* 45:77–80, 1987.
100. Forssell H, Kalso E, Koskela P, et al: Occlusal treatments in temporomandibular disorders: A qualitative systematic review of randomized controlled trials. *Pain* 83:549–560, 1999.
101. Franks AST: The dental arch of patients presenting with temporomandibular joint

dysfunction. *Br J Oral Surg* 5:157–166, 1967.
102. Frey DR, Hatch JP, Van Sickels JE, et al: Effects of surgical mandibular advancement and rotation on signs and symptoms of temporomandibular disorder: a 2-year follow-up study. *Am J Orthod Dentofacial Orthop* 33:490.e1–490.e8, 2008.
103. Fujimura K, Segami N, Sato J, et al: Comparison of the clinical outcomes of patients having sounds in the temporomandibular joint with skeletal mandibular deformities treated by vertico-sagittal ramus osteotomy or vertical ramus osteotomy. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 99:24, 2005.
104. Gazit E, Liebermann MA: Occlusal contacts following orthodontic treatment. *Angle Orthod* 55:316–320, 1985.
105. Gerstner GE, Marchi F, Haerian D: Relationship between antero-posterior maxillomandibular morphology and masticatory jaw movement patterns. *Am J Orthod Dentofacial Orthop* 115:256–266, 1999.
106. Gesch D, Bernhardt O, Kocher T, et al: Association of malocclusion and functional occlusion with signs of temporomandibular disorders in adults: results of the population-based study of health in Pomerania. *Angle Orthod* 74:512–520, 2004.
107. Gianelly AA: Orthodontics, condylar position and TMJ status. *Am J Orthod Dentofacial Orthop* 95:521–523, 1989.
108. Gianelly AA, Anderson CK, Boffa J: Longitudinal evaluation of condylar position in extraction and nonextraction treatment. *Am J Orthod Dentofacial Orthop* 100:416–420, 1991.
109. Gianelly AA, Cozzanic M, Boffa J: Condylar position and maxillary first premolar extraction. *Am J Orthod Dentofacial Orthop* 99:473–476, 1991.
110. Girardot RA Jr: Comparison of condylar position in hyperdivergent and hypodivergent facial skeletal types. *Angle Orthod* 71:240–246, 2001.
111. Glindbueg RW, Laskin DM, Blaustein DI: The effects of immobilization on the primate temporomandibular joint: a histochemical study. *J Oral Maxillofac Surg* 40:3, 1982.
112. Gobetti JP, Turp JC: Fibrosarcoma misdiagnosed as a temporomandibular disorder: a cautionary tale. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 4:404–409, 1998.
113. Goodman P, Greene CS, Laskin DM: Response of patients with myofascial pain-dysfunction syndrome to mock equilibration. *J Am Dent Assoc* 92:955–959, 1976.
114. Graham MM, Buxbaum J, Staling LM: A study of occlusal relationships and the incidence of myofacial pain. *J Prosthet Dent* 47:549–555, 1982.
115. Green JR, Moore CA, Ruark JL, et al: Development of chewing in children from 12 to 48 months: a longitudinal study of EMG patterns. *J Neurophysiol* 77:2704–2716, 1997.
116. Greene CS, Lerman MD, Sutcher HD, Laskin DM: The TMJ pain-dysfunction syndrome: heterogenity of the patient population. *J Am Dent Assoc* 79:1169, 1969.
117. Greene CS, Mohl ND, McNeil C, et al: Temporomandibular disorders and science. *J Prosth Dent* 80:214–215, 1998.
118. Greene CS: The etiology of temporomandibular disorders: implications for treatment. *J Orofac Pain* 15:93–105, 2001.
119. Greene CS, Obrez A: Mandibular repositioning in the treatment of temporomandibular disorder: critical analysis. *Alpha Omegan* 96:40–46, 2003.
120. Griffiths RH: Guidelines for the examination, diagnosis and management of temporomandibular disorders. *J Am Dent Assoc* 106:75–77, 1983.
121. Gunn SM, Woolfolk MW, Faja BW: Malocclusion and TMJ symptoms in migrant children. *J Craniomandib Disord* 2:196–200, 1988.
122. Guralnick W, Kaban LB, Merrill RG: Temporomandibular-joint afflictions. *N Engl J Med* 299:123–129, 1978.
123. Hackney FL, Van Sickels JE, Nummikoski PV: Condylar displacement and temporomandibular joint dysfunction following bilateral sagittal split osteotomy and rigid fixation. *J Oral Maxillofac Surg* 47:223, 1989.
124. Hart DL, Lundquist DO, Davis HC: The effect of vertical dimension on muscular strength. *J Orthop Sports Phys Ther* 3:57–61, 1981.
125. Heikinheimo K, Salmi K, Myllarniemi S, Kirveskari P: A longitudinal study of occlusal disorders at the ages 12 and 15. *Eur J Orthod* 12:190–197, 1990.
126. Helkimo M: Studies on function and dysfunction of the masticatory system: II. Index for anamnestic and clinical dysfunction and occlusal state. *Swed Dent J* 67:101–121, 1974.
127. Henrikson T, Nilner M: Temporomandibular disorders and the need for stomatognathic treatment in orthodontically treated and untreated girls. *Eur J Orthod* 22:283–292, 2000.
128. Hirata RH, Heft MW, Hernandez B, King GJ: Longitudinal study of signs of temporomandibular disorders (TMD) in orthodontically created and untreated groups. *Am J Orthod Dentofacial Orthop* 101:35–40, 1992.
129. Hirsch C, John MT, Drangsholt MT, Mancl LA: Relationship between overbite/overjet and clicking or crepitus of the temporomandibular joint. *J Orofac Pain* 19:218–225, 2005.
130. Hoffman PJ, Silverman SI, Garfinkel L: Comparison of condylar position in centric relation and in centric occlusion in dentulous subjects. *J Prosthet Dent* 30:582–588, 1973.
131. Hori M, Okaue M, Hasegawa M, et al: Worsening of pre-existing TMJ dysfunction following sagittal split osteotomy: a study of three cases. *J Oral Sci* 41:133, 1999.
132. Huang G: Occlusal adjustment for treating and preventing temporomandibular disorders. *Am J Orthod Dentofacial Orthod* 126:138–139, 2004.
133. Huber NU, Hall EH: A comparison of the signs of temporomandibular joint dysfunction and occlusal discrepancies in a symptom-free population of men and women. *Oral Surg Oral Med Oral Pathol* 70:180–183, 1990.
134. Hwang HS, Behrents RG: The effect of orthodontic treatment on centric discrepancy. *J Cranio Pract* 14:132–137, 1996.
135. Ingervall B: Recording of retruded positions of mandible in children: a comparison between registrations in general anaesthesia and with children awake. *Odontol Rev* 19(4):413–421, 1968.
136. Ingervall B: Tooth contacts of the functional and non-functional side in children and young adults. *Arch Oral Biol* 17:191–200, 1972.
137. Ingervall B: Functionally optimal occlusion: the goal of orthodontic treatment. *Am J Orthod* 70:81–90, 1976.
138. Ingervall B: Orthodontic treatment in adults with temporomandibular dysfunction symptoms. *Am J Orthod* 73:551–559, 1978.
139. Ireland VE: The problem of the clicking jaw. *Proc R Soc Med* 44:363–374, 1951.
140. Isaacson D: A biologic concept of occlusion. *J Prevent Dent* 3:12–16, 1976.
141. Israel HA, Diamond B, Saed-Nejad F, Ratcliffe A: The relationship between parafunctional masticatory activity and arthroscopically diagnosed temporomandibular joint pathology. *J Oral Maxillofac Surg* 57:1034–1039, 1999.
142. Israel HA, Syrop SB: The important role of motion in the rehabilitation of patients with mandibular hypomobility: a review of the literature. *Cranio* 15:74–83, 1997.
143. Israel HA, Diamond B, Saed-Nejad F, Ratcliffe A: Osteoarthritis and synovitis as major pathoses of the temporomandibular joint: comparison of clinical diagnosis with arthroscopic morphology. *J Oral Maxillofac Surg* 56:1023–1027, 1998.
144. Iwase M, Sugimori M, Kurachi Y, et al: Changes in bite force and occlusal contacts in patients treated for mandibular prognathism by orthognathic surgery. *J Oral Maxillofac Surg* 56:850, 1998.
145. Iwase M, Ohashi M, Tachibana H, et al: Bite force, occlusal contact area and masticatory efficiency before and after orthognathic surgical correction of mandibular prognathism. *Int J Oral Maxillofac Surg* 35:1102, 2006.

146. Jankelson B, Hoffman GM, Hendron JA Jr: The physiology of the stomatognathic system. *J Am Dent Assoc* 46:375–386, 1952.
147. Johansson A, Unell L, Carlsson GE, et al: Gender difference in symptoms related to temporomandibular disorders in a population of 50-year-old subjects. *J Orofac Pain* 17:29–35, 2003.
148. Johansson A, Unell L, Carlsson GE, et al: Risk factors associated with symptoms of temporomandibular disorders in a population of 50- and 60-year-old subjects. *J Oral Rehabil* 33:473–481, 2006.
149. John MT, Hirsch C, Drangsholt MT, et al: Overbite and overjet are not related to self-report of temporomandibular disorder symptoms. *J Dent Res* 81:164–169, 2002.
150. Johnston LE: Gnathologic assessment of centric slides in postretention orthodontic patients. *J Prosthet Dent* 60:712–715, 1988.
151. Juhl GI, Jensen TS, Norholt SE, et al: Incidence and symptoms of temporomandibular disorders following third molar surgery: a case controlled study. *J Oral Rehabil* 36:199, 2009.
152. Jung H-D, Jung Y-S, Park H-S: The chronologic prevalence of temporomandibular joint disorders associated with bilateral intraoral vertical ramus osteotomy. *J Oral Maxillofac Surg* 67:797–803, 2009.
153. Kaplan RL: Concepts of occlusion: gnathology as a basis for a concept of occlusion. *Dent Clin North Am* 7:577–590, 1963.
154. Karabouta I, Martis C: The TMJ dysfunction syndrome before and after sagittal split osteotomy of the rami. *J Maxillofac Surg* 13:185–188, 1985.
155. Katzberg RW, Westesson P, Tallents RH, Drake CM: Orthodontics and temporomandibular joint internal derangement. *Am J Orthod Dentofacial Orthop* 109:515–520, 1996.
156. Kayser AF: Shortened dental arches and oral function. *J Oral Rehabil* 8:457–462, 1981
157. Kerstens HCJ, Tuinzing DB, Van der Kwast WAM: Temporomandibular joint symptoms in orthognathic surgery. *J Craniomaxillofac Surg* 17:215–218, 1989.
158. Kerstens HCJ, Tuinzing DB, Golding RP, et al: Condylar atrophy and osteoarthrosis after bimaxillary surgery. *Oral Surg Oral Med Oral Pathol* 69:274, 1990.
159. Kim M, Graber T, Viana M: Orthodontics and temporomandibular disorders: a meta-analysis. *Am J Orthod Dentofacial Orthop* 121:438–446, 2002.
160. Kim YG, Oh SH: Effect of mandibular setback surgery on occlusal force. *J Oral Maxillofac Surg* 55:121, 1997.
161. Kircos LT, Ortendahl DA, Arakawa M: Magnetic resonance imaging of the TMJ disc in asymptomatic volunteers. *J Oral Maxillofac Surg* 45:852–854, 1987.
162. Kirveskari P, Alanen P, Jamsa T: Association between craniomandibular disorders and occlusal interferences. *J Prosthet Dent* 62:66–69, 1989.
163. Kirveskari P, Le Bell Y, Salonen M, et al: Effect of elimination of occlusal interference on signs and symptoms of craniomandibular disorder in young adults. *J Oral Rehabil* 16:21–26, 1989.
164. Klar NA, Kulbersh R, Freeland T, Kaczynski R: Maximum intercuspation-centric relation disharmony in 200 consecutively finished cases in a gnathologically oriented practice. *Semin Orthod* 9:109–116, 2003.
165. Klasser GD, Okeson JP: The clinical usefulness of surface electromyography in the diagnosis and treatment of temporomandibular disorders. *J Am Dent Assoc* 137:763–771, 2006.
166. Kremenak CR, Kinser DD, Harman HA, et al: Orthodontic risk factors for Temporomandibular disorders (TMD) I: Premolar extractions. *Am J Orthod Dentofacial Orthop* 101:13–20, 1992.
167. Kremenak CR, Kinser DD, Melcher TJ, et al: Orthodontics as a risk factor for temporomandibular disorders (TMD) II. *Am J Orthod Dentofacial Orthop* 101:21–27, 1992.
168. Kulbersh R, Kaczynski R, Freeland T: Orthodontics and gnathology. *Semin Orthod* 9:93–95, 2003.
169. Kulbersh RDM, Navarro M, Kaczynski R: Condylar distraction effects of standard edgewise therapy versus gnathologically based edgewise therapy. *Semin Orthod* 9:117–127, 2003.
170. Kydd WL: Psychosomatic aspects of temporomandibular joint dysfunction. *J Am Dent Assoc* 59:31–44, 1959.
171. Lader E: Lyme disease misdiagnosed as TMJ syndrome. A case report. *N Y State Dent J* 55(9):50–52, 1989.
172. Larsson E, Ronnerman A: Mandibular dysfunction symptoms in orthodontically treated patients ten years after completion of treatment. *Eur J Orthod* 3:89–94, 1981.
173. Laskin DM: Etiology of the pain-dysfunction syndrome. *J Am Dent Assoc* 79:147–153, 1969.
174. Laskin DM, Ryan WA, Greene CS: Incidence of the temporomandibular symptoms in patients with major skeletal malocclusions: a survey of oral and maxillofacial surgery training programs. *Oral Surg* 61:537–541, 1986.
175. Laskin DM: Temporomandibular disorders: a term past its time? *J Am Dent Assoc* 139:124–128, 2008.
176. LeResche L, Truelove EL, Dworkin SF: Temporomandibular disorders: a survey of dentists' knowledge and beliefs. *J Am Dent Assoc* 124:90–105, 1993.
177. Levitt SR, McKinney MW: Validating the TMJ scale in a national sample of 10,000 patients: demographic and epidemiologic characteristics. *J Orofac Pain* 8:25–35, 1994.
178. Lieberman MA, Gazit E, Fuchs C, Lilos P: Mandibular dysfunction in 10-18 year old school children as related to morphological occlusion. *J Oral Rehabil* 12:209–214, 1985.
179. Lindauer SJ, Sabol G, Isaacson RJ, Davidovitch M: Condylar movement and mandibular rotation during jaw opening. *Am J Orthod Dentofacial Orthop* 107:573–577, 1995.
180. Link JJ, Nickerson JW: Temporomandibular joint internal derangements in an orthognathic surgery population. *Int J Adult Orthodon Orthognath Surg* 7:161–169, 1992.
181. Lobbezoo F, van Denderen RJ, Verheij JG, Naeije M: Reports of SSRI-associated bruxism in the family physician's office. *J Orofac Pain* 15:340–346, 2001.
182. Lopez-Gavito G, Wallen TR, Little RM, Joondeph DR: Anterior open-bite malocclusion: a longitudinal ten-year postretention evaluation of orthodontically treated patients. *Am J Orthod* 87:175–186, 1985.
183. Lundh H, Westesson PL, Kopp S: A three-year follow-up of patients with reciprocal temporomandibular joint clicking. *Oral Surg Oral Med Oral Pathol* 63:530–535, 1983.
184. Luther F: Orthodontics and the temporomandibular joint: where are we now? Part 1. Orthodontics and temporomandibular disorders. *Angle Orthod* 68:295–304, 1998.
185. Luther F: Orthodontics and the temporomandibular joint: where are we now? Part 2. Functional occlusion, malocclusion, and TMD. *Angle Orthod* 68:305–318, 1998.
186. MacMillan HW: Unilateral vs bilateral balanced occlusion. *J Am Dent Assoc* 17:1207–1220, 1930.
187. Magnusson T, Ahlborg G, Finne K, et al: Changes in temporomandibular joint pain-dysfunction after surgical correction of dentofacial deformities. *Int J Oral Maxillofac Surg* 15:707, 1984.
188. Magnusson T, Enbom L: Signs and symptoms of mandibular dysfunction after introduction of experimental balancing-side interferences. *Acta Odontol Scand* 42:129–135, 1984.
189. Magnusson T, Ahlborg G, Svartz K: Function of the masticatory system in 20 patients with mandibular hypo- or hyperplasia after correction by a sagittal split osteotomy. *Int J Oral Maxillofac Surg* 19:289, 1990.
190. Magnusson T, Egermark I, Carlsson GE: A longitudinal epidemiologic study of signs and symptoms of temporomandibular disorders from 15 to 35 years of age. *J Orofac Pain* 14:310–319, 2000.
191. Magnusson T, Egermark I, Carlsson GE: Treatment received, treatment demand, and treatment need for temporomandibular disorders in 35-year-old subjects. *Cranio* 20:11–17, 2002.
192. Magnusson T, Egermarki I, Carlsson GE: A prospective investigation over two decades on signs and symptoms of

temporomandibular disorders and associated variables. A final summary. *Acta Odontol Scand* 63:99–109, 2005.
193. Malki GA, Zawawi KH, Melis M, Hughes CV: Prevalence of bruxism in children receiving treatment for attention deficit hyperactivity disorder: a pilot study. *J Clin Pediatr Dent* 29:63–67, 2004.
194. Management of temporomandibular disorders. National Institute of Health Technology Assessment Conference Statement. *J Am Dent Assoc* 106(1):75–77, 1996.
195. Manfredini D, di Poggio AB, Romagnoli M, et al: Mood spectrum in patients with different painful temporomandibular disorders. *Cranio* 22:234–240, 2004.
196. Mann AW, Pankey LC: Concepts of occlusion: the P.M. philosophy of occlusal rehabilitation. *Dent Clin North Am* 7(3):621–636, 1963.
197. Masserman T: A concept of jaw function with a related clinical application. *J Prosthet Dent* 13:130–140, 1963.
198. McAdam DB: Tooth loading and cuspal guidance in canine and group function occlusions. *J Prosthet Dent* 35:283–290, 1976.
199. McCain JP, Sanders B, Koslin MG, et al: Temporomandibular joint arthroscopy: a 6-year multicenter retrospective study of 4,831 joints. *J Oral Maxillofac Surg* 50:926–930, 1992.
200. McCarty W: Diagnosis and treatment of internal derangements. In Solberg WK, Clark GT, editors: *Temporomandibular joint problems: biologic diagnoses and treatment*, Chicago, 1979, Quintessence Publishing.
201. McLaughlin RP: Malocclusion and the temporomandibular joint—a historical perspective. *Angle Orthod* 85:185–191, 1988.
202. McLean DW: Physiologic vs pathologic occlusion. *J Am Dent Assoc* 25:1583–1594, 1938.
203. McNamara JA Jr, Seligman DA, Okeson JP: Occlusion, orthodontic treatment, and temporomandibular disorders: a review. *J Orofac Pain* 9:73–90, 1995.
204. McNeely ML, Armijo Olivo S, Magee DJ: A systematic review of the effectiveness of physical therapy interventions for temporomandibular disorders. *Phys Ther* 86:710–725, 2006.
205. McNeil C, Mohl ND, Rugh JD, Tanaka TT: Temporomandibular disorders: diagnosis, management, education, and research. *J Am Dent Assoc* 120:253–260, 1990.
206. Medlicott MS, Harris SR: A systematic review of the effectiveness of exercise, manual therapy, electrotherapy, relaxation training, and biofeedback in the management of temporomandibular disorder. *Phys Ther* 86:955–973, 2006.
207. Meng HP, Dibbets JMH, van de Weele L, Boering C: Symptoms of temporomandibular joint dysfunction and predisposing factors. *J Prosthet Dent* 57:215–222, 1987.
208. Mohlin B, Kopp S: A clinical study on the relationship between malocclusions, occlusal interferences and mandibular pain and dysfunction. *Swed Dent J* 2:105–112, 1978.
209. Mohlin B, Ingervall B, Thilander B: Relationship between malocclusion and mandibular dysfunction in Swedish men. *Eur J Orthod* 2:229–237, 1980.
210. Mongini F: Anatomic and clinical evaluation of the relationship between the temporomandibular joint and occlusion. *J Prosthet Dent* 38:539, 1977.
211. Monje F, Delgaad E, Navarro MJ, et al: Changes in temporomandibular joint after mandibular subcondylar osteotomy: an experimental study in rats. *J Oral Maxillofac Surg* 51:1221, 1993.
212. Moseley JB, O'Malley K, Petersen NJ, et al: A controlled trial of arthroscopic surgery for osteoarthritis of the knee. *N Engl J Med* 347:81–88, 2002.
213. Moulton RE: Psychiatric considerations in maxillofacial pain. *J Am Dent Assoc* 51:408, 1955.
214. Moyers RE: An electromyographic analysis of certain muscles in temporomandibular movement. *Am J Orthod* 36:481–515, 1950.
215. Moyers RE: The development of occlusion and temporomandibular joint disorders. In Carlson DS, McNamara JA, Ribbens KA, editors: *Developmental aspects of temporomandibular joint disorders. Monograph 16, Craniofacial Growth Series*, Ann Arbor, Mich, 1985, Center for Human Growth and Development, The University of Michigan, pp 115–120.
216. Muñoz M, Goizueta C, Gil-Díez JL, Díaz FJ: Osteocartilaginous exostosis of the mandibular condyle misdiagnosed as temporomandibular joint dysfunction. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 5:494–495, 1998.
217. Nesbitt BA, Moyers RE, Tenhave T: Adult temporomandibular joint disorder symptomatology and its association with childhood occlusal relations. In Carlson DS, McNamara JA, Ribbens KA, editors: *Developmental aspects of temporomandibular joint disorders. Monograph 16, Craniofacial Growth Series*, Ann Arbor, Mich, 1985, Center for Human Growth and Development, The University of Michigan.
218. NIH Technology Assessment Conference Statement: management of temporomandibular disorders. *J Am Dent Assoc* 127:1595–1606, 1996.
219. NIH Technology Assessment Panel on integration of behavioral and relaxation approaches into the treatment of chronic pain and insomnia: integration of behavioral and relaxation approaches into the treatment of chronic pain and insomnia. *JAMA* 276:313–318, 1996.
220. Nilner M: Prevalence of functional disturbances and disorders of the stomatognathic system in 15-18 year olds. *Swed Dent J* 5:189–197, 1981.
221. Nilsson IM: Reliability, validity, incidence and impact of temporomandibular pain disorders in adolescents. *Swed Dent J Suppl* 7–86, 2007.
222. Nitzan DW, Dolwick MF, Heft MW: Arthroscopic lavage and lysis of the temporomandibular joint: a change in perspective. *J Oral Maxillofac Surg* 48:798–802, 1990.
223. Nomura K, Vitti M, Oliveira AS, et al: Use of the Fonseca's questionnaire to assess the prevalence and severity of temporomandibular disorders in Brazilian dental undergraduates. *Braz Dent J* 18:163–167, 2007.
224. O'Reilly MT, Rinchuse DJ, Close J: Class II elastics and extractions and temporomandibular disorders: a longitudinal prospective study. *Am J Orthod Dentofacial Orthop* 103:459–463, 1993.
225. O'Ryan F, Epker BN: Surgical orthodontics and the temporomandibular joint: II. Mandibular advancement via modified sagittal split ramus osteotomies. *Am J Orthod* 83:418, 1983.
226. O'Ryan F, Epker BN: Temporomandibular joint function and morphology: observations on the spectra of normalcy. *Oral Surg* 58:272–279, 1984.
227. Okeson JP, ed: *Orofacial pain: guidelines for assessment, diagnoses and management*, Chicago, 1996, Quintessence.
228. Okeson JP: Current diagnostic classification schema and assessment of patients with temporomandibular disorders. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 83:61–64, 1997.
229. Okeson JP: *Management of temporomandibular disorders and occlusion*, ed 7, St. Louis, 2012, Elsevier.
230. Okeson JP: *Bell's orofacial pains: the clinical management of orofacial pain*, ed 6, Chicago, 2004, Quintessence Publishing.
231. Okeson JP: The international classification of headache disorders, 2nd ed. *Cephalalgia* 24(Suppl 1):24–160, 2004.
232. Onizawa K, Schmelzen R, Vogt S: Alteration of temporomandibular joint symptoms after orthognathic surgery comparison with healthy volunteers. *J Oral Maxillofac Surg* 53:117, 1995.
233. Owens S, Buschang PH, Throckmorton GS, et al: Masticatory performance and areas of occlusal contact and near contact in subjects with normal occlusion and malocclusion. *Am J Orthod Dentofacial Orthop* 121:602–609, 2002.
234. Pameijer JH, Glickman I, Roeber FW: Intraoral occlusal telemetry: Part III. Tooth contacts in chewing, swallowing and bruxism. *J Periodontol* 40:253–258, 1969.
235. Pameijer JA, Brion W, Glickman I, Roeber FW: Intraoral occlusal telemetry: Part V. Effect of occlusal adjustment upon tooth contact during chewing and swallowing. *J Prosthet Dent* 24:492–497, 1970.
236. Panula K, Somppi M, Finne K, Oikarinen K: Effects of orthognathic surgery on

temporomandibular joint dysfunction: a controlled prospective 4-year follow-up study. *Int J Oral Maxillofac Surg* 29:183–187, 2000.
237. Paquette DE, Beattie JR, Johnston LE, Jr: A long-term comparison of nonextraction and premolar extraction edgewise therapy in "borderline" Class II patients. *Am J Orthod Dentofacial Orthop* 102:1–14, 1992.
238. Parker MW, Holmes EK, Terezhalmy GT: Personality characteristics of patients with temporomandibular disorders: diagnostic and therapeutic implications. *J Orofac Pain* 7:337–344, 1993.
239. Parker WS: Centric relation and centric occlusion: an orthodontic responsibility. *Am J Orthod* 74:481–500, 1978.
240. Peck S: Philosophy and evidence-based orthodontics. *Angle Orthod* 67:403, 1997.
241. Perrott DH, Alborzi A, Kaban LB, Helms CA: A prospective evaluation of the effectiveness of temporomandibular joint arthroscopy. *J Oral Maxillofac Surg* 48:1029–1032, 1990.
242. Perry HT, Lammie GA, Main J, Teuscher GW: Occlusion in a stress situation. *J Am Dent Assoc* 60:626–633, 1960.
243. Perry HT: Relation of occlusion to temporomandibular joint dysfunction. *J Am Dent Assoc* 79:137–141, 1969.
244. Perry HT: Adolescent temporomandibular joint dysfunction. *Am J Orthod* 63:517–525, 1973.
245. Perry HT: Mandibular function: an orthodontic responsibility. *Am J Orthod* 67:316, 1975.
246. Perry HT: Temporomandibular joint and occlusion. *Angle Orthod* 46:284–293, 1976.
247. Peterssen A, Willmar-Hogeman K: Radiographic changes of the temporomandibular joint after oblique sliding osteotomy of the mandibular rami. *Int J Oral Maxillofac Surg* 18:27, 1989.
248. Plata M, Barghi N, Rey R: Clinical evaluation of induced occlusal disharmonies. *J Dent Res* 61:203–208, 1982.
249. Posselt U: Studies in the mobility of the human mandible. *Acta Odontol Scand* 10(Suppl 10):1–160, 1952.
250. Posselt U: The temporomandibular joint syndrome and occlusion. *J Prosthet Dent* 25:432–438, 1971.
251. Poveda Roda R, Bagan JV, Diaz Fernandez JM, et al: Review of temporomandibular joint pathology: Part I. Classification, epidemiology and risk factors. *Med Oral Patol Oral Cir Bucal* 12:E292–E298, 2007.
252. Prentiss HJ: Preliminary report upon the temporomandibular articulation in the human type. *Dent Cosmos* 60:505, 1918.
253. Proffit WR: Who said that? *Am J Orthod* 87:166, 1985.
254. Pullinger AG, Solberg WK, Hollender L, Petersson A: Relationship of mandibular condylar position to dental occlusal factors in an asymptomatic population. *Am J Orthod Dentofacial Orthop* 91:200–206, 1987.
255. Pullinger AG, Seligman DA, Solberg WK: Temporomandibular disorders: Part II. Occlusal factors associated with temporomandibular joint tenderness and dysfunction. *J Prosthet Dent* 59:363–367, 1988.
256. Pullinger AG, Seligman DA: Overbite and overjet characteristics of refined diagnostic groups of temporomandibular disorder patients. *Am J Orthod Dentofacial Orthop* 100:401–415, 1991.
257. Pullinger AG, Seligman DA: Trauma history in diagnostic groups of temporomandibular disorders. *Oral Surg Oral Med Oral Pathol* 71:529–534, 1991.
258. Pullinger AG, Seligman DA: The degree to which attrition characterizes differentiated patient groups of temporomandibular disorders. *J Orofac Pain* 7:196–208, 1993.
259. Pullinger AG, Seligman DA, Gornbein JA: A multiple logistic regression analysis of the risk and relative odds of temporomandibular disorders as a function of common occlusal features. *J Dent Res* 72:968–979, 1993.
260. Pullinger AG, Seligman DA: Quantification and validation of predictive values of occlusal variables in temporomandibular disorders using a multifactorial analysis. *J Prosthet Dent* 83:66–75, 2000.
261. Pullinger AG, Seligman DA: Multifactorial analysis of differences in temporomandibular joint hard tissue anatomic relationships between disk displacement with and without reduction in women. *J Prosthet Dent* 86:407–419, 2001.
262. Ramfjord SP: Dysfunction temporomandibular joint and muscle pain. *J Prosthet Dent* 11:353–374, 1961.
263. Ramfjord SP, Ash MM: *Occlusion*, ed 3, Philadelphia, 1983, Saunders.
264. Ramfjord SP: Current concepts on the development of TMJ dysfunction. In Carlson DS, McNamara JA, Ribbens KA, editors: *Developmental aspects of temporomandibular joint disorders. Monograph 16, Craniofacial Growth Series*, Ann Arbor, Mich, 1985, Center for Human Growth and Development, The University of Michigan, pp 81–97.
265. Randow K, Carlsson K, Edlund J, Oberry T: The effect of an occlusal interference on the masticatory system: an experimental investigation. *Odontol Rev* 27:245–256, 1976.
266. Raphael KG, Klausner JJ, Nayak S, Marbach JJ: Complementary and alternative therapy use by patients with myofascial temporomandibular disorders. *J Orofac Pain* 17:36–41, 2003.
267. Rendell JK, Norton LA, Gay T: Orthodontic treatment and temporomandibular disorders. *Am J Orthod Dentofacial Orthop* 101:84–87, 1992.
268. Reynders RM: Orthodontics and temporomandibular disorders: a review of the literature (1966-1988). *Am J Orthod Dentofacial Orthop* 97:463–471, 1990.
269. Reynolds JM: The organization of occlusion for natural teeth. *J Prosthet Dent* 26:56–67, 1971.
270. Ricketts RM: Clinical implications of the temporomandibular joint. *J Am Dent Assoc* 52:416–439, 1966.
271. Rinchuse DJ, Sassouni V: An evaluation of eccentric occlusal contacts in orthodontically treated subjects. *Am J Orthod Dentofacial Orthop* 82(3):251–256, 1982.
272. Rinchuse DJ, Rinchuse DJ: The impact of the American Dental Association's guidelines for the examination, diagnosis, and management of temporomandibular disorders on orthodontic practice. *Am J Orthod* 83:518–522, 1983.
273. Rinchuse DJ, Sassouni V: An evaluation of functional occlusal interferences in orthodontically treated and untreated subjects. *Angle Orthod* 53(2):122–130, 1983.
274. Rinchuse DJ: Does orthodontics cause TMJ disorders? *Orthod Rev* 1:11, 1987.
275. Rinchuse DJ: Counterpoint: preventing adverse effects on the temporomandibular joint through orthodontic treatment. *Am J Orthod Dentofacial Orthop* 91:500–506, 1987.
276. Rinchuse DJ, Rinchuse DJ: Ambiguities of Angle's classification. *Angle Orthod* 59:295–298, 1989.
277. Rinchuse DJ, Abraham J, Medwid L, Mortimer R: TMJ sounds: are they a common finding or are they indicative of pathosis/dysfunction? *Am J Orthod Dentofacial Orthop* 98:512–545, 1990.
278. Rinchuse DJ: CR-CO discrepancy. *Angle Orthod* 65:4–6, 1995.
279. Rinchuse DJ, Rinchuse DJ: Orthodontics justified as a profession. *Am J Orthod Dentofacial Orthop* 121:93–96, 2002.
280. Rinchuse DJ, Kandasamy S: Evidence-based versus experience-based views on occlusion and TMD. *Am J Orthod Dentofacial Orthop* 127:249–254, 2005.
281. Rinchuse DJ, Sweitzer EM, Rinchuse DJ, Rinchuse DL: Understanding science and evidence-based decision making in orthodontics. *Am J Orthod Dentofacial Orthop* 127:618–624, 2005.
282. Rinchuse DJ, Kandasamy S: Articulators in orthodontics: an evidence-based perspective. *Am J Orthod Dentofacial Orthop* 129:299–308, 2006.
283. Rinchuse DJ, McMinn JT: Summary of evidence-based systematic reviews of canine protected occlusion. *Am J Orthod Dentofacial Orthop* 130:715–720, 2006.
284. Rinchuse DJ, Kandasamy S: Centric relation: a historical and contemporary orthodontic perspective. *J Am Dent Assoc* 137:494–501, 2006.
285. Rinchuse DJ, Kandasamy S, Sciote J: A contemporary and evidence-based view of canine protected occlusion. *Am J Orthod Dentofacial Orthop* 132:90–102, 2007.
286. Riolo ML, Brandt D, Tenhave T: Associations between occlusal characteristics

and signs and symptoms of TMJ dysfunction in children and young adults. *Am J Orthod Dentofacial Orthop* 92:467–477, 1987.
287. Roth RH: Temporomandibular pain-dysfunction and occlusal relationships. *Angle Orthod* 43:136–154, 1973.
288. Roth RH: Functional occlusal for the orthodontist, Part I. *J Clin Orthod* 15(1):32–51, 1981.
289. Roth RH: Functional occlusion for the orthodontist, Part II. *J Clin Orthod* 25:100–123, 1981.
290. Sadowsky C, BeGole EA: Long-term status of temporomandibular joint function and functional occlusion after orthodontic treatment. *Am J Orthod* 78:201–212, 1980.
291. Sadowsky C, Polson AM: Long-term status of temporomandibular disorders and functional occlusion after orthodontic treatment: results of two long-term studies. *Am J Orthod* 86:386–390, 1984.
292. Sanroman JF, Gonzalez JM, Del Hoyo A, et al: Morphometric and morphological changes in the temporomandibular joint after orthognathic surgery: a magnetic resonance imaging and computed tomography prospective study. *J Craniomaxillofac Surg* 25:139, 1997.
293. Schellhas KP, Piper MA, Omlie MR: Facial skeleton remodeling due to temporomandibular joint degeneration: an imaging study of 100 patients. *Am J Neuroradiol* 11:541–551, 1990.
294. Schellhas KP, Piper MA, Bessette RW, et al: Mandibular retrusion, temporomandibular joint derangement, and orthognathic surgery planning. *Plast Reconstr Surg* 90:218, 1992.
295. Schellhas KP, Pollei SR, Wilkes CH: Pediatric internal derangements of the temporomandibular joint: effect on facial development. *Am J Orthod Dentofac Orthop* 104:51–59, 1993.
296. Schiffman E, Fricton JR: Epidemiology of TMJ and craniofacial pain. In Fricton JR, Hathaway KM, editors: *TMJ and craniofacial pain: diagnosis and management*, St. Louis, 1988, Ishiyaku Euroamerica.
297. Schütz TC, Andersen ML, Tufik S: Effects of COX-2 inhibitor in temporomandibular joint acute inflammation. *J Dent Res* 86:475–479, 2007.
298. Schuyler C: Fundamental principles in the correction of occlusal disharmony, natural and artificial. *JADA* 22:1193–1202, 1935.
299. Schuyler C: Factors contributing to traumatic occlusion. *J Prosthet Dent* 11:708–716, 1961.
300. Schwartz LL: A TMJ pain-dysfunction syndrome. *J Chron Dis* 3:284–293, 1956.
301. Schwartz LL, Cobin HP: Symptoms association with the temporomandibular joint. *Oral Surg* 10:339–344, 1957.
302. Seligman DA, Pullinger AG: The role of intercuspal occlusal relationships in temporomandibular disorders: a review. *J Craniomandib Disord* 5:96–106, 1991.
303. Seligman DA, Pullinger AG: The degree to which dental attrition in modern society is a function of age and of canine contact. *J Orofac Pain* 9:266–275, 1995.
304. Seligman DA, Pullinger AG: A multiple stepwise logistic regression analysis of trauma history and 16 other history and dental cofactors in females with temporomandibular disorders. *J Orofac Pain* 10:351–361, 1996.
305. Seligman DA: Emphasis on TMD. *J Am Dent Assoc* 128:140–142, 1997.
306. Seligman DA, Pullinger AG: Analysis of occlusal variables, dental attrition, and age for distinguishing healthy controls from female patients with intracapsular temporomandibular disorders. *J Prosthet Dent* 83:76–82, 2000.
307. Seligman D, Pullinger A: Reply. *J Prosthet Dent* 84:114–115, 2000.
308. Shanahan TE, Alexander L: Mandibular and articular movements. *J Prosthet Dent* 12:82–86, 1962.
309. Shanahan TE, Alexander L: Mandibular and articular movements. Part IV. Concepts of lateral movements and condyle paths. *J Prosthet Dent* 14:279–289, 1963.
310. Shaw DM: Form and function in teeth and a rational unifying principle applied to interpretation. *Int J Orthod* 10:703–718, 1924.
311. Sheppard IM, Jacobson HG, Zaino C, Poppel MH: Dynamics of occlusion. *J Am Dent Assoc* 58(3):77–84, 1959.
312. Sheppard IM, Sheppard SM: Maximal incisal opening: a diagnostic index? *J Dent Med* 20:13–15, 1965.
313. Sheppard IM, Sheppard SM: Range of condylar movement during mandibular opening. *J Prosthet Dent* 15:263–271, 1965.
314. Shupe RJ, Mohammed SE, Christensen LV, et al: Effects of occlusal guidance on jaw muscle activity. *J Prosthet Dent* 51:811–818, 1984.
315. Sicher H: Temporomandibular articulation in mandibular overclosure. *J Am Dent Assoc* 36:131–139, 1948.
316. Sicher H: Positions and movements of the mandible. *J Am Dent Assoc* 48:620–625, 1954.
317. Sicher H: Structural and functional basis for disorders of the temporomandibular joint articulation. *J Oral Surg* 13:275–279, 1955.
318. Sicher H, DuBrul EL: *Oral anatomy*, St. Louis, 1970, Mosby.
319. Silverman MM: Comparative accuracy of the gnathological and neuromuscular concepts. *J Am Dent Assoc* 96:559–565, 1978.
320. Smith A, Freer TJ: Post-orthodontic occlusal function. *Austral Dent J* 34:301–309, 1989.
321. Solberg WK, Bibb CA, Nordstrom BB, Hanson TL: Malocclusion associated with temporomandibular joint changes in young adults at autopsy. *Am J Orthod* 89:326–330, 1986.
322. Sostmann M, Meyer J, Berten JL: TMJ function following orthognathic surgery. *Dtsch Stomatol* 41:487–489, 1991.
323. Storey AT: Orthodontic treatment and temporomandibular function: the etiology of TM disorders. In Carlson DS, editor: *Craniofacial growth theory and orthodontic treatment. Monograph 23, Craniofacial Growth Series*, Ann Arbor, Mich, 1999, Center for Human Growth and Development, The University of Michigan, pp 106–107.
324. Stowell AW, Gatchel RJ, Wildenstein L: Cost-effectiveness of treatments for temporomandibular disorders: biopsychosocial intervention versus treatment as usual. *J Am Dent Assoc* 138:202–208, 2007.
325. Sved A: Equilibration as a post-retention measure. *Dent Clin North Am* 815–820, 1960.
326. The American Academy of Orofacial Pain: *Temporomandibular disorders, guidelines for classification, assessment, and management*, Carol Stream, Ill, 1993, Quintessence Publishing Co Inc.
327. Thilander B, Rubio G, Pena L, de Mayorga C: Prevalence of temporomandibular dysfunction and its association with malocclusion in children and adolescents: an epidemiologic study related to specified stages of dental development. *Angle Orthod* 72:146–154, 2002.
328. Thompson JR: Concepts regarding function of the stomatognathic system. *J Am Dent Assoc* 48:626–637, 1954.
329. Thompson JR: Abnormal function of the stomatognathic system and its orthodontic implications. *Am J Orthod* 48:758–765, 1962.
330. Thompson JR: Differentiation of functional and structural dental malocclusion and its implication to treatment. *Angle Orthod* 42:252–262, 1972.
331. Thompson JR: Abnormal function of the temporomandibular joints and related musculature: orthodontic implications, Part I. *Angle Orthod* 56(2):143–163, 1986.
332. Thompson JR: Abnormal function of the temporomandibular joints and related musculature: orthodontic implications, Part II. *Angle Orthod* 56(3):181–195, 1986.
333. Throckmorton GS, Buschang PH, Hayasaki H, Pinto AS: Changes in the masticatory cycle following treatment of posterior unilateral crossbite in children. *Am J Orthod Dentofacial Orthop* 120:521–529, 2001.
334. Timm TA, Herremans EL, Ash M: Occlusion and orthodontics. *Am J Orthod* 70:138–145, 1976.
335. Timmis DP, Aragon SB, Van Sickels JE: Masticatory dysfunction with rigid and nonrigid osteosynthesis of sagittal split osteotomies. *Oral Surg Oral Med Oral Pathol* 62:119–123, 1986.
336. Truelove E, Huggins KH, Mancl L, Dworkin SF: The efficacy of traditional, low-cost and nonsplint therapies for temporomandibular disorder: a randomized controlled trial. *J Am Dent Assoc* 137:1099–1107, 2006.
337. Tsukiyama Y, Baba K, Clark GT: An evidence-based assessment of occlusal

adjustment as a treatment for temporomandibular disorders. *J Prosthet Dent* 86:57–66, 2001.
338. Turk DC: Psychosocial and behavioral assessment of patients with temporomandibular disorders: diagnostic and treatment implications. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 83:65–71, 1997.
339. Turner JA, Dworkin SF, Mancl L, et al: The roles of beliefs, catastrophizing, and coping in the functioning of patients with temporomandibular disorders. *Pain* 92:41–51, 2001.
340. Upton G, Scott R, Hayward J: Major maxillomandibular malrelations and temporomandibular joint pain-dysfunction. *J Prosthet Dent* 51:686, 1984.
341. Vanderos A: The relationship between craniomandibular dysfunction and malocclusion in white children with unilateral cleft lip and palate. *Cranio* 7:200–204, 1989.
342. Vesper M, Röder K, Siegert J, et al: Acute temporomandibular joint arthritis after Lyme borreliosis. *Mund Kiefer Gesichtschir* 4:258–260, 2001.
343. Wabeke KB, Hansson TL, Hoogstraten J, van der Kuy P: Temporomandibular joint clicking: a literature overview. *J Craniomandib Disord* 3:163–173, 1989.
344. Wang B, Yang C, Cai XY, et al: Malocclusion as a common occurrence in temporomandibular joint arthroscopic disc repositioning: outcomes at 49 days after surgery. *J Oral Maxillofac Surg* 69:1587–1593, 2011.
345. Wänman A: Longitudinal course of symptoms of craniomandibular disorders in men and woman: a 10-year followup study of an epidemiologic sample. *Acta Odontol Scand* 54:337–342, 1996.
346. Weiland FJ: The role of occlusal discrepancies in the long-term stability of the mandibular arch. *Eur J Orthod* 16:521–529, 1994.
347. Westermark A, Shayeghi F, Thor A: Temporomandibular dysfunction in 1516 patients before and after orthognathic surgery. *Int J Adult Orthodon Orthognath Surg* 16:145–151, 2001.
348. Westesson P-L, Eriksson L, Kurita K: Reliability of a negative clinical temporomandibular joint examination: prevalence of disk displacement in asymptomatic temporomandibular joints. *Oral Surg Oral Med Oral Path* 68:551–554, 1989.
349. White CS, Dolwick MF: Prevalence and variance of temporomandibular dysfunction in orthognathic surgery patients. *Int J Adult Orthodon Orthognath Surg* 7:7–14, 1992.
350. Will LA, Joondeph DR, Hohl TH, et al: Condylar position following mandibular advancement: its relationship to relapse. *J Oral Maxillofac Surg* 42:578, 1984.
351. Williamson EH: Occlusion: understanding or misunderstanding. *Angle Orthod* 46:86–93, 1976.
352. Williamson EH: Temporomandibular dysfunction in pretreatment adolescent patients. *Am J Orthod* 72:429–433, 1977.
353. Wisth PJ: Mandibular function and dysfunction in patients with mandibular prognathism. *Am J Orthod* 85:193–198, 1984.
354. Witter DJ, Elteren PV, Kayser AF, Van Rossum GM: Oral comfort in shortened dental arches. *J Oral Rehab* 17:137–143, 1990
355. Woda A, Vigneron P, Kay D: Non-functional and functional occlusal contacts: a review of the literature. *J Prosthet Dent* 42:335–341, 1979.
356. Wolford LM, Reiche-Fischel O, Mehra P: Changes in TMJ dysfunction after orthognathic surgery. *J Oral Maxillofac Surg* 61:655, 2002.
357. Wood DP: The CR-CO discrepancy: author's response. *Angle Orthod* 65:6–8, 1995.
358. Wyatt WE: Preventing adverse effects on the temporomandibular joint through orthodontic treatment. *Am J Orthod Dentofacial Orthop* 91:493–499, 1987.
359. Yamada K, Hanada K, Hayashi T, et al: Condylar bony change, disk displacement, and signs and symptoms of TMJ disorders in orthognathic surgery patients. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 91:603, 2001.
360. Zarb GA, Speck JE: The treatment of temporomandibular joint dysfunction: a retrospective study. *J Prosthet Dent* 38:420, 1977.

10

Nasal Airway Considerations in the Evaluation and Treatment of Dentofacial Deformities

JEFFREY C. POSNICK, DMD, MD

- Frequent Causes of Chronic Obstructive Nasal Breathing
- The Effects of Respiratory Pattern and Jaw Posture on Maxillofacial Growth
- Simultaneous Management of Chronic Obstructed Nasal Breathing and the Presenting Dentofacial Deformity
- Approaching the Nasal Airway Through Le Fort I Down-Fracture
- Management of Residual Postoperative Nasal Obstruction
- Effects of Maxillary Arch Expansion and Nasal Floor Recontouring on the Airway
- Comprehensive Approach to Chronic Nasal Airway Obstruction
- Case Presentation
- Conclusions

The efficiency of care for the individual with a dentofacial deformity requires the collaborative management of any presenting upper airway obstruction, facial dysmorphology, and malocclusion or dental rehabilitative needs. At the time of orthognathic surgery, the surgeon has an opportunity to further contribute to the patient's quality of life by seamlessly addressing any longstanding breathing and sinus drainage difficulties that coexist with the jaw deformity. Breathing difficulties may range from isolated symptomatic nasal airway obstruction to obstructive sleep apnea and should not be overlooked. The impact and frequency of the combined occurrence of nasal obstruction, sinus disease, and maxillary deformity have been recognized and discussed in established literature for well over 100 years:

"Irregularities of the teeth being so frequently associated with some pathologic obstruction of the nasal passages or nasopharynx, this fact should ever be present in the operator's mind and suitable examination being made; and in case [as is often found] the oral deformity be complicated by the presence of hypertrophied faucal tonsils, adenoid hypertrophies in the vault of the pharynx, or obstruction of the nasal passages, the orthodontist's work can only be made complete by the assistance of a rhinologist and laryngologist."[2]

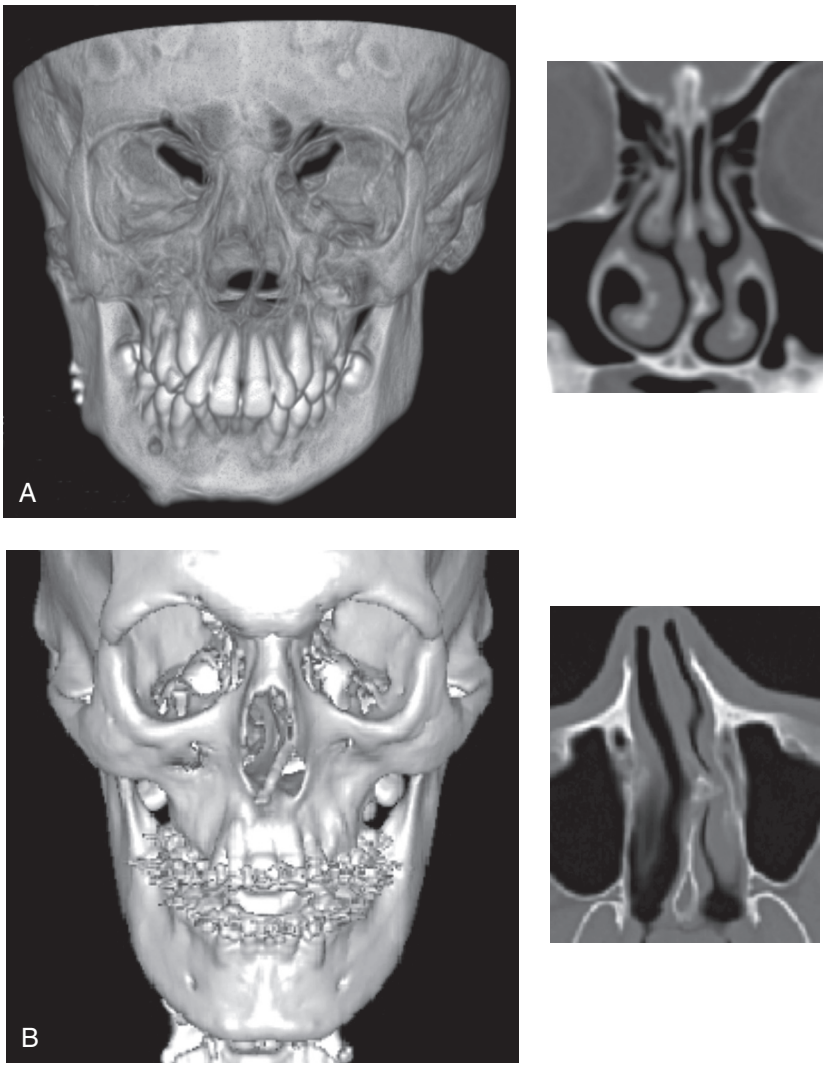
—EDWARD H. ANGLE, MD, DDS (1907)

"The nasal fossae are bounded by the maxillary bones and by bones attached to the maxillae; therefore deformities of maxillary bones are bound to influence the size, shape and patency of the nasal fossae. Nasal obstruction may be due directly to deformity or to lack of development of the maxillae. Any [intranasal] bony [obstructions] are removed [surgically] until ample breathing space is established. If nasal obstruction is due simply to lack of size of the nasal fossae and there are erupted [permanent] molar teeth [on each side], then the treatment consists of placing a jackscrew across the mouth from one upper molar to the other. As the screw is spread, the intermaxillary suture opens, the maxillae separate, and the nasal obstruction is relieved."[10]

—VILRAY PAPIN BLAIR, AM, MD, FACS (1914)

Frequent Causes of Chronic Obstructive Nasal Breathing

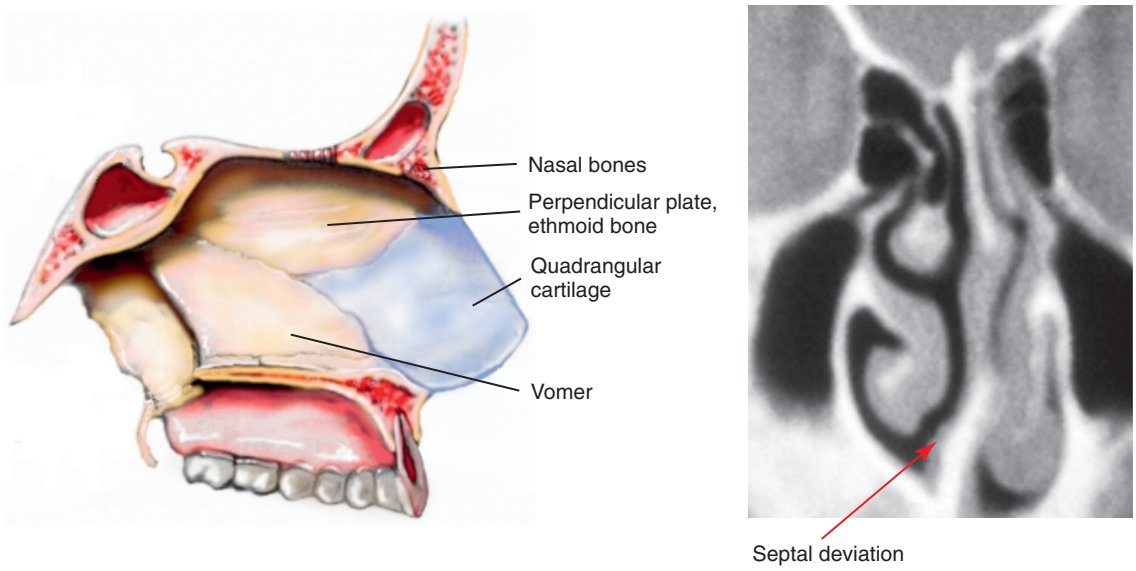
Nasal septoplasty is a generic descriptor of the surgical correction of *septal thickenings and deflections* frequently carried out to improve nasal airflow and sinus drainage (Figs. 10-1 and 10-2).[3,7,20,30,52,53,57,75,80,88,89,91,93,94] *Enlarged inferior turbinates* also commonly obstruct nasal breathing and interfere



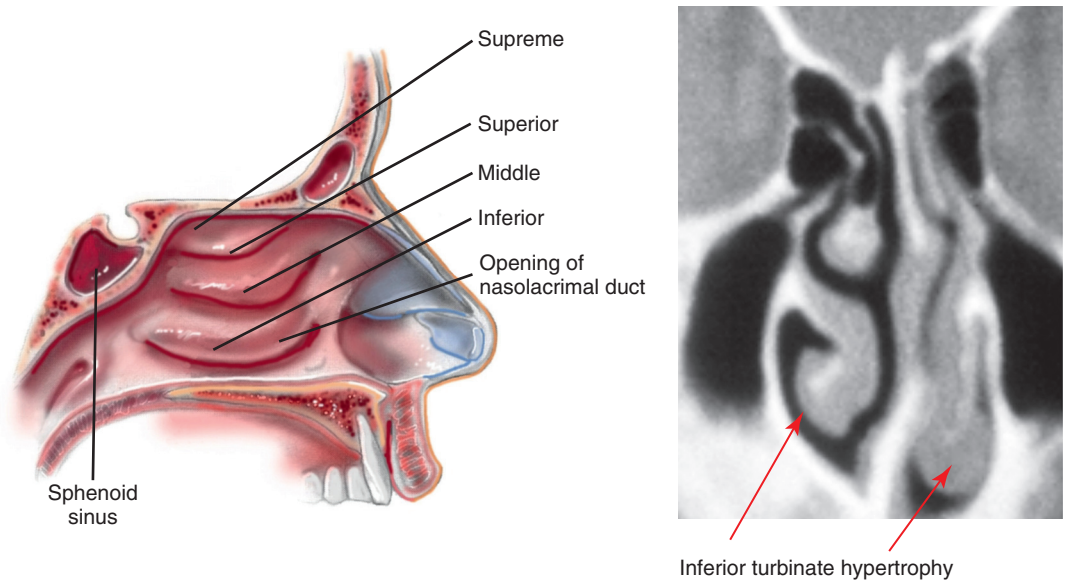
• **Figure 10-1 A,** Three-dimensional maxillofacial and coronal section computed tomography scan views of a child with mandibular deficiency during late mixed dentition. **B,** Three-dimensional maxillofacial and coronal section computed tomography scan views of a teenager with maxillary deficiency and relative mandibular excess. For both patients, the scans indicate a deviated nasal septum in combination with enlarged inferior turbinates, with an expected increase in nasal airway resistance and diminished breathing space.

with sinus drainage (Fig. 10-3). Hypertrophic inferior turbinates that are only minimally responsive to medical treatment are best managed by partial (surgical) reduction.[8,23,77] A *tight nasal inlet* or constricted pyriform apertures occur in conjunction with a narrow maxillary arch (Fig. 10-4).[58,86,90,95] An *elevated floor of the nose* is a frequent finding in the presence of anterior vertical maxillary excess (i.e., long face growth pattern) (Fig. 10-5).[63,81,112] A *scarred nasal vestibule* seen with a repaired cleft nasal malformation may be another cause of persistent nasal obstruction (Fig. 10-6). Nasal septal deformities, inferior turbinate enlargement, a tight nasal inlet, and an elevated nasal floor all commonly coexist with maxillary deformities (Figs. 10-7 and 10-8).[1,12-14] Favorable access to the nasal septum, the inferior turbinates, the pyriform apertures, and the nasal floor to correct these airway obstructions and deformities is possible through the Le Fort I down-fracture osteotomy that is commonly used in orthognathic surgery (Figs. 10-9, 10-10, and 10-11).[82-85]

Maxillofacial literature has traditionally addressed aspects of nasal obstruction, nasal anatomy, and sinus drainage in the patient who is undergoing orthognathic surgery, but often only in subjective and limited ways.[8,18,24,27,32,33,36,38,55,56,71,73,74,76,103,104,107] Authors traditionally discuss the effects of Le Fort I osteotomies on nasal morphology (i.e., aesthetics) and occasionally recommend adjunctive soft-tissue techniques in the hopes of limiting suboptimal aesthetics (e.g., the alar cinch stitch).[37] Little consideration is given to either baseline upper airway findings or how the maxillary surgery may affect long-term nasal breathing. Warren and colleagues discusses issues of nasal airway changes after Le Fort I osteotomy with maxillary



• **Figure 10-2** Sagittal midfacial illustration and coronal section computed tomography scan view of the skeletal (vomer and perpendicular plate of ethmoid) and cartilage (quadrangular) components of the septum of the nose. The computed tomography scan view indicates deviation and buckling of the septum, which obstructs the left side of the nose. *Modified from an original illustration by Bill Winn.*



• **Figure 10-3** Sagittal midfacial illustration and coronal section computed tomography scan showing an axial view of the turbinates. The CT scan view indicates asymmetrical inferior turbinate hypertrophy. *Modified from an original illustration by Bill Winn.*

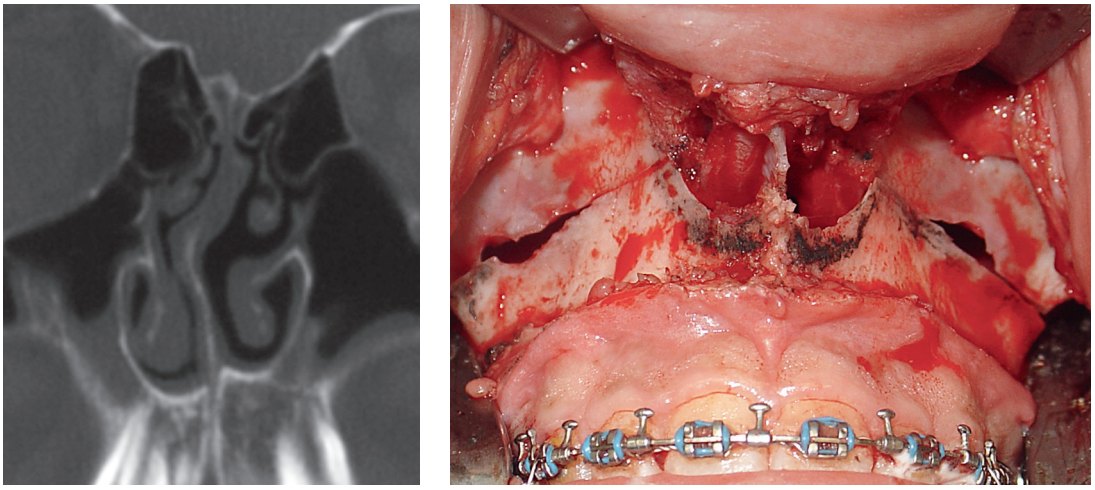
impaction, advancement, and transverse widening.[108] They used a pressure flow meter to assess nasal airway breathing before and after maxillary osteotomies.[21,29] Turvey and colleagues recognized preexisting nasal obstruction in many of their orthognathic patients.[103] They suggested methods of avoiding adverse effects on nasal airway resistance that may result from Le Fort I maxillary osteotomy procedures.[104] Moses and coworkers reported about a series of patients who were treated for nasal obstruction and sinus drainage difficulties after Le Fort I maxillary procedures were carried out for long face growth patterns (e.g., vertical maxillary excess with anterior open bite).[71] They concluded that Le Fort I impaction frequently aggravates preexisting nasal airway obstruction and sinus disease. Williams and colleagues examined the nasal airway function (breathing) in a consecutive series of subjects (n = 50) before and 5 months after undergoing (1) Le Fort I osteotomy with advancement and vertical lengthening (2) maxillary segmentation with transverse expansion (14 of 50 subjects, 28%); and (3) recontouring of the nasal floor and pyriform rims with a rotary drill. Despite these favorable nasal airway surgical maneuvers, when ignoring potential pathology of the septum (e.g., buckling and deviation) and the inferior

Text continued on p. 295

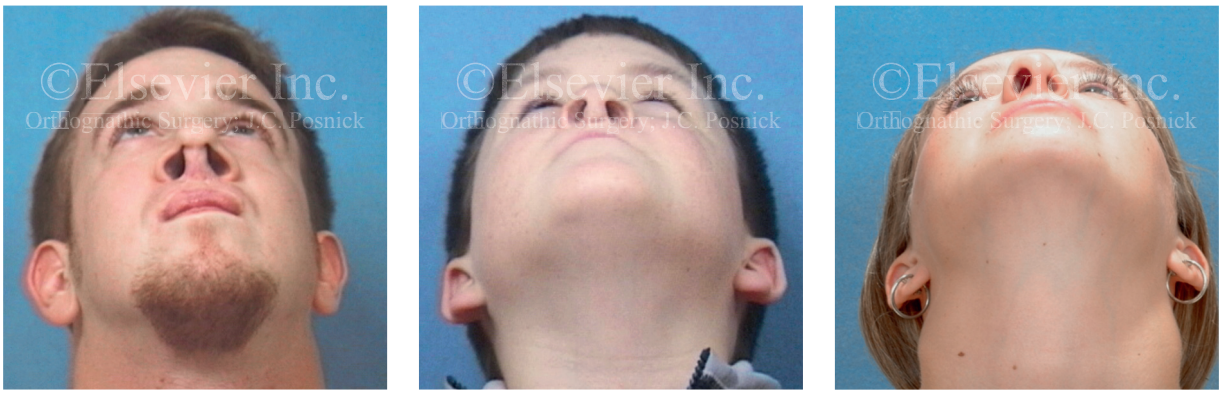
©Elsevier Inc.
Orthognathic Surgery: J.C. Posnick

• **Figure 10-4** A woman in her early 20s shown before and after orthognathic surgery for a long face growth pattern (see Fig. 21-5). Reconstruction also included segmental maxillary osteotomies with arch expansion. This approach also widens the nasal cavity to decrease intranasal airway resistance and improve breathing.

©Elsevier Inc.
Orthognathic Surgery: J.C. Posnick

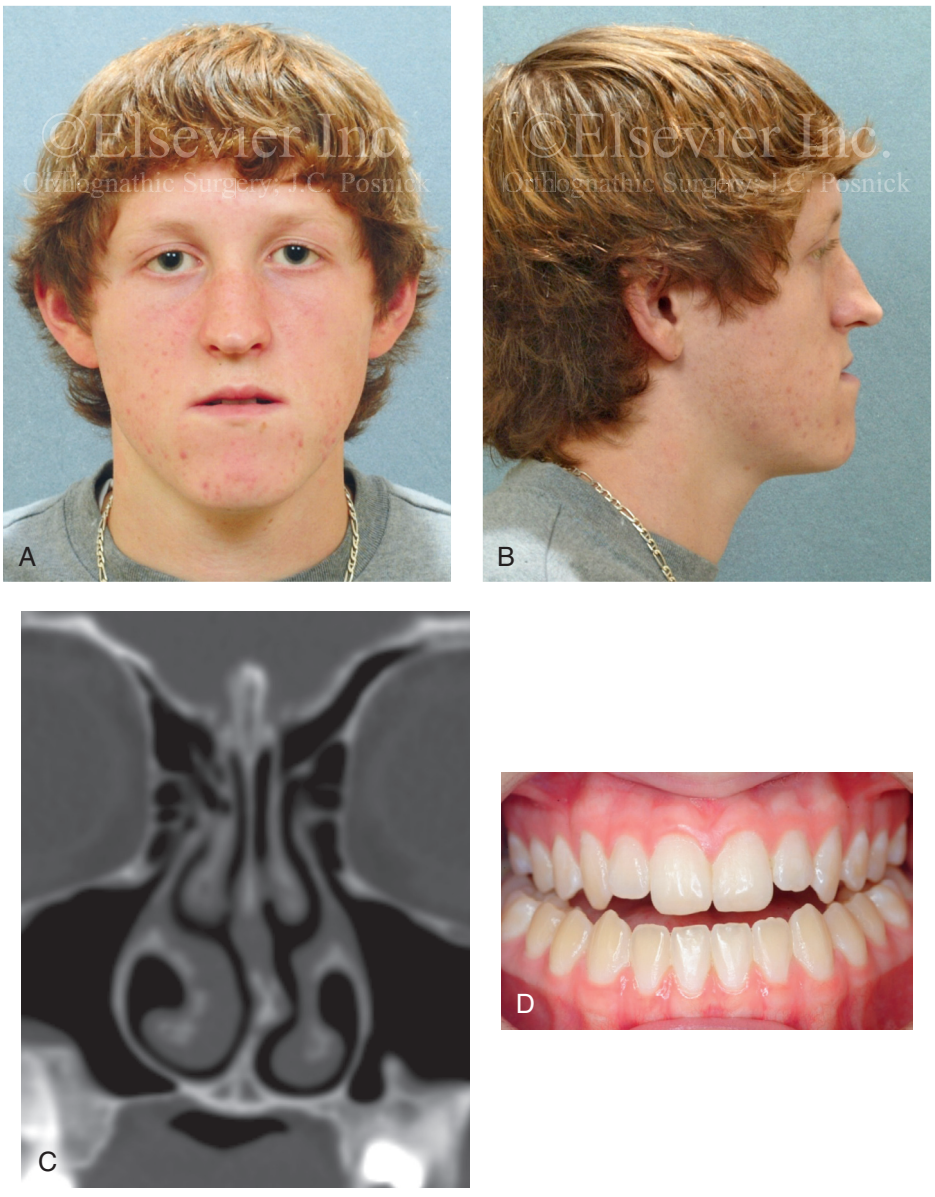


• **Figure 10-5** A typical teenager with a long face growth pattern demonstrates physical findings that are consistent with a lifelong history of obstructed nasal breathing (see Fig. 7-2). These findings include septal deviations, inferior turbinate hypertrophy, a narrow (tight) nasal aperture, and an elevated nasal floor.

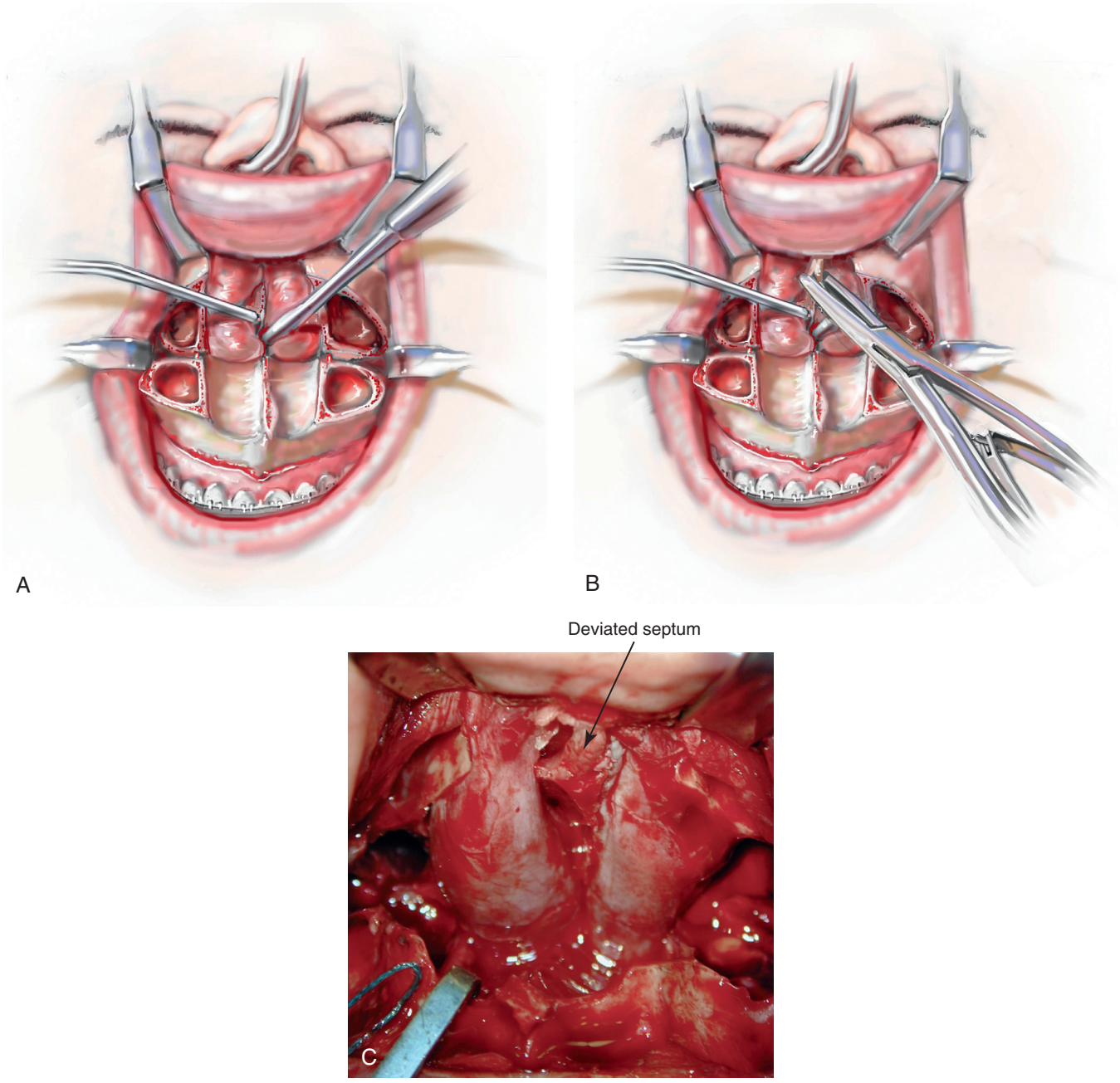

©Elsevier Inc.
Orthognathic Surgery: J.C. Posnick

• **Figure 10-6** Individuals born with cleft lips and palates who have undergone primary repair and secondary reconstruction may present with constrictions of both the internal and external nasal valves. This may be worse as a result of the surgical maneuvers that were previously carried out. Two children with bilateral cleft lip and palate and one with unilateral cleft lip and palate are shown to demonstrate this point. Nasal soft-tissue procedures—including "columella lengthening" in the cases of the bilateral cleft lips and palates and the overzealous resectioning of nasal soft tissues in the case of the unilateral cleft lip and palate—were performed. In all three cases, the nasal valves are constricted (i.e., there is airway obstruction), and the aesthetics are suboptimal.

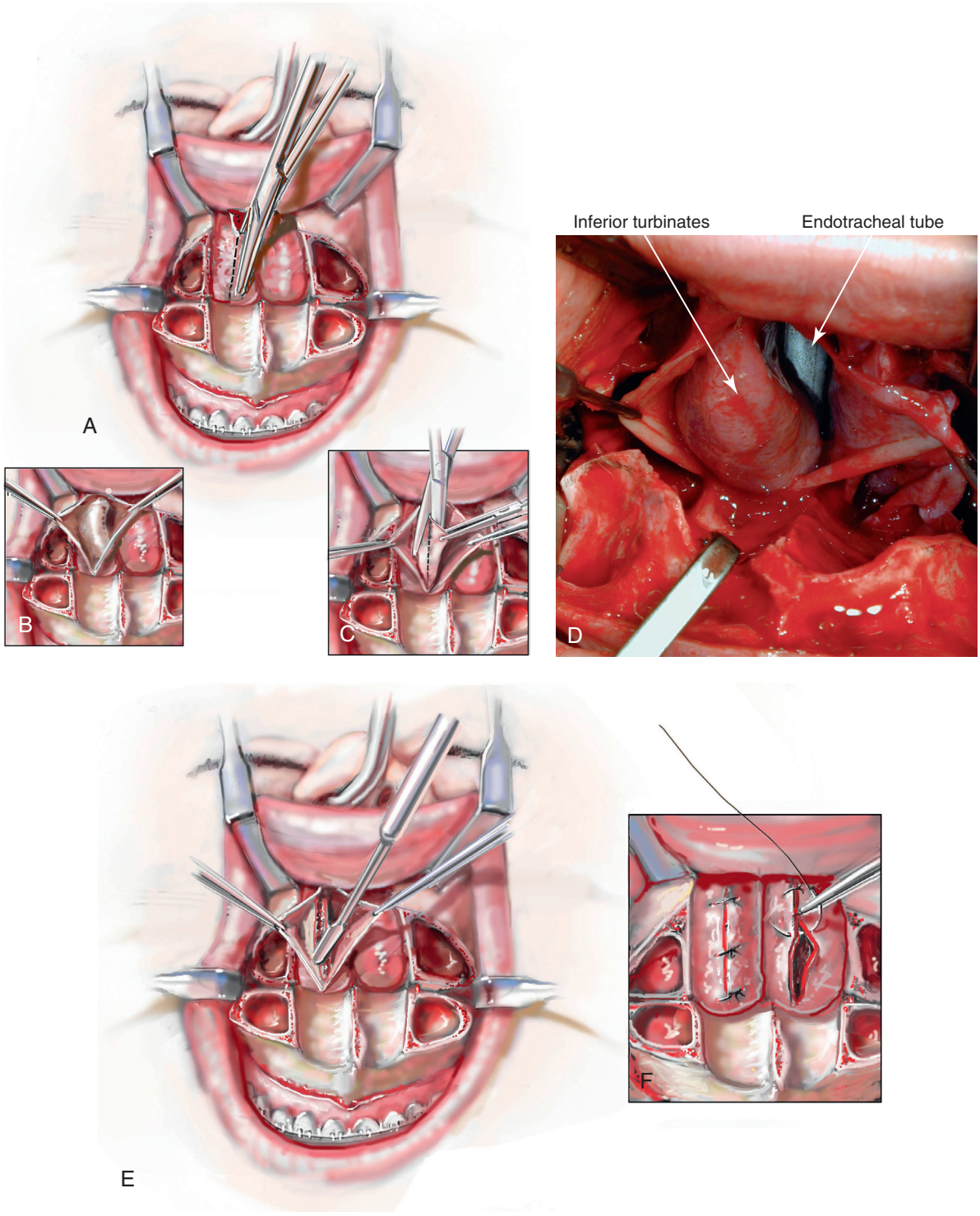
• **Figure 10-7** **A** and **B,** A teenage girl with unilateral cleft lip and palate shows multiple causes of nasal obstruction. These include—as shown in **C** and **D**—septal deviations, inferior turbinate hypertrophy, nasal aperture narrowing, and nasal floor deformities, including a residual unrepaired alveolar cleft.



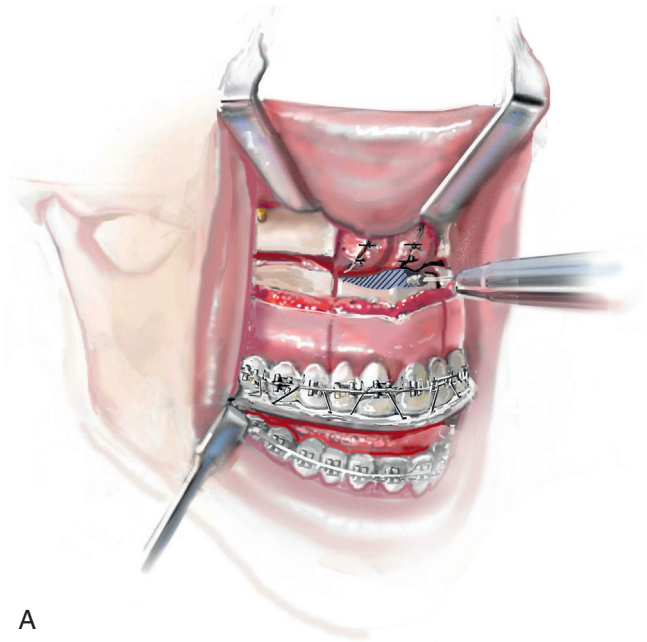
• **Figure 10-8 A** and **B,** A teenager with a maxillary deficiency and a relative mandibular excess growth pattern. **C** and **D,** The physical findings of septal deviations, inferior turbinate hypertrophy, and a narrow (tight) nasal aperture are consistent with the patient's history of nasal airway obstruction.



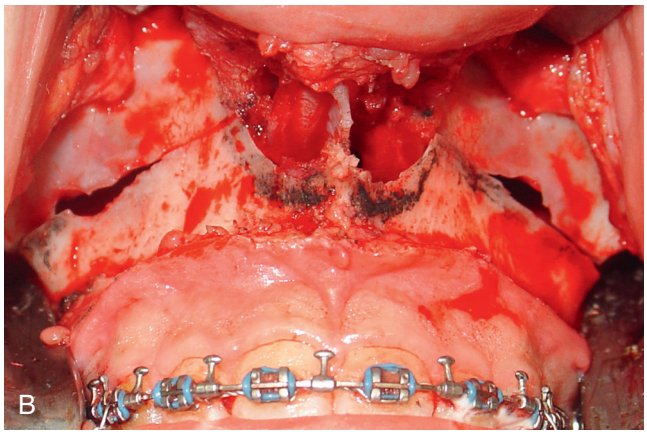
• **Figure 10-9** Illustrations and intraoperative view of a deformed nasal septum seen through a Le Fort I down-fracture. This is a demonstration of the technique used to carry out the submucous resection of septal bone and cartilage. When indicated on the basis of the patient's history and clinical examination, attention is turned to the removal of the deviated portions of the septum of the nose. **A,** With an elevator, the submucosal dissection of the cartilaginous (quadrangular cartilage) and bony (vomer and perpendicular plate of ethmoid) septum is carried out. **B,** The deviated and thickened aspects of the cartilaginous and bony septum are resected with a rongeur. The more anterior components of the cartilaginous septum that provide needed support to the nasal dorsum and tip are not disturbed. This includes at least 1 cm of the dorsal and caudal cartilage. **C,** An intraoperative view through a Le Fort down-fracture shows a deviated septum.



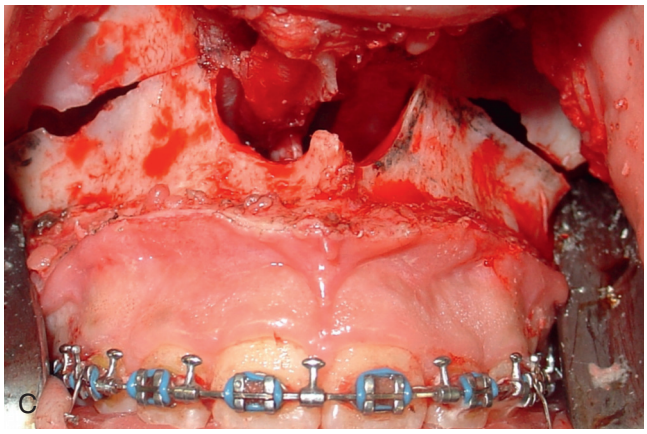
• **Figure 10-10** Illustrations and intraoperative view of inferior turbinates seen through a Le Fort I down-fracture. This is a demonstration of the technique used to carry out a partial turbinate reduction. **A,** An incision parallel and just below each inferior turbinates is completed through the nasal mucosa. **B,** The elevated nasal mucosa flaps expose the enlarged inferior turbinate. **C,** The resection of the inferior aspect of the hypertrophic inferior turbinate is completed with the use of a straight (Mayo) scissors. **D,** An intraoperative view of the elevated nasal flaps. The endotracheal tube is also visualized. **E,** After the partial resection of each inferior turbinate, the raw surfaces are cauterized. **F,** The nasal mucosa flaps are then closed with interrupted resorbable sutures.



A



B



C

• **Figure 10-11** Illustration and intraoperative views of the nasal rims and floor seen through a Le Fort I down-fracture to demonstrate the techniques of rim, nasal floor, and anterior nasal spine recontouring with the use of a bur on a rotary drill. **A,** Illustration of recontouring with rotary drill. **B** and **C,** Intraoperative views before and after recontouring, respectively.

turbinates (e.g., hypertrophy/enlargement), a full 20% of their study subjects experienced a worsening of their nasal airway function after surgery.[113a]

Unfortunately, in the individual with a dentofacial deformity, a methodic approach to the evaluation and management of chronic nasal airway obstruction at the time of orthognathic surgery is often not undertaken. We suggest that any upper airway obstructions be assessed by the orthognathic surgeon with the same vigor as the effects of the presenting jaw deformities on malocclusion are evaluated (Figs. 10-12 and 10-13).[51,83-85,87]

The Effects of Respiratory Pattern and Jaw Posture on Maxillofacial Growth

The link between an open mouth or oral breathing pattern and maxillofacial growth has been documented in the literature.[4,5,11,17,24,25,28,31,41,43,45-48,59-66,96,97,105,106,109,110] There has been both animal experimental and human clinical research to support a link between oral respiration, open mouth posture, and the development of maxillomandibular disharmonies. With the use of rhesus monkeys as an experimental model, Harvold and colleagues completely blocked the nares of newborn monkeys.[45-48] The researchers observed consistent facial morphologic changes that gradually occurred with growth in the presence of a constant open mouth and tongue protrusive mandibular posture. The research of this group documented that a constantly open mouth (i.e., clockwise rotation of the mandible) in a growing rhesus monkey was followed by the vertical lengthening of the maxilla. The mandibular inferior border then gradually steepens, with a resulting obtuse gonial angle. Through years of primate laboratory research, Harvold and coworkers confirmed that there are significant correlations between alterations in respiratory patterns (i.e., forced mouth breathing), mandibular (open mouth) posture, tongue protrusion, and characteristic maxillomandibular dysmorphic growth (i.e., long face growth pattern).[45-48]

In human children who have significant nasal obstruction and a resulting open mouth posture, clinical observational studies document a similar downward and backward rotation of the mandible with hypererruption of the maxillary dentition.[59-65] Over several years, the affected children develop what we now recognize as a *long face growth pattern.* This is documented as a vertically long and horizontally retrusive maxillomandibular complex. The term *adenoid facies* was coined to describe the child who presents with a long, narrow, flat face; protruding teeth; and lips that are widely separated at rest. These features are often accompanied by an anterior open-bite malocclusion and lip strain when attempting mouth closure. To better understand the concept, Linder-Aronson and Woodside completed a study in which they analyzed the faces of children with chronic nasal obstruction who were scheduled to undergo tonsillectomies and adenoidectomies at a pediatric hospital in Stockholm, Sweden.[63] They

documented a tendency toward a long lower anterior facial height in this group of children. Interestingly, they found that, after the adenoids were removed and with enough time, there was at least some improvement in the children's facial morphology. Unfortunately, the structure of these long faces did not return to normal. With or without adenoidectomy being carried out during childhood, many of these children reach adolescence with persistent nasal airway obstruction in combination with significant jaw dysmorphology and malocclusion (i.e., the long face growth pattern). It is our recommendation that, by adolescence, all aspects (i.e., airway obstruction, skeletal dysmorphology, and malocclusion) be simultaneously addressed to improve function in the head and neck (e.g., breathing, swallowing, speech, chewing, lip closure) and to enhance facial aesthetics.[83-85]

Simultaneous Management of Chronic Obstructed Nasal Breathing and the Presenting Dentofacial Deformity

Posnick and colleagues conducted a clinical study with the use of the *Nasal Obstruction Symptom Evaluation* (NOSE) survey questionnaire to prospectively assess a consecutive series of patients (N = 43) who reported both chronic nasal obstruction and the presence of a significant dentofacial deformity with malocclusion that involved at least the maxilla.[83] The NOSE questionnaire is considered to be a useful marker of symptomatic nasal airway obstruction.[42,98-101] Each study subject was documented to have septal deviation and inferior turbinate enlargement that were consistent with airway symptoms. Any presenting physical findings of a tight nasal inlet or an elevated nasal floor were not specifically studied but were clinically addressed in each patient, when indicated. Each study patient simultaneously underwent septoplasty, the reduction of the inferior turbinates, and the surgical correction of the presenting jaw deformity that included, at minimum, a Le Fort I osteotomy. Septoplasty and inferior turbinate reduction were performed at the time of Le Fort I osteotomy through exposure provided by the down-fracture. The consecutive series of patients (N = 43) who met the inclusion criteria were given the NOSE survey questionnaire before they underwent surgery. The NOSE questionnaire was completed in each patient 3 months after surgery and again at least 6 months after surgery.

The study group included 16 males and 27 females with a mean age of 25 years (range, 15 to 55 years). All patients were treated with a comprehensive orthodontic and orthognathic surgical approach that included a Le Fort I osteotomy as part of the surgical correction. The most frequent pattern of jaw deformity identified in the study group was a long face growth pattern (i.e., maxillary vertical excess with Class II malocclusion), which was seen in 17 out of 43 patients (40%). Simultaneous maxillofacial procedures completed in addition to the Le Fort I osteotomy, septoplasty, and inferior turbinate reduction varied. Twenty-nine of the 43 patients (67%) underwent segmentation of the maxilla to widen the arch width or to correct the curve of Spee. Forty-two of the patients (98%) underwent simultaneous sagittal split ramus osteotomies, 16 (37%) underwent the simultaneous removal of the third molars, and 7 (16%) also underwent suction-assisted lipectomy of the neck. Thirty-seven patients (86%) underwent oblique osteotomy of the chin with reshaping (i.e., osseous genioplasty).

The mean weighted NOSE score before surgery was 63.7. At the 3-month postoperative interval, the mean NOSE score had dropped to 6.9. On the basis of the nonparametric Wilcoxon signed-rank test, this is a significant finding ($P < .001$). The mean NOSE score at 6 months after surgery dropped even further to 6.3. This improvement in nasal breathing remained both clinically and statistically significant as compared with what was found before surgery. The findings confirmed an improved nasal airway by 3 months after surgery among patients who had undergone combined septoplasty, inferior turbinate reduction, and Le Fort I osteotomy; these improvements continued and were maintained at 6 months. All study patients experienced improvements in nasal breathing as a result of the combination of procedures that were carried out.

The incidence of complications that resulted from the intranasal procedures performed simultaneously with the Le Fort I osteotomy as reported by Posnick and colleagues was minimal and not greater than those reported in the literature for isolated procedures. Posnick and colleagues also reported about the incidence of Le Fort I osteotomy–specific complications in the same cohort. The complications were few and not greater than those reported when a Le Fort I osteotomy was carried out as an isolated procedure.

Approaching the Nasal Airway Through Le Fort I Down-Fracture (Video 1 and 2)

A Le Fort I osteotomy provides excellent access to the nasal septum, the inferior turbinates, the pyriform rims, the nasal floor, the anterior nasal spine, the maxillary sinus cavities, and the ostium, thereby allowing for the correction of any presenting nasal airway obstruction or maxillary sinus pathology at the time of down-fracture (see Figs. 10-9, 10-10, and 10-11).[82,83] Interestingly, most otolaryngologists/head and neck surgeons are not used to viewing these structures from this perspective (i.e., through the down-fracture), whereas orthognathic surgeons are. There are several technical details to consider when completing septoplasty or inferior turbinate reduction through the exposure provided by the Le Fort I osteotomy. As always, care is taken to maintain the integrity of the nasal mucosa when down-fracturing the maxilla. Nevertheless, tears in the mucosa are common. After the down-fracture and disimpaction of the maxilla, an elevator is used to achieve the submucosal exposure of all

buckled and deviated portions of the cartilaginous and bony septum. With the use of a rongeur, the buckled or deviated portions of the cartilaginous and bony septum are removed (i.e., submucosal resection); however, adequate dorsal and caudal components of the cartilaginous septum are preserved for structural support. If a cosmetic rhinoplasty is a future consideration, then the preservation of as much useful septal cartilage as possible for later tip grafting is important. Next, an incision that is parallel and just below each inferior turbinate is completed through the nasal mucosa. If the nasal mucosa was torn during the down-fracture, then the exposure provided is used and extended as needed for the additional visualization of each inferior turbinate. With the use of a scissors (e.g., Mayo scissors), the inferior aspect of each enlarged inferior turbinate can be excised via partial resection. Other surgical techniques to reduce enlarged inferior turbinates are described, such as intramural ablation. The direct visualization of the more cephalic positioned nasotracheal tube within the surgical field limits the chance of injury during the turbinate reduction procedure. There is sufficient room for the surgeon to work around the endotracheal tube. With no oxygen leaks in the ventilation system, the cauterization of the exposed raw surface of each inferior turbinate is completed. Suture repair of the nasal floor mucosa is then accomplished. No nasal packs or splints are required for postoperative management. With this approach, the author has not experienced significant intraoperative bleeding or postoperative airway compromise.

Management of Residual Postoperative Nasal Obstruction

When the baseline presurgical nasal airway obstruction is clarified and then managed simultaneously through the Le Fort I down-fracture, residual nasal obstruction is uncommon. When a degree of postsurgical nasal obstruction persists, it is generally the result of an incomplete presurgical diagnosis or the inadequate operative correction of all of the aspects that are causing the blockage (e.g., constricted aperture, elevated floor, enlarged inferior turbinates, septal deviations). After septoplasty and inferior turbinate procedures and after routine postoperative intranasal swelling subsides, continued nasal obstruction may also occur as a result of scar formation.[67,79,83] Should a degree of nasal obstruction persist, it is important to confirm that residual septal deviation, turbinate hypertrophy, and nasal valve (internal and external) stenosis are not factors. If these causes are ruled out and if postsurgical obstruction remains, scar bands of varied thickness are likely to be the cause. If so, the intranasal synechiae can extend from the operated septum to the inferior turbinates or to the healing areas along the nasal mucosal floor. The resulting scar bands are usually small to moderate in size, and they can often be corrected in the surgeon's office with the use of local spray anesthesia and scissors transaction under endoscopic visualization. Occasionally, dense, thick synechiae occur; these may contract a straightened septum into a lateralized position. For more extensive scar bands and when further septoplasty or turbinate reduction is required, management in the operating room via an intranasal approach (with the placement of a separating splint between the septum and the lateral nose to prevent the adjacent fresh mucosal edges from re-creating the scar contracture) is preferred.

Effects of Maxillary Arch Expansion and Nasal Floor Recontouring on the Airway

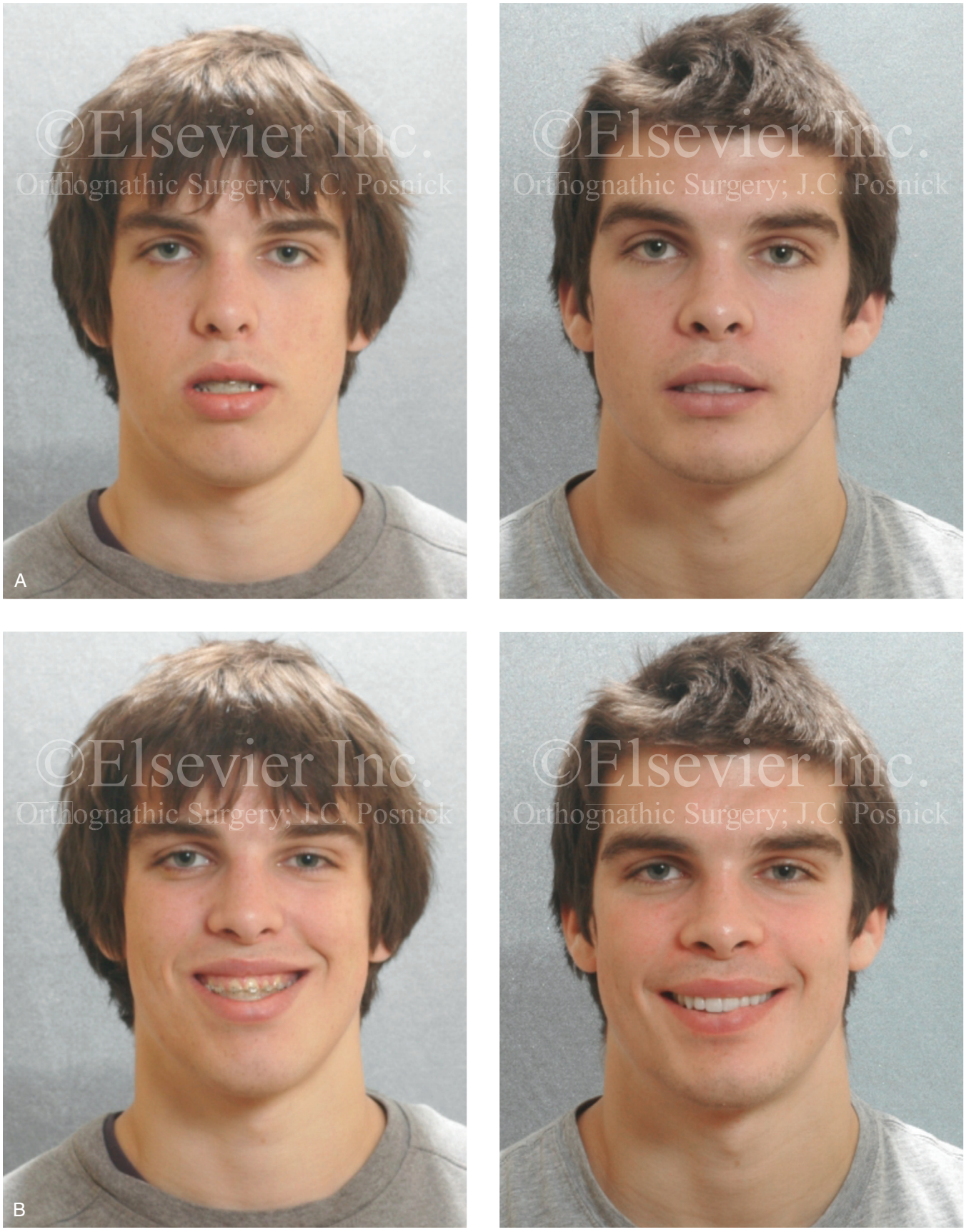
Nasal Floor Recontouring: Effects on the Airway (Video 3 and 4)

The presence of excess vertical alveolar height of the anterior maxilla above the roots of the teeth results in a *high nasal floor* that constricts the nasal passageways, thereby causing increased nasal airway resistance.[24,113] This is a frequent occurrence in patients with a long face growth pattern (see Chapter 21). The need for vertical intrusion (i.e., superior repositioning) of the anterior maxilla to reduce the gummy smile, to improve lip closure, and to reduce mentalis strain is generally required as part of the long face correction (see Figs. 10-5, 10-11, and 10-13). As a result of maxillary intrusion, any baseline nasal obstruction caused by a high nasal floor will worsen unless it is simultaneously addressed. The *need to recontour the nasal floor, the pyriform rims, and the anterior nasal spine region* is also frequent in patients with cleft lips and palates (e.g., unilateral or bilateral cleft lip and palate) that present with a maxillary deformity. *Recontouring to lower and level the nasal floor, to widen the pyriform rims, and to reduce and reshape the anterior nasal spine* can simultaneously be performed through the Le Fort I down-fracture. This will both enhance upper lip and nose aesthetics and reduce nasal airway resistance. The recontouring procedure is carried out with a watermelon-shaped bur on a low-speed rotary drill and a rongeur under direct visualization either before or after the maxilla is secured (i.e., fixed with plates and screws) in its new location (see Chapter 15 and Fig. 10-11).

Maxillary Arch Expansion: Effects on the Airway

Studies predominantly show that *maxillary arch expansion* will improve a *tight nasal inlet* and thereby decrease nasal airway resistance.[15,22,68] The documented improved nasal breathing is believed to result from a widened airway space, primarily at the level of the *internal nasal valve* brought about by maxillary arch expansion.* Seeberger and colleagues[92] and Wreidt and colleagues[114] examined patients via acoustic rhinometry before and after surgically assisted rapid maxillary expansion and recorded profound increases in total nasal volumes. They also reported that their patients experience better nasal airflow with a distinct subjective

*References 6, 9, 16, 22, 26, 34, 39, 40, 44, 49, 62, 68, 69, 102, 108, 111



• **Figure 10-12** A 20-year-old man with a combined long face growth pattern (environmental) and a right side hemimandibular elongation (hereditary). There is an asymmetrical Class III negative overjet malocclusion. The patient had a lifelong history of obstructed nasal breathing with documented septal deviation, hypertrophic inferior turbinates, and an elevated nasal floor. He was referred to this surgeon for evaluation, and he agreed to a comprehensive orthodontic and orthognathic approach. With the relief of dental compensation, the patient's surgery included a maxillary Le Fort I osteotomy (horizontal advancement and vertical shortening); bilateral sagittal split ramus osteotomies (mandibular straightening and asymmetrical correction); osseous genioplasty (vertical shortening); and septoplasty, inferior turbinate reduction, and nasal floor recontouring. **A,** Frontal views in repose before and after reconstruction. **B,** Frontal views with smile before and after reconstruction.

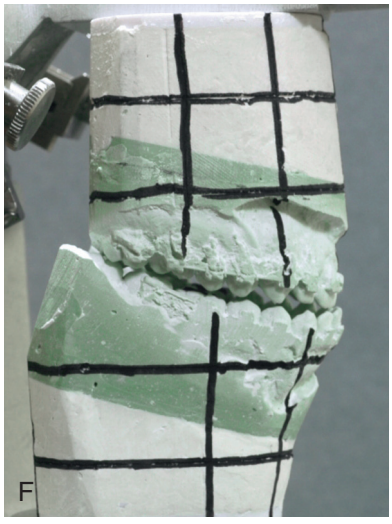
• **Figure 10-12, cont'd C,** Oblique facial views before and after reconstruction. **D,** Profile views before and after reconstruction.

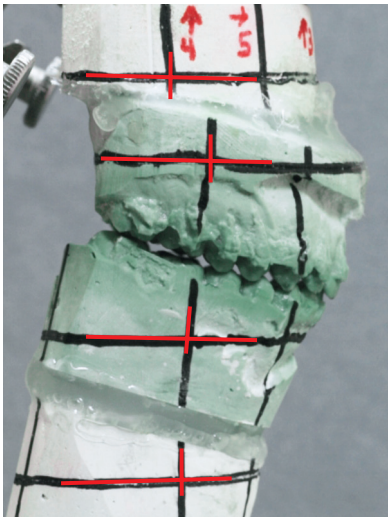
Continued

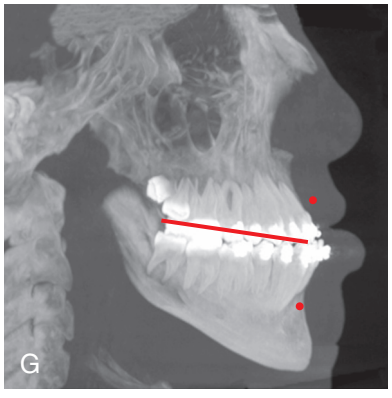
Prior to treatment

Pre surgery

After treatment







• **Figure 10-12, cont'd E,** Occlusal views before retreatment, after orthodontic decompensation, and after treatment. **F,** Articulated dental casts that indicate analytic model planning. **G,** Lateral cephalometric radiographs before and after reconstruction.

improvement in nasal breathing. Maxillary widening may be accomplished with a number of techniques (e.g., rapid palatal expansion, surgically assisted rapid palatal expansion, segmental Le Fort I osteotomy). The method selected is dependent on the age of the patient and the combination of airway, occlusal, and facial aesthetic needs. The frequent need for arch expansion in the individual with a long face growth pattern is well recognized (see Fig. 10-4), and it is also common with other dentofacial deformities (e.g., maxillary deficiency with relative mandibular excess, primary mandibular deficiency, bimaxillary dental protrusion, and cleft maxillary deformities). Expiratory pressure, expiratory flow values, and inspiratory flow values have all been documented to improve immediately after lateral nasal wall widening. These benefits should remain long term unless lateral wall relapse occurs.

Comprehensive Approach to Chronic Nasal Airway Obstruction

The fact that Posnick and colleagues showed a more significant symptomatic improvement in nasal breathing in their study patients as compared with that found by Stewart and colleagues when septoplasty only was carried out is not surprising.[100] This likely resulted from the strict patient selection process and the other procedures that were simultaneously carried out (e.g., inferior turbinate reduction, transverse widening of the maxilla, horizontal maxillary advancement, lowering the nasal floor by recontouring).

An interesting observation emerged from the COG-ENT chronic rhinosinusitis study that was completed in 2001.[98-101] The COG-ENT researchers found that patients who were undergoing functional endoscopic sinus surgery in combination with septoplasty and inferior turbinate reduction showed even greater clinical improvement in nasal breathing as compared with those undergoing functional endoscopic sinus surgery alone. This confirms the added value to the airway (i.e., the improved breathing) related to carefully selected diagnosis-driven procedures being simultaneously carried out.[19,35,50,54,70,72,78]

Conclusions

Orthognathic surgeons have the opportunity to improve disease-specific quality-of-life issues for their patients at the time of Le Fort I osteotomy by performing diagnosis-driven intranasal procedures in those with symptomatic chronic nasal airway obstruction and sinus drainage difficulties. The improvement of nasal airflow often enhances the patient's sense of smell, sinus drainage, sleep comfort, daytime alertness, dental health, and ability to perform exercise. Ignoring any baseline nasal floor and pyriform rim skeletal deformities at the time of orthognathic surgery is also likely to result in suboptimal facial aesthetics.

Case Presentation

A classic example of the combined occurrence of chronic nasal airway obstruction and dentofacial deformity is seen in the description of a 15-year-old high school student who arrived for evaluation accompanied by her parents (see Fig. 10-13).

Since early childhood, the patient had a forced mouth-breathing pattern, and snoring at night was documented. With exercise, she had difficulty getting enough air through the nose. She was seen by an otolaryngologist/head and neck surgeon and found to have a deviated nasal septum, a hypertrophic inferior turbinate, and a tight nasal aperture. However, no treatment was carried out earlier during her life. As a teenager, she presented with a long face growth pattern, an anterior open bite, and a Class II excess overjet

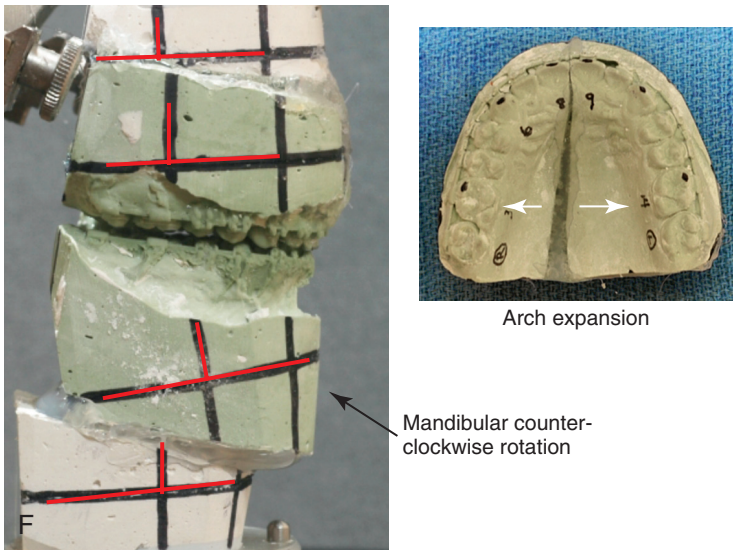
• **Figure 10-13** A teenage girl with a long face growth pattern is treated with a comprehensive orthodontic and surgical approach. After orthodontic (dental) decompensation, her surgical procedures included a maxillary Le Fort I osteotomy in segments (vertical intrusion, horizontal advancement, and arch expansion) bilateral sagittal split osteotomies of the mandible (horizontal advancement and counterclockwise rotation); oblique osteotomy of the chin (vertical shortening and horizontal advancement); and septoplasty, inferior turbinate reduction, and recontouring of the nasal floor. **A,** Frontal views in repose before and after treatment. **B,** Frontal views with smile before and after treatment.

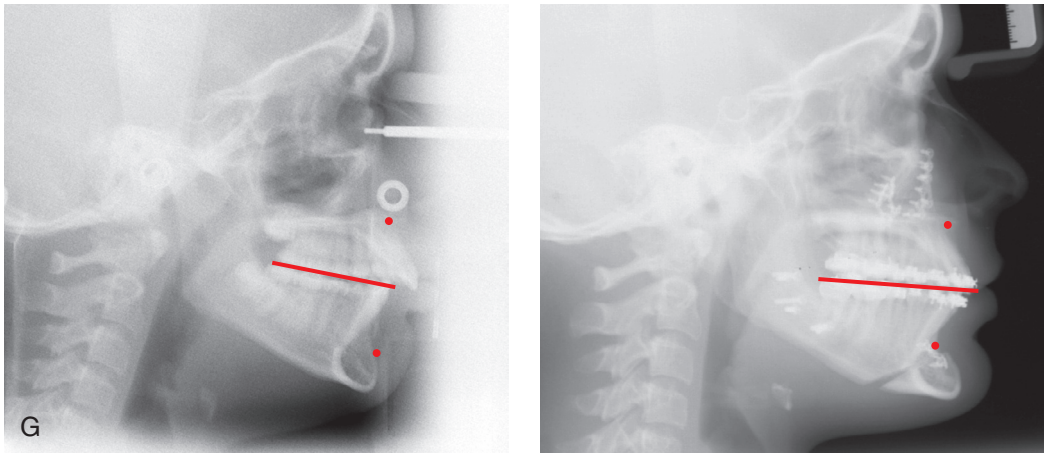
Pre surgery

After treatment

• **Figure 10-13, cont'd C,** Oblique facial views before and after treatment. **D,** Profile views before and after treatment. **E,** Occlusal views with orthodontics in progress and then after surgery.

Continued





• **Figure 10-13, cont'd F,** Articulated dental casts that indicate analytic model planning. **G,** Lateral cephalometric views before and after treatment.

malocclusion. There were also concerns about a gummy smile, difficulty maintaining lip closure, speech articulation errors, and a weak profile.

Both orthodontic and surgical consultation recommended a comprehensive approach as the most biologic way to improve the upper airway obstruction; to improve the occlusion and the long-term dental health; to improve lip closure; and to enhance facial aesthetics. Orthodontic treatment was initiated to relieve dental compensation, and jaw and intranasal surgery followed.

Management of Dentofacial Deformity Required the Following:

- Maxillary Le Fort I osteotomy in three segments
- Bilateral sagittal split ramus osteotomies of the mandible
- Oblique osteotomy of the chin

Management of Chronic Nasal Airway Obstruction Required the Following:

- Submucous resection of deflected and buckled portions of the nasal septum
- Reduction of the hypertrophic inferior turbinates
- Maxillary transverse expansion (Le Fort I osteotomy in segments) with nasal aperture widening
- Recontouring (lowering) of the nasal floor and the anterior nasal spine

The patient is shown before and 1 year after surgery and the completion of orthodontic treatment.

References

1. Angell EC: Treatment of irregularities of the permanent or adult teeth. *Dent Cosmos* 540–544, 1860.
2. Angle EH: *Malocclusion of teeth*, ed 7, Philadelphia, 1907, SS White Dental Mfg Co.
3. Arunachalam PS, Kitcher E, Gray J, et al: Nasal septal surgery: evaluation of symptomatic and general health outcomes. *Clin Otolaryngol Allied Sci* 26:367–370, 2001.
4. Bacon WH, Turlet JC, Krieger J, Stierle JL: Craniofacial characteristics in patients with obstructive sleep apnea syndrome. *Cleft Palate J* 25:374–378, 1988.
5. Baik UB, Suzuki M, Ikeda K, et al: Relationship between cephalometric characteristics and obstructive sites in obstructive sleep apnea syndrome. *Angle Orthod* 72(2):124–134, 2002.
6. Basciftci FA, Mutlu N, Karaman AI, et al: Does the timing and method of rapid maxillary expansion have an effect on the changes in nasal dimensions? *Angle Orthod* 72(2):118–123, 2002.
7. Bateman ND, Woolford TJ: Informed consent for septal surgery: the evidence-base. *J Laryngol Otol* 117(3):186–189, 2003.
8. Bell WH, Sinn D: Turbinectomy to facilitate superior movement of the maxilla by Le Fort I osteotomy. *J Oral Surg* 37:129–130, 1979.
9. Bicakci AA, Agar U, Sokucu O, et al: Nasal airway changes due to rapid maxillary expansion timing. *Angle Orthod* 75:1–6, 2005.
10. Blair VP: *Surgery of the mouth and jaws*, St. Louis, 1914, Mosby.
11. Bresolin D, Shapiro PA, Shapiro GG, et al: Mouth breathing in allergic children: its relationship to dentofacial development. *Am J Orthod* 84:334, 1983.
12. Brown GVI: The application of orthodontia principles to the prevention of nasal disease. *Dental Cosmos* 45:765–775, 1903.
13. Brown GVI: The surgical and therapeutic aspect of maxillary readjustment with special reference to nasal stenosis, hare lip, cleft palate and speech. *Dent Cosmos* 51:7–17, 1909.
14. Brown GVI: *The surgery of oral and facial diseases and malformations*, ed 4, London, 1938, Kimpton.
15. Ceroni Compadretti G, Tasca I, Alessandri-Boneti G, et al: Acoustic rhinomanometric measurements in children undergoing rapid maxillary expansion. *Int J Pediatr Otorhinolaryngol* 70(1):27–34, 2006.
16. Cistulli PA, Palmisano RG, Poole MD: Treatment of obstructive sleep apnea syndrome by rapid maxillary expansion. *Sleep* 21(8):831–835, 1998.
17. D'Ascanio L, Lancione C, Pompa G, et al: Craniofacial growth in children with nasal septum deviation: a cephalometric comparative study. *Int J Pediatr Otorhinolaryngol* 74:1180, 2010.
18. De Mol van Otterloo JJ, Leezenberg JA, Tuinzing DB, van der Kwast WA: The influence of the Le Fort I osteotomy on nasal airway resistance. *Rhinology* 28(2):107–112, 1990.
19. De Vito A, Berrettini S, Carabelli A, et al: The importance of nasal resistance in obstructive sleep apnea syndrome: a study with positional rhinomanometry. *Sleep Breath* 5(1):3–11, 2001.
20. Dinis PB, Haider H: Septoplasty: long-term evaluation of results. *Am J Otolaryngol* 23:85–90, 2002.
21. Djupesland PG, Rotnes JS: Accuracy of acoustic rhinometry. *Rhinology* 39(1):23–27, 2001.
22. Doruk C, Sokucu O, Sezer H, Canbay EI: Evaluation of nasal airway resistance during rapid maxillary expansion using acoustic rhinometry. *Eur J Orthod* 26:397–401, 2004.
23. Egeli E, Demirci L, Yazycy B, Harputluoglu U: Evaluation of the inferior turbinate in patients with deviated nasal septum by using computed tomography. *Laryngoscope* 114(1):113–117, 2004
24. Ellis E: The nature of vertical maxillary deformities: implications for surgical intervention. *J Oral Maxillofac Surg* 43:756–762, 1985.
25. Enache AM, Nimigean VR, Mihaltan F, et al: Assessment of sagittal and vertical skeletal patterns in Romanian patients with obstructive sleep apnea. *Rom J Morphol Embryol* 51:505–508, 2010.
26. Enoki C, Valera FC, Lessa FC, et al: Effect of rapid maxillary expansion on the dimension of the nasal cavity and on nasal air resistance. *Int J Pediatr Otorhinolaryngol* 70(7):1225–1230, 2006.
27. Erbe M, Lehotay M, Gode U, et al: Nasal airway changes after Le Fort I–impaction and advancement: anatomical and functional findings. *Int J Oral Maxillofac Surg* 30(2):123–129, 2001.
28. Fields DH: Relationship between vertical dentofacial morphology and respiration in adolescents. *Am J Orthod* 99:147–154, 1991.
29. Foy R: Contribution rhinométrique à l'étude de la respiration nasale. Annals des maladies de l'oreille, du larynx, du nez et du pharynx. *T XXXVI* 130–149, 1910.
30. Freng A, Kvam E: Facial sagittal growth following partial basal resection of the nasal septum: a retrospective study in man. *Eur J Orthod* 1:89–96, 1979.
31. Freng A: Dentofacial development in the long-lasting nasal stenosis. *Scand J Dent Res* 89:260–267, 1979.
32. Gotzfried HF, Hellmut M: On the improvement of nasal breathing following mid-face osteotomies, and possible reasons for the phenomenon. *J Oral Maxillofac Surg* 12:29, 1984.
33. Gotzfried HF, Masing H: Improvement of nasal breathing in cleft patients following midface osteotomy. *Int J Oral Maxillofac Surg* 17:41–44, 1988.
34. Gray LP: Results of 310 cases of rapid maxillary expansion selected for medical reasons. *J Laryngol Otol* 89(6):601–614, 1975.
35. Gryczynska D, Powajbo K, Zakrzewska A: The influence of tonsillectomy on obstructive sleep apnea children with malocclusion. *Int J Pediatr Otorhinolaryngol* 32(Suppl):S225–S228, 1995.
36. Guenther TA, Sather AH, Kern EB: The effect of Le Fort I maxillary impaction on nasal airway resistance. *Am J Orthod* 85(4):308–315, 1984.
37. Guymon M, Crosby DR, Wolford LM: The alar base cinch suture to control nasal width in maxillary osteotomies. *Int J Adult Orthodon Orthognath Surg* 2:89–95, 1988.
38. Haarmann S, Budihardja AS, Wolff KD, Wargerin K: Changes in acoustic airway profiles and nasal airway resistance after Le Fort I osteotomy and functional rhinosurgery: a prospective study. *Int J Oral Maxillofac Surg* 38:321–325, 2009.
39. Haas J: The treatment of maxillary deficiency by opening the midpalatal suture. *Angle Orthod* 35(3):200–217, 1965.
40. Haas AJ: Palatal expansion: just the beginning of dento-facial orthopedics. *Am J Orthod* 57(3):219–225, 1970.
41. Handelman CS, Osborne G: Growth based on nasopharynx and adenoid development from one to eighteen years. *Angle Orthod* 46:243–258, 1976.
42. Hannley M, Stewart MG, Witsell D: A nose for evidence: report of the Nasal Obstruction and Septoplasty Effectiveness (NOSE) study. *AAO-HNS Bulletin* 9:42–43, 2003.
43. Hannuksela A: The effects of moderate and severe atopy on the facial skeleton. *Eur J Orthod* 3:187–193, 1981.
44. Hartgerink DV, Vig PS, Abbot DW: The effect of rapid maxillary expansion on nasal airway resistance. *Am J Orthod* 92:381–389, 1987.
45. Harvold EP: The role of function in the etiology and treatment of malocclusion. *Am J Orthod* 54:883–898, 1968.
46. Harvold EP, Chierici G, Varervik K: Experiments on the development of dental malocclusion. *Am J Orthod* 61:38–44, 1972.
47. Harvold EP, Vargervik K, Chiericu G: Primate experiments on oral sensation and dental malocclusion. *Am J Orthod* 63:494–508, 1973.
48. Harvold EP, Tomer BS, Chierici G, Vargervik K: Primate experiments in oral respiration. *Am J Orthod* 79:359–372, 1981.
49. Hershey HG, Stewart BL, Warren DW: Changes in nasal airway resistance associated with rapid maxillary expansion. *Am J Orthod* 69:274–284, 1976.

50. Holmberg H, Linder-Aronson S: Cephalometric radiographs as a means of evaluating the capacity of the nasal and nasopharyngeal airway. *Am J Orthod* 76:479–490, 1979.
51. Hooper RG: Forced inspiratory nasal flow-volume curves: a simple test of nasal airflow. *Mayo Clin Proc* 76(10):990–994, 2001.
52. Jessen M, Malm L: The importance of nasal airway resistance and nasal symptoms in the selection of patients for septoplasty. *Rhinology* 22:157–164, 1984.
53. Jensen M, Ivarsson A, Malm L: Nasal airway resistance and symptoms after functional septoplasty: comparison of findings at 9 months and 9 years. *Clin Otolaryngol* 14:231–234, 1989.
54. Kerr P, Millar T, Buckle P, Kryger M: The importance of nasal resistance in obstructive sleep apnea syndrome. *J Otolaryngol* 21(3):189–195, 1992.
55. Kirk WS, Bolz EA: Pre-orthognathic surgery evaluation of nasal obstruction. *Int J Adult Orthodon Orthognath Surg* 3:109–115, 1988.
56. Kunkel M, Hochban W: The influence of maxillary osteotomy on nasal airway patency and geometry. *Mund Kiefer Gesichtschir* 1:194–198, 1997.
57. Lee BJ, Chung YS, Jang YJ: Overcorrected septum as a complication of septoplasty. *Am J Rhinol* 18(6):393–396, 2004.
58. Lee KS, Yang CC, Huang JK, et al: Congenital pyriform aperture stenosis: surgery and evaluation with three-dimensional computed tomography. *Laryngoscope* 112(5):918–921, 2002.
59. Linder-Aronson S, Backstrom A: A comparison between mouth and nose breathers with respect to occlusion and facial dimension. *Odontol Rev* 11:343–376, 1960.
60. Linder-Aronson S, Aschan G: Nasal resistance to breathing and palatal height before and after expansion of the median palatine suture. *Odontol Rev* 14:254–270, 1963.
61. Linder-Aronson S: Adenoids: their effect on mode of breathing and nasal airflow and their relationship to characteristics of the facial skeleton and the dentition. A biometric, rhino-manometric and cephalometro-radiographic study on children with and without adenoids. *Acta Otolaryngol Suppl* 265:1–132, 1970.
62. Linder-Aronson S, Lindgren J: The skeletal and dental effects of rapid maxillary expansion. *Br J Orthod* 6:25–29, 1979.
63. Linder-Aronson S, Woodside DG: *Excess face height malocclusion: etiology, diagnosis and treatment*, Carol Stream, IL, 2000, Quintessence Publishing.
64. Lundstrom A: The importance of genetic and non-genetic factors in the facial skeleton studies in 100 pairs if twins. *Trans Eur Orthod Soc* 1954.
65. Lundstrom A, Woodside DG: Individual variation in growth directions expressed at the chin and the midface. *Eur J Orthod* 2:65–79, 1980.
66. Lundstrom A: Nature versus nature in dento-facial variation. *Eur J Orthod* 6(2):77–91, 1984.
67. McKee GJ, O'Neill G, Roberts C, et al: Nasal airflow after septorhinoplasty. *Clin Otolaryngol* 19:254–257, 1994.
68. Mitsuda ST, Pereira MD, Passos AP, et al: Effects of surgically assisted rapid maxillary expansion on nasal dimensions using acoustic rhinometry. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 109:191–196, 2010.
69. Monini S: Rapid maxillary expansion for the treatment of nasal obstruction in children younger than 12 years. *Arch Otolaryngol Head Neck Surg* 135(1):22–27, 2009.
70. Montgomery W, Vig PS, Staab EV, Matteson SR: Computed tomography: a three-dimension study of the nasal airway. *Am J Orthod* 6:363–375, 1979.
71. Moses JJ, Lange CR, Arredondo A: Endoscopic treatment of sinonasal disease in patients who have had orthognathic surgery. *Br J Oral Maxillofac Surg* 38(3):177–184, 2000.
72. Nimubona L, Jokic M, Moreau S, et al: Obstructive sleep apnea syndrome and hypertrophic tonsils in infants. *Arch Pediatr* 7(9):961–964, 2000.
73. Nustad RA, Fonseca RJ, Zeitler DZ: Evaluation of maxillary sinus disease in maxillary orthognathic surgery patients. *Int J Adult Orthodon Orthognath Surg* 1(3):195–202, 1986.
74. O'Ryan F, Carlotti A: Nasal anatomy and maxillary surgery. III. Surgical techniques for correction of nasal deformities in patients undergoing maxillary surgery. *Int J Adult Orthodon Orthognath Surg* 4:157–174, 1989.
75. Onerci TM, Ayhan K, Ogretmenoglu O: Two consecutive cases of cerebrospinal fluid rhinorrhea after septoplasty operation. *Am J Otolaryngol* 25(5):354–356, 2004.
76. Panula K, Finne K, Oikarinen K: Incidence of complications and problems related to orthognathic surgery: a review of 655 patients. *J Oral Maxillofac Surg* 59:1128–1136, 2001.
77. Passali D, Anselmi M, Lauriello M, et al: Treatment of hypertrophy of the inferior turbinate: long-term results in 382 patients randomly assigned to therapy. *Ann Otol Rhinol Laryngol* 108:569–575, 1999.
78. Perkins JA, Sie KCY, Milczuk H, et al: Airway management in children with craniofacial anomalies. *Cleft Palate Craniofac J* 34:135, 1997.
79. Phagoo SB, Watson RA, Pride NB: Use of nasal peak flow to assess nasal patency. *Allergy* 52(9):901–908, 1997.
80. Pirila T, Tikanto J: Unilateral and bilateral effects of nasal septum surgery demonstrated with acoustic rhinometry, rhinomanometry and subjective assessment. *Am J Rhinol* 15:127–133, 2001.
81. Posnick JC: Maxillary excess with or without mandibular deficiency. In Posnick JC, editor: *Craniofacial and maxillofacial surgery in children and young adults*, Philadelphia, 2000, W.B. Saunders Company, pp 1025–1056.
82. Posnick JC: Le Fort I osteotomy, sagittal splitting of the mandible, and genioplasty: historical perspective and step-by-step approach. In Posnick JC, editor: *Craniofacial and maxillofacial surgery in children and young adults,* vol 2, Philadelphia, 2000, W.B. Saunders Company, pp 1082–1102.
83. Posnick JC, Fantuzzo JJ, Troost T: Simultaneous intranasal procedures to improve chronic obstructive nasal breathing in patients undergoing maxillary (Le Fort I) osteotomy. *J Oral Maxillofac Surg* 65:2273–2281, 2007.
84. Posnick JC, Agnihotri N: Consequences and management of nasal airway obstruction in the dentofacial deformity patient. *Curr Opin Otolaryngol Head Neck Surg* 18:323–331, 2010.
85. Posnick JC, Agnihotri N: Managing chronic nasal airway obstruction at the time of orthognathic surgery: a twofer. *J Oral Maxillofac Surg* 69:695–701, 2011.
86. Rhee JS, Poetker DM, Smith TL, et al: Nasal valve surgery improves disease-specific quality of life. *Laryngoscope* 115(3):437–440, 2005.
87. Ricketts RM: Respiratory obstruction syndrome. *Am J Orthod* 54:495–507, 1968.
88. Samad I, Stevens HE, Maloney A: The efficacy of nasal septal surgery. *J Otolaryngol* 21:88–91, 1992.
89. Sandham A, Murray JAM: Nasal septal deformity in unilateral cleft lip and palate. *Cleft Palate Craniofac J* 30:222, 1993.
90. Santiago-Diez de Bonilla J, McCaffrey TV, Kern EB: The nasal valve: a rhinomanometric evaluation of maximum nasal inspiratory flow and pressure curves. *Ann Otol Rhinol Laryngol* 95(3 Pt 1):229–232, 1986.
91. Schwab JA, Pirsig W: Complications of septoplasty. *Facial Plastic Surg* 13(1):3–14, 1997.
92. Seeberger R, Kater W, Davids R, Thiele OC: Long term effects of surgically assisted rapid maxillary expansion without performing osteotomy of the pterygoid plates. *J Craniomaxillofac Surg* 38:175–178, 2010.
93. Sessions RB, Troost T: The nasal septum. In Cummings CW, editor: *Otolaryngology: head and neck surgery*, St. Louis, 1999, Mosby–Year Book, pp 786–806.
94. Siegel NS, Gliklich RE, Taghizadeh F, et al: Outcomes of septoplasty. *Otolaryngol Head Neck Surg* 122:228–232, 2000.
95. Sillman JH: Dimensional changes in the dental arches: longitudinal study from birth to 25 years. *Am J Orthod Dentofacial Orthop* 50:824, 1964.
96. Solow B, Nielsen SS, Greve E: Airway adequacy, head posture, and craniofacial

morphology. *Am J Orthodont Dentofacial Orthoped* 87:214–223, 1984.
97. Sousa JB, Anselmo-Lima WT, Valera FC, et al: Cephalometric assessment of the mandibular growth pattern in mouth-breathing children. *Int J Pediatr Otorhinolaryngol* 69:311, 2005.
98. Stewart EJ, Robinson K, Wilson JA: Assessment of patient's benefit from rhinoplasty. *Rhinology* 34:57–59, 1996.
99. Stewart MG, Witsell DL, Smith TL, et al: Development and validation of the Nasal Obstruction Symptom Evaluation (NOSE) scale. *Otolaryngol Head Neck Surg* 130:157–163, 2004.
100. Stewart MG, Smith TL, Weaver EM, et al: Outcomes after nasal septoplasty: results from the Nasal Obstruction Septoplasty Effectiveness (NOSE) study. *Otolaryngol Head Neck Surg* 130:283–290, 2004.
101. Stewart MG, Smith TL: Objective versus subjective outcomes assessment in rhinology. *Am J Rhinol* 19(5):529–535, 2005.
102. Timms DJ: The effect of rapid maxillary expansion on nasal airway resistance. *Br J Orthod* 13(4):221–228, 1986.
103. Turvey TA: Management of the nasal apparatus in maxillary surgery. *J Oral Surg* 38:331–335, 1980.
104. Turvey TA: Alteration in nasal airway resistance following superior repositioning of the maxilla. *Am J Orthod* 99:109–114, 1984.
105. Tweed CH: *Clinical orthodontics*, St. Louis, 1966, Mosby.
106. Valera FC, Travitzki LV, Mattar SE, et al: Muscular, functional and orthodontic changes in preschool children with enlarged adenoids and tonsils. *Int J Pediatr Otorhinolaryngol* 67:761, 2003.
107. Vargervik K, Mill AJ, Chierici G, et al: Morphologic response to changes in neuromuscular patterns experimentally induced by altered mode of respiration. *Am J Orthod* 85:115–124, 1984.
108. Walker D, Turvey TA, Warren D: Alterations in nasal respiration and nasal airway size following superior repositioning of the maxilla. *J Oral Maxillofac Surg* 46:276–281, 1988.
109. Warren DW, Hershey HG, Turvey TA, et al: The nasal airway following maxillary expansion. *Am J Orthod* 91:111–116, 1987.
110. Weber ZJ, Preston CB, Wright PG: Resistance to nasal air flow related to changes in head posture. *Am J Orthodon Dentofacial Orthop* 82:536–545, 1981.
111. Wenzel A, Henriksen J, Melsen B: Nasal respiratory resistance and head posture: effect of intra-nasal corticosteroid (Budesonide) in children with asthma and perennial rhinitis. *Am J Orthod* 84:422–426, 1983.
112. Wertz RA: Changes in nasal air flow incident to rapid maxillary expansion. *Angle Orthod* 38(1):1–11, 1968.
113. West RA, McNeill RW: Maxillary alveolar hyperplasia, diagnosis and treatment planning. *J Maxillofac Surg* 3:239, 1975.
113a. Williams BJD, Isom A, Laureano Filho JR, O'Ryan FS: Nasal airway function after maxillary surgery: A prospective cohort study using the nasal obstruction symptom evaluation scale. *J Oral Maxillofac Surg* 71:343–350, 2013.
114. Wriedt S, Kunkel W, Zentner A, Wahlmann U: Surgically assisted rapid palatal expansion. An acoustic rhinometric, morphometric and sonographic investigation. *J Orthofac Orthop* 62:107–115, 2001.

11

Anesthesia Techniques, Blood Loss/Fluid Replacement, Airway Management & Convalescence in the Treatment of Dentofacial Deformities

JEFFREY C. POSNICK, DMD, MD

- Use of Deliberate Hypotensive Anesthesia to Limit Blood Loss
- Use of Hemorrhage Depressors to Limit Blood Loss
- Deliberate Hypotensive Anesthesia Used in Orthognathic Surgery: Review of the Literature
- The Need for Blood Replacement in Orthognathic Surgery
- Perioperative Airway Management in the Patient with a Dentofacial Deformity
- In-Hospital Convalescence After Orthognathic Surgery
- At-Home Convalescence and Outpatient Care After Orthognathic Surgery
- Patient Education Materials

Use of Deliberate Hypotensive Anesthesia to Limit Blood Loss

Historical Perspective

Deliberate hypotensive anesthesia (DHA) to provide a less bloody field and better operative conditions was first proposed by Harvey Cushing in 1917 for neurosurgical procedures.[58] Since then, a spectrum of pharmacologic agents (e.g., nitroglycerine, calcium-channel blocking agents, short-acting beta-adrenergic blockers, purine compounds) and techniques have been used in a variety of combinations in an attempt to accomplish the same objectives.*

In 1950, Enderby and colleagues demonstrated the value of DHA to reduce blood loss in a series of 35 patients.[78-81] Eighteen of the 35 (51%) were judged to have an excellent reduction in blood loss during deliberate hypotension, and 8 of the 35 (23%) were judged to have a moderate reduction. The authors attributed this inconsistent effectiveness to individual vascular responses to the hypotensive drugs that were used. They surmised that bleeding at the surgical site could be further minimized if the wound itself was kept uppermost (rather than dependent) through the use of operating room table positioning (e.g., reverse Trendelenburg position for head and neck procedures). Through special positioning, arterial vessels in the operative field would have less pressure, veins would drain more easily, and thus bleeding at the surgical site should be less.

One of the earliest studies to quantify reductions in blood loss with DHA was performed by Eckenhoff and Rich, who completed research in patients who were undergoing a variety of operations, including rhinoplasty,

*References 5, 8, 20, 26, 33, 36, 40, 54, 55, 63, 65, 67, 82, 88, 89, 98, 99, 111, 116, 119, 128, 150, 151, 155, 160, 166, 168, 178, 181, 182, 190, 191, 196-198, 209, 219, 221, 224, 237, 241-244, 258, 265, 270, 272.

portacaval shunt, and craniotomy for aneurysm.[72,73] Half of the patients underwent DHA (n = 115), and the other half (n = 116) went without hypotension. For each of the procedures, blood loss decreased by 50% or more with hypotension techniques. This was the first DHA study to measure blood loss with the use of a control group. By doing so, it greatly added to the power of the research confirming the value of DHA.

Evidence supporting the use of DHA to reduce blood loss has most commonly been found in studies of orthopedic procedures. Thompson and colleagues studied hypotensive anesthesia to reduce blood loss during total hip arthroplasty.[255] The mean arterial pressure (MAP) of the patients (n = 30) was reduced to 50 mm Hg. The techniques used to do this included sodium nitroprusside (n = 12) and high inspired concentrations of halothane (n = 9); a control group of patients (n = 9) was normotensive. Blood loss was 1200 mL for the normotensive control subjects but only 400 mL for both hypotensive groups. No anesthesia-specific complications were seen. In 1979, Eerola and colleagues completed a controlled study of 55 patients who were undergoing total hip arthroplasty.[76] They found that the 38 patients who were given pentolinium tartrate and halothane for DHA had less blood loss. That same year, Vazeery and colleagues studied patients who were undergoing total hip replacement (n = 25). The patients were given sodium nitroprusside to lower arterial blood pressure.[262] They had significantly less blood loss than the control group (n = 25), which was not undergoing deliberate hypotension. The value of DHA to reduce blood loss has also been confirmed for other procedures, including craniotomy, middle-ear surgery, radical cancer operations, and a variety of head and neck surgeries.

Didier and colleagues suggested that the depression of cardiac output correlated better with a dry field than did arterial blood pressure.[66] To determine whether a decrease in MAP or cardiac output was the primary cause of the decreased blood loss, Sivarajan and colleagues studied "healthy" (American Society of Anesthesiologist I) subjects (n = 20) who were undergoing bilateral sagittal split ramus osteotomies of the mandible.[239] Cardiac output decreased 37% with the use of trimethaphan, but it decreased only 27% with sodium nitroprusside. Interestingly, blood loss was similar for both groups, although cardiac output was two times greater with nitroprusside. The authors concluded that blood pressure rather than cardiac output was the primary determinant of how much blood was lost.

Techniques to Induce Deliberate Hypotensive Anesthesia

Currently, the general consensus is that most patients will have less blood loss if the MAP is decreased to 50 mm Hg to 65 mm Hg in the surgical field. It is also believed that patient positioning and attention to ventilation will significantly influence venous return and therefore play important roles in minimizing blood loss. Clinical studies have focused on the use of a variety of specific drugs and combinations of drugs to keep the arterial blood pressure at the surgical site at the level believed to significantly decrease blood loss (i.e., 50 mm Hg to 65 mm Hg).

Body positioning (e.g., reverse Trendelenburg position for head and neck procedures), the hemodynamic effects of mechanical ventilation (e.g., maintaining normal or low carbon dioxide [CO_2] levels), and changes in the heart rate and the circulatory volume are factors that can be manipulated with drugs to lower blood pressure in the surgical field to the desired level. The effective use of these physiologic maneuvers helps to decrease the dose of potentially toxic drugs that are needed to produce the preferred level of hypotension.

The ideal agents for inducing hypotension should have the following characteristics: 1) ease of administration; 2) a predictable and dose-dependent effect; 3) rapid onset; 4) rapid recovery from effects; 5) quick elimination without the production of toxic metabolites; and 6) minimal effects on blood flow to the vital organs. Many anesthetic and vasoactive drugs have been used successfully in clinical practice to induce DHA for orthognathic procedures. These agents primarily include volatile anesthetics; direct-acting vasodilating drugs; beta-adrenergic receptor–blocking drugs; and combined alpha- and beta-adrenergic receptor–blocking drugs. Examples of such agents include sodium nitroprusside, nitroglycerin, esmolol, and labetalol.

Clearly, one can decrease MAP by increasing the amount of inhaled anesthetics such as sevoflurane, isoflurane, and desflurane. Certain drugs that permit moment-to-moment controlled blood pressure are the most popular. The differences in pharmacologic properties among these agents suggest that combinations of these drugs may provide a better pharmacologic profile than could be provided by any agent used alone.[234]

Effects of Deliberate Hypotensive Anesthesia on Organ Function

DHA decreases arterial blood pressure by decreasing cardiac output, by decreasing systemic vascular resistance, or by decreasing a combination of the two. Cardiac output must remain sufficiently high not only to provide adequate oxygen and energy substrates to vital organ systems but also to remove metabolic waste products before any accumulation can cause tissue damage. The effect of DHA on cardiac output depends on the balance of its effects on preload, afterload, myocardial contractility, and heart rate. Homeostatic mechanisms also come into play. Other important factors include the physical condition of the specific patient, the anesthesiologist's administration of additional drugs, and the pattern of ventilation used intraoperatively. The constant assessment of the patient's intravascular fluid volume throughout surgery is also required to ensure optimal vital organ function.

Inadequate oxygen supply to the brain and myocardium as a result of inadequate tissue blood flow or perfusion

pressure is the principal hazard of DHA, particularly with regard to the effects on cerebral metabolic homeostasis.* Studies to date have shown that the responsible use of DHA does not produce permanent changes in cerebral hemodynamics. For example, the cognitive performance of elderly patients on psychological tests given several days after total hip arthroplasty that involved DHA did not differ from their performance before surgery. The current clinical rationale for studying a lower limit for MAP (e.g., 50 mm Hg to 55 mm Hg) in normotensive patients is based on the belief that this range represents the lowest MAP at which autoregulation of the cerebral blood flow is still intact. However, for chronically hypertensive patients or for patients who are older, the curve is thought to shift to the right. In other words, a higher MAP is required in these individuals to maintain autoregulation. The partial pressure of carbon dioxide in the blood ($paCO_2$) is also an important consideration during DHA. This is because cerebral blood flow changes linearly with $paCO_2$. Whether one drug is better than another for preserving cerebral blood flow is debated.

The preservation of cardiac function is essential, but it is known to be altered by DHA techniques.† Interestingly, cardiac function is better preserved with isoflurane than with equal hypotensive doses of halothane. Studies have shown that isoflurane is suitable for healthy patients who are undergoing DHA. Clinical studies also show that the cardiovascular effects of general anesthetic doses of propofol are similar to those of isoflurane. During deliberate hypotension, the maintenance of an oxygen supply that is sufficient for the metabolic needs of the myocardium is of primary importance. As a general rule, patients with known or suspected ischemic heart disease should not undergo DHA.

Renal blood flow normally equals 20% to 25% of cardiac output. Renal circulation is greatly influenced by autoregulatory mechanisms.[61,110,159] Clinical studies suggests that normovolemic patients typically experience the rapid recovery of urine production after the discontinuation of DHA.[255] Thompson and colleagues could find no significant changes in serum creatinine, blood urea nitrogen, or serum or urinary electrolytes for patients (n = 30) who had undergone DHA.[255]

Monitoring During Induced Deliberate Hypotensive Anesthesia

Beat-to-beat measurement of arterial blood flow is mandatory for patients who are undergoing clinically significant deliberate decreases in blood pressure. The usual practice is to insert an arterial catheter for the continuous monitoring of blood pressure. Perhaps most importantly, the catheter allows for the intermittent sampling of arterial blood for acid–base status (i.e. metabolic acidosis). It also allows for the confirmation of adequacy of oxygenation and ventilation, and for serial hematocrit measurements. Electrocardiographic monitoring is also essential for detecting signs of inadequate myocardial perfusion or oxygen delivery, such as S-T segment or rhythm changes. The correlation between end tidal CO_2 and $paCO_2$ is considered somewhat unreliable during hypotension. Nevertheless, capnography still provides useful information during DHA. It may also help to avoid hyperventilation and its resultant hypocapnea, which could be harmful; the decrease in $paCO_2$ could further reduce cerebral blood flow during moderate hypotension. Pulse oximetry and temperature monitoring should also be routinely used, and the measurement of urine output is also essential. Serum electrolyte, blood gas, and hematocrit levels should also be measured at intervals.

Complications Associated with Deliberate Hypotensive Anesthesia

The precise incidence of complications with the use of DHA is difficult to determine.[19,75,41,152,179,248,253] Current data regarding non-fatal complications indicate that hypotension of 50 mm Hg to 65 mm Hg is not a risk factor for young healthy patients (American Society of Anesthesiologists [ASA] 1 and 2). Alternatively, for those individuals with underlying organ dysfunction, the use of DHA may put them at increased risk. Thus, all potential candidates should undergo a complete history and physical examination before surgery. The decision to use DHA should not be made without careful preoperative consideration of patient-specific risk factors.

Special Considerations for the Orthognathic Patient

Before beginning an orthognathic procedure, the logistics of patient positioning, the expected length of the surgery, and the confirmation of patient-specific risk factors for DHA should be discussed by the anesthesiologist and the surgeon.[264] Better drugs, more accurate monitoring, a greater level of experience with the techniques, and the consistency of the surgical procedures themselves have permitted a higher percentage of patients to benefit from DHA.* Nevertheless, relative contraindications include a history of cerebrovascular or cardiovascular disease, renal dysfunction, liver dysfunction, peripheral vascular disease, and severe anemia. Fortunately, the orthognathic patient rarely suffers from these dysfunctions. Exceptions include older individuals who are undergoing orthognathic surgery for the treatment of obstructive sleep apnea.[228,261] Given current demographics, this is likely to represent an ever-increasing patient population (see Chapter 26).

*References 13, 14, 10, 11, 9, 12, 83, 113, 117, 133, 135, 139, 145, 158, 161, 163, 174, 178, 188, 195, 221, 249, 251, 269, 273.

†References 1-3, 18, 44, 57, 60, 74, 87, 143, 193, 208, 220.

*References 7, 25, 38, 46, 49, 50, 68, 95-97, 100, 154, 157, 205, 211, 216, 229, 257, 266, 274.

Use of Hemorrhage Depressors to Limit Blood Loss

Tranexamic Acids

Tranexamic acid is a synthetic amino acid that inhibits fibrinolysis and that has been shown to reduce blood loss and the need for blood transfusion during specific surgical procedures, including total knee arthroplasty, spine surgery, cardiac surgery, and orthognathic surgery.* It has also been used for the treatment of postoperative bleeding in anticoagulated patients after oral surgery (e.g., the extraction of teeth).[32,212] **Senghore and Harris** showed that a single intravenous preoperative dose of tranexamic acid was effective for preventing excessive postoperative bleeding in healthy adult patients who were undergoing third molar extraction.[232]

Choi and colleagues completed a double-blind, randomized, controlled trial to study the effects of tranexamic acid on blood loss during orthognathic surgery.[51] This trial included 73 consecutive patients who were undergoing bimaxillary orthognathic osteotomies. Each study patient received either a bolus of tranexamic acid (20 mg/kg) or a placebo (normal saline) intravenously just before surgery. All patients underwent DHA in accordance with standard hospital protocol. Intraoperative blood loss, operative time, the transfusion of blood products, and perioperative hemoglobin and hematocrit levels were recorded. The total blood loss and the blood loss during the maxillary (Le Fort I) procedure were reduced significantly in the group that received tranexamic acid as compared with the control group. The difference in blood loss during maxillary surgery was 428 mL versus 643 mL. The difference in total blood loss was 878 cc versus 1257 cc. The authors concluded that the use of a preoperative intravenous bolus of tranexamic acid (20 mg/kg) reduces blood loss as compared with a placebo during bimaxillary orthognathic osteotomies.

Desmopressin

Desmopressin acetate is an analog to the naturally occurring hormone vasopressin.[4,114,115,138,147,222,225,245] It enhances the vascular endothelial cell release of a number of compounds, including high multimetric von Willebrand factor, factor 7c, factor 7ag, and tissue-type plasminogen activator. Desmopressin by itself has been suggested to be the factor that is responsible for the increased hemostatic activity seen in patients with liver dysfunction and uremia. Additional observations indicate that desmopressin may improve platelet function.

Both of these hemorrhage depressors—desmopressin and tranexamic acid—have been used in combination with DHA during orthognathic surgery at the University of Göteborg Hospital since 1999. **Zellin and colleagues** retrospectively evaluated and compared the total perioperative blood loss for a group of patients who were assigned to either DHA or a combination of DHA and the use of both of these hemorrhage depressors (i.e., desmopressin and tranexamic acid).[276] Thirty orthognathic patients who were consecutively operated on via a standardized Le Fort I osteotomy were assigned to either the control group (n = 15) or the treatment group (n = 15). Both groups underwent DHA. The treatment group received additional hemorrhagic depressors (i.e., both tranexamic acid and desmopressin). The mean blood loss was 740 cc in the control group and 400 cc in the treatment group. This was a statistically significant reduction in blood loss in the treatment group (1 g tranexamic acid intravenously and 0.3 μg per kilogram of body weight of desmopressin subcutaneously).

Deliberate Hypotensive Anesthesia Used in Orthognathic Surgery: Review of the Literature

Schaberg and colleagues were the first to evaluate blood loss and the human physiologic response with the use of DHA in the form of sodium nitroprusside during orthognathic surgery.[227] They studied a consecutive series of ASA I patients undergoing Le Fort I maxillary osteotomies. Each was placed in reverse Trendelenburg position (10 degrees), and sodium nitroprusside was used to induce hypotension to not less than 80 mm Hg systolic pressure or 60 mm Hg MAP. The mean estimated blood loss was 256 ± 35 mL. No control group was used for comparison.

Washburn described a retrospective series of 58 individuals who underwent maxillofacial procedures (n = 53 orthognathic patients) in which morphine was used to supplement halothane to induce hypotension.[267] The goals were to limit blood loss and to improve the surgical field. There was one death (possibly from halothane-induced liver failure), and only one patient required blood replacement.

Golia and colleagues studied nine patients who underwent either maxillary or combined maxillary and mandibular osteotomies; the DHA techniques that were used included halothane, nitrous oxide, and supplemental doses of muscle relaxants and narcotics.[106] Nitroglycerin was added for the purpose of lowering the MAP during periods with a high potential for increased blood loss (e.g., Le Fort I down-fracture). Estimated blood loss averaged 439 mL and the hemoglobin/hematocrit level fell from 13.8 hemoglobin/41 hematocrit to 12 hemoglobin/36 hematocrit (4.9%). The authors concluded that nitroglycerin was a safe and efficacious agent to help control MAP and blood loss in the surgical field during maxillary and mandibular osteotomies. No control group was used.

McNulty and colleagues completed a study of 12 patients who underwent orthognathic procedures (i.e., Le Fort I osteotomy or sagittal split ramus osteotomy of the

*References 21-23, 27, 28, 39, 41, 42, 59, 71, 77, 85, 120, 122, 125, 126, 129-132, 171, 186, 187, 214, 233, 238, 252, 254, 277.

mandible) under DHA.[169] The anesthesia agents that were used included fentanyl, isoflurane, and bolus doses (i.e., 10 mg to 20 mg) of labetalol to control the blood pressure in an attempt to improve the surgical field and limit blood loss. The authors reported clinical satisfaction with the surgical field and no complications. No control group was used.

Blau and colleagues compared esmolol in 15 patients with sodium nitroprusside in 15 other patients as the primary drugs for DHA to limit blood loss during Le Fort I osteotomy.[31] All study patients were physically rated as ASA I or II. The mean MAP averaged 58 mm Hg to 60 mm Hg, with a target range set at 55 mm Hg to 65 mm Hg. The mean heart rate was maintained at approximately 70 beats/min with esmolol and 100 beats/min with nitroprusside. The surgeons generally rated bleeding as mild to moderate and the surgical field as "drier" with esmolol. The total measured blood loss was less with esmolol (i.e., 436 mL with esmolol versus 895 mL with nitroprusside). The authors found the advantages of esmolol as compared with nitroprusside to include greater control of the blood pressure, the reduction of blood loss, and a drier surgical field.

Precious and colleagues studied 50 orthognathic surgery patients in a prospective, randomized, blocked, stratified, and single-blind fashion.[206] All patients underwent either sagittal split ramus osteotomies of the mandible, Le Fort I osteotomy, or genioplasty. One group of patients (n = 25) received DHA, whereas the other group (n = 25) received anesthesia with no specific attempt to reduce blood pressure during the operation. The surgeon, who was unaware of which group the patient had been assigned to, rated the surgical field every 15 minutes. At the completion of surgery, three different methods were used to estimate or calculate blood loss. The duration of the procedure was recorded from the time of the first incision to the time of the last stitch placement. The estimated blood loss was significantly less when DHA was used. The surgical field was also rated better, but there was no significant difference in the duration of the procedure with or without the use of DHA. The authors concluded that DHA results in both reduced blood loss and improved visibility in the surgical field.

Enlund and colleagues studied 36 patients who were undergoing orthognathic surgery.[84] Each patient was randomly assigned to be either hypotensive (n = 18) or normotensive (n = 18) during surgery. Hypotension was achieved with the use of isoflurane to a MAP of 50 mm Hg to 64 mm Hg. The hypotensive group had significantly less blood loss per minute. Only two of the patients in each group underwent bimaxillary osteotomies. A majority of the patients in each group underwent only mandibular ramus osteotomies. None of the patients in either group required blood replacement.

Dolman and colleagues completed a prospective, randomized, blinded-to-the-surgeon clinical trial of DHA in a consecutive series of orthognathic patients.[69] The purpose of the study was to compare the quality of the surgical field, the blood loss, and the operative time with either hypotensive or normotensive anesthesia during Le Fort I osteotomy. The study patients (n = 23) were randomized into normotensive or hypotensive anesthesia treatment groups. No local anesthesia with a vasoconstrictor was used by the surgeon. In the experimental group, hypotensive anesthesia was maintained with propofol (3 mg/kg/hr), sufentanyl (0.5 mg/kg/hr), and isoflurane (0.5% in 100% oxygen). The quality of the surgical field in both groups was assessed intraoperatively by direct observation and again postoperatively with video imaging. The surgical time was measured on the videotape, and blood loss was measured by volumetric and gravimetric techniques. There was a statistically significant correlation between the surgeon's perception of the quality of the surgical field and the blood pressure. There was a statistically significant reduction in blood loss when using hypotensive anesthesia. However, there was no significant reduction in operative time when using hypotensive anesthesia. The authors concluded that hypotensive anesthesia is a valuable technique for reducing blood loss and improving the quality of the surgical field during Le Fort I osteotomy.

Praveen and colleagues completed a prospective randomized clinical study to assess DHA and blood loss during orthognathic surgery.[204] A total of 53 patients who were undergoing orthognathic surgery were included in the study. The patient ages ranged from 15 to 33 years. They were randomly allocated to have either normotensive anesthesia or to be given DHA. Median blood loss with DHA was 200 mL; under normotensive anesthesia, it was 350 mL. The authors concluded that there was a pronounced reduction in blood loss during orthognathic operations with the patient under DHA as compared with normotensive anesthesia.

Manola and colleagues completed a prospective randomized comparison of three types of pharmacologic agents to induce hypotension during functional endoscopic sinus surgery.[165] The patients were divided into three equal groups in accordance with the agents used: 1) sufentanyl/sevoflurane; 2) remifentanil/propofol; and 3) fentanyl/isoflurane. Evaluation included surgical field rating and measured blood loss. The authors found that the quantity of blood loss and the visibility of the surgical field were improved with the use of either remifentanil and sufentanyl as compared with fentanyl.

Choi and colleagues completed a comparative study to report about the use of three different pharmacologic drugs to induce DHA in patients who were undergoing orthognathic surgery (i.e., Le Fort I and sagittal split ramus osteotomies of the mandible).[49] The 50 adult patients were randomly allocated to receive either nitroglycerin (n = 25) or remifentanil (n = 25). The MAP and the heart rate were measured at intervals. The heart rates and blood pressures were significantly better controlled with the use of remifentanil as compared with nitroglycerin. Interestingly, there were no significant differences in the surgical field ratings or the measured blood loss between the two groups.

Farah and colleagues completed a comparative study to report on the use of two different pharmacologic protocols to induce DHA in patients submitted to orthognathic surgery.[90] Twenty ASA 1 patients between the ages of 17 and 44 years were randomly assigned into two groups: group 1 (clonidine and remifentanil) and group 2 (dexmedetomidine and isoflurane). Other drugs that were used as part of the anesthesia regimen were common to both groups. Specific patient parameters were assessed, including arterial blood pressure; heart rate; temperature during the intraoperative and postoperative periods; incidence of nausea and vomiting; postoperative pain; awakening time; extubation time; and postanesthesia recovery time. The results of the study showed that there were no significant differences between the two groups with respect to physiologic responses or surgical time. Both study protocols proved to be effective and safe. Each was suggested as a satisfactory option to achieve DHA during orthognathic procedures when there is an expectation of significant blood loss.

To review, the literature confirms that DHA is effective for decreasing blood loss and providing better visibility in the surgical field during orthognathic procedures. There are many available drugs and techniques that have been used successfully to lower arterial blood pressure to accomplish these goals. The mechanisms of action of available drugs differ and produce complex changes in reflexes and subsequently the blood flow to various organs. For the healthy young patient, complications are rare. The anesthesiologist and the surgeon must assess each patient carefully and consider the potential risks and complications as well as the benefits before using DHA techniques during orthognathic surgery.

The Need for Blood Replacement in Orthognathic Surgery

Background

For individuals who are undergoing complex orthognathic surgery (i.e., Le Fort I and sagittal split ramus osteotomies of the mandible)—often in combination with other simultaneous procedures (e.g., genioplasty, liposuction, septoplasty, inferior turbinate reduction, removal of wisdom teeth)—a preoperative doctor–patient discussion of the need for intraoperative blood replacement is not just an academic issue.[92,103,107,149,163,201] Published studies indicate that individuals who are undergoing bimaxillary surgery require blood replacement in the range of 2.5% to 75%.[15,64,138,142,148,156,200,218,223,226,235,259,274] Rummasak and colleagues retrospectively studied patients who underwent bimaxillary osteotomies (n = 208) during a 4-year timeframe from 2005 to 2009 at a single institution. The authors reviewed possible factors for intraoperative blood loss and looked for statistical significance. They documented that, with consistent anesthesia techniques, the two factors of greatest importance for blood loss were 1) the experience of the surgeon and 2) the overall operative time.[223]

Madsen and colleagues conducted a prospective study to evaluate the predictive value of the viscoelastic properties of whole blood samples collected preoperatively in relation to intraoperative blood loss in patients who were undergoing orthognathic surgery (n = 41). They concluded that, with other variables controlled for, the etiology of intraoperative bleeding can be predicted by means of preoperative thromboelastography. This is a global method that addresses the complex interplay among coagulation factors, blood platelets, and components of the fibrinolytic system. Blood platelet count, activated partial thromboplastin time, prothrombin time, plasma fibrinogen concentration, and D-dimer concentration were determined by routine methods. The authors conclude that thromboelastography results in individual patients can be used for the evaluation of bleeding risk. Further studies will be required to confirm the validity of their findings.[162]

Methods to limit the need for blood replacement continue to evolve and include refinements in DHA techniques as well as in surgical management (i.e., patient selection, collaborative clinical efforts, and the efficiency of the operation and the postoperative care).[102] Despite stringent donor screening criteria and the rigorous testing of every unit of blood, there remains a risk for the transmission of several pathogens.[70,93,104,108,134,177] For viruses such as the human immunodeficiency virus (1 in 2,135,000), hepatitis B virus (1 in 205,000), and hepatitis C virus (1 in 2,000,000), this is due mainly to "window-period" donations.[34,247] The risks of newly emerging pathogens that can affect blood safety (e.g., Creutzfeldt-Jakob disease) remain unclear. There are other pathogens (e.g., cytomegalovirus, parvovirus B19) that are common in the general donor population and that may pose a risk in the immunosuppressed patient.[213] In addition, allogenic transfusion has been shown quantitatively to be an important contributor to postoperative infection.[94,260]

The options of autogenous and donor-directed blood banking and their popularity for elective surgery are attributed to their perceived safety, to refinements in blood bank administration, and to public concern about the hazards of allogenic blood replacement.[16,86,91,105,118,167,184,210,240] In at least one state (California) and one nation (Germany), the preoperative discussion of blood replacement options is mandated by law and must be documented in the medical record. Other alternatives to allogenic transfusion include acute normovolemic hemodilution, intraoperative blood salvage, and perioperative therapy with recombinant human erythropoietin (Procrit).[35,101,136,170,173,180,230,231]

The absolute need for and actual use of blood transfusion in the practice of orthognathic surgery remains dependent on considerations that relate to total whole body oxygen delivery and its ability to meet the body's metabolic needs.[56,121,175,189] Many individual hospitals have instituted strict criteria for the transfusion of blood products.[215] Although mandated criteria may decrease the use of blood transfusions, it remains unclear if complications or delays in recovery have occurred as a result.[250]

Review of Study

Posnick and colleagues completed a retrospective study to review blood replacement practices in a consecutive series of a single surgeon's experience with patients who all underwent, at a minimum, simultaneous Le Fort I maxillary osteotomy, bilateral sagittal split ramus osteotomies of the mandible, septoplasty, and inferior turbinate reduction procedures.[202] Many patients also underwent additional adjunctive procedures.

A chart review of a consecutive series of the surgeon's patients (n = 34) who underwent elective bimaxillary orthognathic surgery (Le Fort I osteotomy and sagittal split osteotomies of the mandible) in combination with intranasal (septoplasty and inferior turbinate reduction) and often other simultaneous procedures during a 5-month time frame at a single institution was carried out (see Table 11-1). The chart review occurred at least 3 months after surgery for all patients.

Each patient was also evaluated for complications specific to the procedures that were carried out. Potential complications specific to the orthognathic procedures included infection that required the extended use of antibiotics or drainage procedures; dental injury; fibrous union; aseptic necrosis; bleeding that required secondary treatment; and oroantral, oronasal, and orocutaneous fistulas. Potential complications specific to the intranasal procedures included postoperative nasal bleeding that required packing or cauterization; the need for postoperative blood transfusion specific to nasal bleeding; the presence of septal perforation; and postoperative nasal obstruction that required additional procedures within 3 months after orthognathic surgery. Potential perioperative airway compromise was also reviewed, including the need for delayed extubation, reintubation, or tracheotomy. Potential complications specific to transfusions, including infections and autoimmune reactions, were also sought. The patient records that were reviewed included the office chart; the hospital records; and the data stored at the Red Cross (hospital) blood bank. Specific study data collected for each patient (n = 34) can be found in Table 11-1.

The study group included 15 male patients and 19 female patients. The mean age was 22 years and ranged from 13 to 65 years; the median age was 22 years. Thirteen of the 34 patients were less than 18 years old. All patients were treated with a combined orthodontic and surgical (orthognathic and intranasal) approach. The most frequent pattern of jaw deformity that was identified for the study group was asymmetric mandibular excess in 9 out of 34 patients (26%); this was followed by a cleft-craniofacial syndrome being found in 7 out of 34 patients (21%) and a long face growth pattern in 7 out of 34 patients (21%). Additional simultaneous facial procedures that were completed varied. Forty-four percent of the patients (15 out of 34) underwent the simultaneous removal of wisdom teeth. Twenty-one percent of the patients (7 out of 34) also underwent suction-assisted lipectomy of the neck. Eighty-eight percent of the patients (30 out of 34) underwent an osseous genioplasty. Eighteen percent of the patients (6 out of 34) underwent iliac (hip) bone grafting. Fifty-nine percent of the patients (20 out of 34) underwent maxillary (Le Fort I) segmentation. Six percent of the patient (2 out of 34) underwent otoplasty. For all patients, the intranasal procedures included both septoplasty (submucosal resection) and inferior turbinate reduction.

All patients were either extubated in the operating room or within the first hour after surgery while in the recovery room. No patients required overnight intubation, reintubation, or tracheotomy. With respect to complications, no patients required nasal packing or postdischarge blood transfusion. No patient required a return to the operating room for the control of bleeding or airway management.

Twenty-six of the 34 study patients (76%) elected to auto-donate (bank) blood in advance of their surgeries. Two of the 34 study patients auto-donated two units of blood, whereas 24 of the 34 auto-donated just one unit. Only one of the 26 patients that auto-donated received back her banked blood (1 out of 34 or 3% of study patients); she received just one unit of autogenous blood. One of the 34 study patients received a cross-matched anonymously donated (i.e. allogenic) unit (1 out of 34 or 3%). This patient did not elect to auto-donate blood in advance of surgery, and she received just the one unit of blood. Actual autogenous blood use within the group that banked blood was assessed to determine the proportion of discarded stored units. On the basis of the total units of blood that were pre-donated, 28 out of 29 (97%) of the stored units were not used and therefore discarded.

The records of each study patient (n = 34) were specifically reviewed for possible complications associated with the harvesting of autogenous blood and receiving a blood transfusion. Neither of the two patients who received packed red blood cells (one autogenous and the other homologous) experienced intraoperative or postoperative cardiovascular or respiratory compromise. There was no evidence of an adverse event such as an allergic reaction, a transfusion-related infection, hemolysis related to incompatible blood type, or an alteration of the immune system in either patient. According to a review of the blood bank records and discussions with the patients and their families, no complications were known to have occurred as a result of the blood-harvesting process.

Controversies in Blood Replacement for Orthognathic Surgery

Autogenous blood transfusion is the collection and then transfusion of a patient's own blood. Allogenic blood is collected from someone other than the patient. During recent years, insights into the use of autogenous blood led to a complete revision of the National Heart, Lung and Blood Institute's recommendations regarding the use of autogenous blood. Despite the reduction of the risk of transmitting viruses such as human immunodeficiency

TABLE 11-1 Blood Replacement for Complex Orthognathic Surgery

Patient			Jaw Deformity				
Entry No.	Gender	Age (in years)	Pattern	Additional Procedures	Blood Products	Preoperative Hematocrit Level	Lowest Hematocrit Level and Location (OR versus RR)
1	F	63	Mand def	Chin; neck lipo	None	40	28 RR
2	F	26	Mand def	2 segs; chin	1 auto, discarded	33	29 RR
3	M	39	Max and mand def	3 segs; chin; ICBG; neck lipo	2 auto, discarded	38	27 RR
4	M	35	Long Face	Third molars; 2 segs; chin	2 auto, discarded	38	35 RR
5	M	20	Asym mand excess	Third molars; 2 segs; chin	1 auto, discarded	37	32 OR
6	F	21	Asym mand excess	2 segs; chin	1 auto, discarded	32	26 OR
7	F	20	Long Face	3 segs; chin	1 auto, discarded	35	29 OR
8	F	16	Long Face	Third molars; chin	1 auto, discarded	35	30 RR
9	M	18	Cleft Jaw	ICBG; 2 segs; chin	1 auto, discarded	37	29 RR
10	F	19	Long Face	1 seg; chin	1 homo, given	34	26 OR
11	F	19	Cleft Jaw	Chin; ICBG; third molars	1 auto, discarded	39	27 OR
12	F	13	HFM	Third molars; chin	1 auto, discarded	38	32 RR
13	F	13	Max Def/Mand excess	Chin; scalp exc; third molars	1 auto, discarded	28	26 OR
14	F	14	Cleft Jaw	ICBG; 3 segs; chin; third molars	None	35	31 OR
15	M	18	Asym mand excess	Chin; ICBG; third molars	1 auto, discarded	40	39 OR
16	F	16	HMF	Chin; third molars	1 auto, discarded	36	27 RR
17	F	19	Asym mand excess	Chin	None	38	32 RR
18	M	17	Max Def/Mand excess	3 segs; chin; third molars	1 auto, discarded	38	33 RR
19	M	25	Asym mand excess	2 segs	1 auto, discarded	45	39 RR
20	F	16	Max Def/Mand excess	3 segs; chin	1 auto, discarded	38	28 RR
21	M	17	Asym mand excess	2 segs; chin; third molars	1 auto, discarded	35	30 RR
22	M	15	Cleft Jaw	2 segs; chin; third molars	1 auto, discarded	40	35 RR

Continued

TABLE 11-1 Blood Replacement for Complex Orthognathic Surgery—cont'd

Patient			Jaw Deformity				
Entry No.	Gender	Age (in years)	Pattern	Additional Procedures	Blood Products	Preoperative Hematocrit Level	Lowest Hematocrit Level and Location (OR versus RR)
23	M	22	Max Def/Mand excess	Chin	None	43	38 RR
24	F	15	Cleft Jaw	3 segs; chin; ICBG; neck lipo	1 auto, discarded	33	28 RR
25	F	14	Long Face	3 segs; chin; otoplasty; third molars; neck lipo	1 auto, given	38	26 RR
26	F	23	Max mand def	ICBG; chin; 3 segs	1 auto, discarded	33	27 OR
27	F	26	Asym mand excess	3 segs; chin	1 auto, discarded	35	29
28	M	17	Asym mand excess	3 segs	1 auto, discarded	39	34
29	M	14	Cleft jaw	ICBG; otoplasty; chin; third molars	None	33	30
30	F	55	Long Face	3 segs; chin; neck lipo	1 auto, discarded	31	26
31	F	28	Long Face	3 segs; chin	1 auto, discarded	33	30
32	M	18	Max Def/Mand excess	Chin; third molars	1 auto, discarded	34	33
33	M	24	Max Def/Mand excess	Chin; neck lipo	1 auto, discarded	36	34
34	M	22	Asym mand excess	Chin	1 auto, discarded	39	35

Asym, asymmetric; *auto,* autologous blood donation; *def,* deficiency; *exc,* excision; *F,* female; *HFM,* hemifacial microsomia; *homo,* homologous blood donation; *ICBG,* iliac crest bone graft; *lipo,* liposuction; *M,* male; *mand,* mandibular; *max,* maxillary; *OR,* Operating Room; *RR,* Recovery Room; *seg(s),* segment(s). From Posnick JC, Rabinovich A, Richardson DT: Blood replacement for complex orthognathic surgery, *J Oral Maxillofac Surg* 68:54–59, 2010.

virus, hepatitis B virus, or hepatitis C virus, the transfusion of autogenous blood remains safer than that of allogenic blood, and it is considered appropriate for properly selected patients. However, there is still a risk of adverse reactions, bacterial contamination, and errors in the identification of the units. Directed donations (i.e., blood donations from a friend or family member) for a designated patient are not as safe as the patient's own blood and must not be considered equivalent to an autogenous blood transfusion.

If transfusion is likely for a planned surgical procedure, there are several theoretical ways of obtaining autogenous blood, including 1) preoperative autogenous blood donation; 2) intraoperative blood salvage; and 3) acute normo-volemic hemodilution. In general, autogenous (intraoperative) blood collection techniques are considered undesirable for orthognathic surgery as a result of the associated saliva contamination. Acute normo-volemic hemodilution is the removal of blood and the simultaneous infusion of cell-free solutions to maintain intravascular volume before surgical blood loss. The removed blood is transfused during or after surgery, as needed, to maintain the desired hemoglobin levels. The use of this technique requires significant clinical expertise because of its associated physiologic consequences, and there is increased risk with DHA during orthognathic surgery.

The current recommendation of the National Heart, Lung and Blood Institute's conference report is that autogenous blood donations are indicated for patients who are having surgical procedures for which blood is usually crossmatched.[185] The less likely the transfusion for a specific

procedure, the more likely that donated blood will not be used. The recommendations are that patients should not be encouraged to donate blood for autogenous use during surgery unless there is a greater than 10% likelihood that they will need it. The historic literature indicates a wide range (i.e., 2.5% to 75%) with regard to the use of transfusion during bimaxillary orthognathic surgery. The review study performed by Posnick and colleagues indicated only a 6% need.[202] In current practice, the need for blood replacement during bimaxillary orthognathic surgery may be closer to 2%. The high-risk individual (e.g., an adult with obstructive sleep apnea and other comorbidities) can usually be recognized from the outset, and preparations can be made in advance (i.e., blood typing and cross-matching).

Unfortunately, it is not possible to give a quantitative answer to the question, *"When should autogenous or allogenic blood be transfused?"* Practical guidelines have been based on hemoglobin levels, but the inadequacy of this parameter is well known. In general, transfusions should be strongly considered when the patient's hemoglobin level is at 7.0 g per liter (hematocrit = 21) or less. However, in the majority of cases, orthognathic surgery is carried out in young, healthy patients who are free of systemic disease. Thus, there is no absolute hemoglobin level that should automatically trigger a transfusion. A transfusion should be given only on the basis of sound clinical judgment.

Another common question is, *"Should the indications for the use of autogenous blood transfusion differ from those for the use of allogenic blood transfusion?"* If precise indications for a blood transfusion could be defined, autogenous blood would not be given more frequently than allogenic. Although the benefits of allogenic and autogenous blood transfusions are the same, the risks are not. Therefore, the risk-to-benefit ratio clearly supports the more liberal use of autogenous blood. It has been documented that patients who preoperatively donate blood are more likely to be transfused earlier and more frequently than patients who do not auto-donate. Studies have challenged the use of autologous blood donations for elective surgery, because there is the possibility of blood overcollection, the wasting of autologous units, and the costs of preparation and storage. Cohen developed an interesting mathematical model to analyze the use of preoperative autogenous blood donation.[53] The model clarifies how clinicians can customize autogenous blood collection for individual patients to minimize waste (i.e., the need to discard non-transfused blood).

A limited survey of hospitals in the United States determined that the fee generated by the blood bank for each unit of autologous blood that is harvested is estimated to be $255. This includes the storage and delivery of the blood to the operating room or for the disposal of the blood product (i.e., hazardous waste) if it is not used. This fee is typically paid in full by the patient's medical insurance carrier. If the operative procedure for which the blood is donated is not covered by the patient's medical insurance carrier, then the patient is expected to pay the fee.

A critical review of the literature and the evaluation of current clinical practice indicates that, with the effective use of DHA techniques, only a small percentage of individuals who are undergoing even complex orthognathic and intranasal surgery should require blood replacement. Close collaboration between the surgical and anesthesia teams and the recovery of patients in a safely monitored environment will continue to improve operating room and postoperative safety and to reduce blood loss and the need for transfusion in the orthognathic patient.

Perioperative Airway Management in the Patient with a Dentofacial Deformity

A basic airway decision for the surgeon and the anesthesiologist to make for the individual who is to undergo orthognathic surgery and who therefore requires general anesthesia is whether to use induced anesthesia and then directly intubate with or without the use of a Glide Scope (Bell Medical Inc., St. Louis, MO); to use awake fiber-optic intubation; or to use a percutaneous technique (e.g., cricothyroidotomy and tracheostomy). The patient with a dentofacial deformity who presents for general anesthesia should be considered to have a difficult airway until it is proven otherwise (Fig. 11-1).[45,48,123,127,141,146,172,203,207,217]

Head and Neck Examination

The head and neck examination of a patient who is scheduled to undergo endotracheal intubation should include his or her mouth-opening ability; the ability to protrude the lower jaw; any limitations of neck extension; the measurement of the thyromental distance; and the Mallampati test.[140,164,165] These parameters cover the standard reference points to determine whether or not to expect a difficult airway. These parameters represent components of the basic head and neck airway evaluation recommended in the guidelines of the American Society of Anesthesiologists.[6]

1. *Mouth-opening ability.* This is measured as the interincisor distance. A value of less than 4 cm has been proposed as an indicator of possible difficult intubation. Fibrous or bony temporomandibular joint ankylosis will absolutely diminish mouth opening. Muscle spasms or pain may also limit mouth opening, but these factors can be overcome with muscle relaxants and analgesics.
2. *The inability of the individual to protrude the lower jaw forward.* When the mandibular incisors cannot protrude in front of the maxillary incisors, this is considered to be an indicator of a difficult intubation. It is often an indirect test for mandibular retrognathia. In addition, it can be a deceiving indicator of normal jaw morphology in the individual with a normal mandible but with maxillary deficiency.
3. *Limited neck extension.* An angle of less than 20 degrees as measured between the occlusal surface of the maxillary teeth and the neck when it is in full extension is felt to be suggestive of a difficult direct laryngoscopy. A

• **Figure 11-1** Perioperative risk to the airway and its management should be considered during all phases of orthognathic surgery, including before nasotracheal intubation, during surgery, at the time of extubation, and soon after extubation. Individuals who are scheduled for orthognathic surgery who have been recognized to be at high risk for airway compromise should be discussed in advance with the anesthesia team to arrange for special needs. Three patient examples in which these issues came into play are shown: an adult with Noonan syndrome; a teenage girl with Treacher Collins syndrome; and a teenager with the Klippel–Feil anomaly. Each of these patients also has the potential for cervical spine anomalies with limited neck range of motion. The Risk of spinal cord injury should also be evaluated before orthognathic surgery.

percentage of dentofacial deformity patients will have cervical spine anomalies that prevent adequate and safe neck extension.

4. *The Mallampati test.* The visibility of the pharyngeal structures when the mouth is wide open is considered to be of limited use as an individual assessment. However, when this is considered in combination with other physical findings, it is of some value. The dentofacial deformity patient with baseline obstructive sleep apnea is likely to have a Class IV Mallampati classification. The Mallampati classification is determined by having the awake patient in the sitting position open his or her mouth widely and then protrude the tongue completely forward. The clinician then documents which structures can be visualized.
   Class I: The soft palate, the fauces, the entire uvula, and the pillars are visualized.
   Class II: The soft palate, the fauces, and a portion of the uvula are visualized.
   Class III: The soft palate and the base of the uvula are visualized.
   Class IV: Only the hard palate is visualized.
5. *The thyromental distance.* This is often used as an indirect gauge of mandibular morphology. The ratio of a patient's overall height to his or her thyromental distance may be used as an indirect assessment of mandibular deficiency.

All patients with dentofacial deformities should have documentation by the anesthesiologist of the airway examination, as stated previously. The preoperative evaluation maybe conducted in a clinic setting or immediately before surgery. It is also recommended that the evaluation document the following:

- The status of the teeth (e.g., mobility, missing teeth, removable and fixed appliances, general oral heath);
- The neck circumference (this is an indirect indicator of obstructive sleep apnea)
- The overall body habitus
- Any presenting dentofacial deformity (e.g., maxillomandibular deficiency)
- Any anatomic cause of nasal airway obstruction (e.g., septal deviation, posterior choanal atresia, enlarged turbinates)

Prevention of Complications during Tracheal Intubation

According to the American Society of Anesthesiologists, four principles are central to the prevention of complications during tracheal intubation:

1. *The maintenance of oxygenation is essential.* Preoxygenation is performed before the induction of anesthesia. Mask ventilation is also used in between attempts at tracheal intubation.
2. *Trauma to the upper airway must be prevented.* The first attempt at tracheal intubation should be performed under optimal conditions (i.e., patient positioning, preoxygenation, and equipment preparation).
3. *The anesthesiologist should have a backup plan in place before initiation of the primary technique.* He or she must have the skills and equipment needed to execute the

backup plan for the patient with a difficult airway. The recent introduction of video-assisted laryngoscopy instruments have lessened the need for more invasive methods to secure the airway, such as the use of a fiber-optic bronchoscope and percutaneous techniques. The backup plan should include the eventuality that, if noninvasive techniques do not restore oxygenation, then cricothyroidotomy (which is generally the percutaneous airway method of choice) is ready to go. In an actual *"cannot intubate—cannot ventilate"* situation, immediate percutaneous access to the cricothyroid membrane is essential.

4. *The anesthesiologist should seek the best help available as soon as difficulty with tracheal intubation is experienced.* He or she should be aware of available backup personnel or colleagues who can be quickly called in to assist during an emergency.

Video 5

NOTE: In the patient with a dentofacial deformity, serious airway morbidity (e.g., cardiopulmonary arrest, or unnecessary tracheostomy)—although infrequent—is a much worse outcome than performing an awake fiber-optic intubation that may not have been necessary.

The presurgical airway evaluation by an experienced anesthesiologist provides a reasonable indication of potential intubation difficulty and should always be performed. The anesthesiologist must then make a judgment regarding whether anesthesia induction followed by direct laryngoscopy (as a first effort) followed by mask ventilation with percutaneous rescue (if direct laryngoscopy cannot be accomplished) is likely to be successful. The intrinsic limitations of the pre-anesthesia airway assessment mean that the preparation of an airway strategy for the management of unanticipated difficulties is the ultimate key to safe practice.

When the patient with a dentofacial deformity is felt to be at high risk for challenging airway management, advance planning ensures that necessary equipment and skilled personnel are available. The *"difficult airway"* is defined as the clinical situation in which a conventionally trained anesthesiologist experiences difficulty with face-mask ventilation of the upper airway, difficulty with tracheal intubation, or both. By definition, the difficult airway represents a combination of patient-specific factors, the specific clinical setting, and the skills of the individual practitioners.

Adequate *face-mask ventilation* may not be possible as a result of inadequate mask seal, excessive gas leak, or excessive resistance to the ingress or egress of gas. Difficulty with laryngoscopy is said to occur when it is not possible to visualize any portion of the vocal cords. *Difficult tracheal* intubation is defined as a tracheal intubation that has required multiple attempts in the presence or absence of true tracheal pathology. *Failed intubation* is defined as the inability to place an endotracheal tube after multiple intubation attempts.

The practical guidelines for the management of the difficult airway have been reported by the American Society of Anesthesiologists Task Force on Management of the Difficult Airway.[6] The guidelines recommend the following: 1) a detailed evaluation of the airway; 2) a general physical examination; 3) additional patient-specific evaluations, as needed; 4) basic preparation for difficult airway management; 5) a strategy for the intubation of the difficult airway; 6) a strategy for the extubation of the difficult airway; and 7) arranging for follow-up care.

The anesthesiologist should have a preformulated strategy for the intubation of the patient with a dentofacial deformity and a difficult airway. This strategy should include the following:

1. An assessment of the likelihood and anticipated clinical impact of four basic problems that may occur alone or in combination:
   - Difficult ventilation
   - Difficult intubation
   - Difficulty with patient cooperation
   - Difficult tracheostomy (cricothyroidotomy)
2. A consideration of the relative clinical merits and feasibility of the three basic airway management choices:
   - Awake fiber-optic intubation versus intubation after the induction of general anesthesia
   - The use of noninvasive techniques for the initial approach to intubation (e.g., direct conventional laryngoscopy, laryngeal mask airway–assisted airway attainment, video-assisted laryngoscopy) versus the use of invasive techniques (e.g., percutaneous cricothyroidotomy, tracheostomy)
   - The preservation of spontaneous ventilation during intubation attempts versus the ablation of spontaneous ventilation during intubation attempts
3. The anesthesiologist must select a primary approach to the difficult airway:
   - Awake fiber-optic intubation in the patient who cannot be adequately ventilated and who will be difficult to intubate
   - The patient who can be adequately ventilated but who will be difficult to intubate
   - The life-threatening situation in which the patient cannot be ventilated or intubated and who will require immediate cricothyroidotomy or tracheostomy
4. The anesthesiologist should formulate in advance an alternative plan in the event that the primary approach is ineffective.

Special Airway Considerations in the Patient with a Dentofacial Deformity

The patient with a dentofacial deformity who is scheduled to undergo orthognathic surgery requires nasotracheal intubation as a first choice. The consideration of patient-specific intubation difficulties should be discussed between the surgeon and the anesthesiologist before arrival in the operating room (see Figure 11-1).

Difficulty with passing the endotracheal tube through the external nasal valve and the nasal cavity:

- The dentofacial deformity patient will often have an obstructed nasal airway as a result of a combination of scarred external nasal valves; a deviated septum; hypertrophic turbinates; a constricted bony nasal cavity (e.g., floor of the nose, lateral nasal walls); or posterior choanal atresia.

Difficulty with passing the endotracheal tube past the soft palate and nasopharyngeal walls into the oropharynx:

- In the patient with a cleft palate, a pharyngoplasty may be in place. This will result in limited lateral ports if a superiorly based pharyngeal flap is in place. There may be a limited central port if an orticochea (sphincteroplasty) flap is in place. Either of these forms of pharyngoplasty in the patient with a cleft palate may interfere with the passage of a nasotracheal tube.
- If the endotracheal tube cannot be passed through the nose and into the oropharynx, then the insertion of the endotracheal tube through a submental (skin) incision maybe carried out. This will accomplish intubation objectives but avoid interference with the occlusion during surgery. To accomplish the submental approach, the endotracheal tube is first passed through the oral cavity and into the pharynx. With the use of a laryngoscope, it is then passed through the vocal cords. The patient will be adequately ventilated while the submental approach is accomplished. A sharp submental incision is made, and this is followed by blunt dissection just behind the mandible and then into the mouth. The end of the endotracheal tube is pulled through the floor of the mouth and out of the submental incision for the administration of anesthesia.

Difficulty with inserting and using the laryngoscope to visualize the vocal cords as a result of limited mouth opening:

- The presenting dentofacial malformation may result in limited mouth opening (e.g., Treacher Collins syndrome, hemifacial microsomia)
- Temporomandibular joint pathology (bony or fibrous ankylosis) will limit the extent of mouth opening (i.e., the interincisal distance).
- Masticatory muscle spasm will limit mouth opening and may be confused with bony or fibrous temporomandibular joint ankylosis.

Difficulty with visualizing the vocal cords as a result of mandibular hypoplasia with a retro and superior positioned tongue within a limited floor of the mouth space:

- The presenting mandibular malformation or deformity (e.g., moderate to severe hypoplasia) will result in the retro and superior positioning of all of the soft tissues of the floor of the mouth and the anterior neck. This will result in limited visualization of the vocal cords. This may be true even for a non-obese patient in the neck-extended position during a full jaw-thrust maneuver.

Difficulty with visualizing the vocal cords as a result of limitations in the range of motion of the neck:

- A percentage of patients with dentofacial deformities will have congenital limitations in the range of motion of the neck (e.g., hemifacial microsomia [Goldenhar variant], Klippel–Feil anomaly). This should be assessed before the patient arrives in the operating room.
- For the patient with baseline cervical spine anomalies, evaluation by a spine surgeon and specialized radiograph studies may be indicated in the outpatient setting before arrival in the operating room.
- Possible advantages of intraoperative spinal monitoring should be discussed before arrival in the operating room.

It is likely that the orthognathic surgeon will be most familiar with the nasal, oral, pharyngeal, temporomandibular joint, and cervical spine patient-specific anatomy. Potential locations of obstruction and limitations in neck and jaw range of motion should be discussed before arrival in the operating room. The surgeon should be ready to assist with the passage of the nasotracheal tube through the external nasal valve and the nasal cavity and into the oropharynx. The anesthesiologist will then complete direct laryngoscopy, video-assisted laryngoscopy (i.e., GlideScope), or fiber-optic intubation to pass the endotracheal tube through the vocal cords.[144,146] In anticipation of the possible need for percutaneous airway management, the team should have the skill and readiness of equipment to complete either a cricothyroidotomy or a formal tracheostomy, as indicated.

Immediate Preintubation Preparation of the Airway

Preparation of the nasopharynx is useful before attempting nasotracheal intubation. It begins with questioning the awake patient about the ease of breathing through one nares versus the other. Preparation of the nasal airway with topical agents just before entering the operating theater is also routine; this allows the agents to reach peak effect before intubation. Topical vasoconstrictors such as oxymetazoline (a direct-acting sympathomimetic vasoconstrictor) or Neo-Synephrine (an alpha-adrenergic agonist and a potent vasoconstrictor) will reduce nasal mucosal edema, thereby improving the patency of the nares and also reducing the potential for bleeding.

After the nasal airway is decongested and sufficiently anesthetized, sequential dilation with a nasopharyngeal

airway tube (nasal trumpet) is recommended to clarify the expected ease or difficulty of passage of the nasotracheal tube. The nasopharyngeal airways should be lubricated with either standard surgical lubricant or topical lidocaine jelly, which may also be mixed with topical vasoconstrictor. The use of benzocaine spray as a topical anesthetic is another option; however, as a result of the rapid uptake of this drug, the high rate of absorption through the mucus membranes, and the risk of toxicity judicious use is warranted. Methemoglobinemia has been reported to occur with benzocaine spray and other local anesthetics. This may occur when the ferrous ion (Fe^{+2}) of the hemoglobin molecule is converted into the ferric ion (Fe^{+3}), thereby resulting in decreased oxygen-carrying capacity. If this occurs, treatment includes supplemental oxygen and intravenous methylene blue.

Glycopyrrolate (Robinul) is a synthetic quaternary amine with muscarinic anticholinergic and antisialagogue properties. The expected sympathetic reflex that occurs during the induction of general anesthesia may result in copious secretions in the oropharynx, thereby obscuring laryngoscopy and making a successful intubation difficult. Secretions can be managed with an antisialagogue (e.g., glycopyrrolate), which will also improve the oropharyngeal surgical field. However, glycopyrrolate will tend to increase heart rate and blood pressure as a result of its anticholinergic effects. This may need to be attenuated with appropriate beta-blocker or vasodilator therapy.

A commonly used protocol for the preintubation preparation of the nasopharyngeal airway includes the following:

- Pretreatment with the judicious use of a topical vasoconstrictor (e.g., oxymetazoline, Neo-Synephrine) to shrink the nasal mucosa
- Pretreatment with a topical anesthetic, including topical lidocaine jelly
- Sequential dilation with lubricated nasopharyngeal airways (i.e., the nasal trumpets) to ease the passage of the nasotracheal tube

Strategy for Extubation after Orthognathic Surgery

After orthognathic surgery, all patients should be considered to have a difficult airway at the time of extubation. An extubation strategy is a logical extension of the planned intubation strategy for the patient with a difficult airway. The extubation strategy should be specific to the patient, and it should take into account preoperative anatomic factors; preexisting medical conditions; intraoperative findings; patient-specific swelling and intra-oral bleeding; and the patient's response to the anesthesia techniques that were used.[37,43,62,124,257,278]

Factors to consider include the following:

- Baseline preoperative obstructive sleep apnea
- Baseline limited neck range of motion
- Excessive sedation with the inadequate return of reflexes
- Continued intranasal or intraoral hemorrhage with pooling in the pharynx, which may go unrecognized and cannot be easily suctioned
- Excessive swelling within the intranasal, intraoral, and pharyngeal components of the upper airway
- Laryngeal swelling or spasm
- The potential for immediate postextubation vomiting
- The patient-specific presence of in-place intermaxillary wires, elastics, or intra-oral splints

The formulated extubation strategy should include the following:

- Strong consideration of the advantages of awake extubation versus extubation before the return of consciousness
- The formulation of an airway management plan to be implemented if the patient is not able to maintain adequate ventilation after extubation
- An evaluation of the general and patient specific clinical factors that may result in difficulty with mask ventilation after extubation
- Consideration for the short-term maintenance of a device that can serve as a guide for expedited reintubation. A semi-rigid airway device called a *tube exchanger* can be inserted through the lumen of the nasotracheal tube and into the trachea before the nasotracheal tube is removed. This device can then be used to facilitate the reinsertion of the nasotracheal tube should reintubation be required. It also allows for the limited ability to both oxygenate and ventilate the patient during the performance of reintubation maneuvers.
- A plan for percutaneous intubation (i.e., cricothyroidotomy or tracheostomy) with available equipment at the location of patient extubation (i.e., operating room, recovery room, or intensive care unit) with the skilled personnel required to complete the procedure, if required
- Discussion between surgeon and the anesthesiologist regarding patient-specific limitations in mouth opening (e.g., intermaxillary wires, elastics, masticatory muscle spasms, intra-oral splints)

Extubation Criteria for the Orthognathic Patient

With the administration of neuromuscular blocking agents as well as multiple combinations of various classes of drugs including opioids, barbiturates, and benzodiazepines, the modern hypothesis of general anesthesia has evolved to be understood as a process that requires a state of unconsciousness of the brain as well as a blockade of noxious stimuli, both centrally and peripherally. Recognition of the stage or plane of anesthesia by the clinician is important at all times

but especially at the end of the operation, when the patient must be recovered in a safe, efficient manner.

There are four classically described stages of anesthesia that are based on physical signs (e.g., respiratory status, hemodynamic status, ocular signs, muscle tone) that demonstrate the depth of anesthesia.

- Stage I is present when there is analgesia and amnesia but no loss of consciousness.
- Stage II is the period after a loss of consciousness at which point the patient's reflexes are still present, the breathing pattern is irregular, and there may be excitability. The pupils will be dilated, and there will be a disconjugate gaze.
- Stage III is considered surgical anesthesia, when there will be lack of response to surgical stimulation. Loss of the eyelash reflex is a reliable indicator of this stage. Within Stage III, there are additional planes or levels of consciousness that correspond with the amount of anesthetic delivered. The plane of anesthesia that is desired depends on the surgery being performed.
- Stage IV describes cardiovascular and respiratory failure as a result of the overadministration of anesthetic, which results in brain stem and medullary depression. This is potentially fatal.

When the need for surgical anesthesia is over, the inhalational and intravenous agents are stopped, and the patient will emerge from Stage III anesthesia (i.e., the surgical plane of anesthesia). It is important to support the patient through Stage II and into Stage I, which is when he or she can generally be safely extubated.

Potential pitfalls of the premature extubation of any patient include laryngospasm, excessive secretions or bleeding leading to airway compromise or aspiration, and inadequate airway drive as a result of residual anesthetic agents or narcotics, with the resultant need for reintubation. *Patients who are undergoing orthognathic surgery have additional and unique immediate postextubation airway concerns.* **Consequently, these patients need to meet more stringent criteria for extubation than would be anticipated for a non-intranasal or non-intraoral procedure.** Mouth opening will be limited as a result of the presence of surgical trauma and swelling of the muscles of mastication. Guiding elastics to secure the jaws together and an intra-oral (occlusal) splint are likely to be in place. Concerns about osteotomy stability may limit the forces that can be safely applied on the jaws should either mask ventilation or reintubation be required. Partial obstruction of the nasopharynx as a result of varied degrees of mucous, fresh blood, and clotting is to be expected. For all of these reasons, in the event of an airway emergency after extubation, the orthognathic patient will be difficult to mask ventilate, and there will be only limited time to consider an alternative approach to resecure the airway. The option of blind nasotracheal intubation will be even more difficult than it would have been before surgery. Orotracheal intubation will require the removal of elastics and be difficult at best. The inability to adequately mask or reintubate the patient either orally or nasally will necessitate an emergency cricothyroidotomy or tracheotomy.

Negative pressure pulmonary edema is an uncommon complication that occurs during the extubation of the trachea in approximately 0.1% of cases.[29] It is believed to be a result of laryngospasm. Upper airway obstruction from glottis closure leads to marked inspiratory efforts, which generates negative intrathoracic pressure. This may cause negative-pressure pulmonary edema and, rarely, hemoptysis. Immediately after extubation, the patient will be noted to have marked respiratory distress followed by edema fluid and decreased arterial oxygen saturation, with rising partial pressure of CO_2 levels. A chest radiograph will likely show bilateral pulmonary infiltrates. The most effective form of management is reintubation followed by positive end-expiratory pressure for 12 to 24 hours.

Before the Extubation of the Orthognathic Patient

- Any deliberate hypotensive techniques should be ceased with a return to preoperative hemodynamic stability.
- Any hypothermia should be addressed with rewarming techniques, including temperature-controlled intravenous fluids and warming blankets.
- The patient's breathing pattern should be regular, with adequate tidal volume (i.e., 6 to 10 mL/kg) and rate of respiration (i.e., 10 to 20 breaths/min).

If the patient was chemically paralyzed, this must be reversed with appropriate agents, (e.g. neostigmine and an anticholinergic agent such as glycopyrrolate). Reversal should be monitored with a twitch monitor, which consists of two electrodes placed at a motor nerve. The ulnar nerve is commonly stimulated to observe the contraction of the adductor pollicis muscle. The return of train-of-four twitches with a ratio of more than 0.9 and with sustained tetany is a method that is used to provide information about the depth of neuromuscular blockade throughout the case and through its reversal at the end. It also allows for the differentiation of depolarizing and nondepolarizing neuromuscular blockade.

- If bispectral index monitoring is used to assess the depth of anesthesia, a return to preoperative levels should be documented.
- Adequate analgesia is maintained with appropriate opioids, but titration with maintenance to a physiologic respiratory rate and tidal volume is required.
- The patient should be sufficiently awake and should have spontaneous eye opening with a conjugate gaze.
- The patient should be able to follow commands (e.g., sustained head lift and hand grip).
- A nasopharyngeal airway (i.e., a nasal trumpet) may be placed to assist with ventilation after extubation.

Checklist of Benchmark Airway Parameters Before Extubation

1. Spontaneously ventilating
2. Paralysis reversed adequately (i.e., sustained tetany without fade for >5 sec)
3. Vital signs stable
4. No longer in Stage II anesthesia
5. Negative inspiratory force of <–20 mm Hg (i.e., you can take off the bag, cover the hole with your hand, and have the patient suck in while you are watching the pressure gauge)
6. "Leak test" for the evaluation of pharyngeal and laryngeal edema
7. Benchmark respiratory parameters:
   - Tidal volume >6 cc/kg
   - Vital capacity >15 cc/kg
   - Blood oxygen saturation >90%
   - Respiratory rate <25 breaths/min
   - $paCO_2$ <45 mmHg

In-Hospital Convalescence After Orthognathic Surgery

After extubation, initial management takes place in a recovery room setting. Transfer to a monitored bed either in an intensive care unit or a step-down or telemetry unit is generally preferred. Continuous awareness of the potential for impending respiratory compromise and the need for monitoring to confirm hemodynamic stability is essential early after extubation.[263]

Airway Needs

Respiratory issues are of immediate postoperative concern in the orthognathic surgery patient.* The individual must be able to maintain his or her airway as documented by the ability to adequately oxygenate and ventilate. Multiple factors may compromise the airway, including limitations in *nasal breathing* as a result of the following: 1) the Le Fort I down-fracture with tears in the nasal mucosa; 2) specific intranasal procedures that were carried out (i.e., septoplasty, inferior turbinate reduction); and 3) edema after nasotracheal tube removal. The *oral airway* will be partially obstructed by the following: 1) ongoing bleeding, secretions, and swelling; 2) the use of guiding elastics; and 3) the presence of an acrylic intra-oral occlusal splint secured to the maxillary dentition. A nasopharyngeal trumpet may be helpful to improve the nasal airway early after extubation. The guiding elastics should be removed, if needed, to improve the oral airway. Active and frequent bedside suctioning is carried out. Supplemental oxygen given via a face tent provides an additional reserve through the nose and mouth; it is titrated to maintain pulse oximetry (i.e., oxygen saturation) of more than 95%, as described later in this chapter.

*References 17, 30, 47, 52, 72, 109, 112, 137, 141, 148, 153, 176, 183, 192, 194, 199, 246, 268, 271, 275.

Monitoring Equipment

Continuous pulse oximetry is essential. This allows for the beat-to-beat monitoring of hemoglobin saturation. It uses a pair of light-emitting diodes to measure the amount of hemoglobin in the oxyhemoglobin formation as a percentage. Normal oxygen saturation ranges from 97% to 100%. It is important to remember that pulse oximetry is not a measure of ventilation but rather of oxygenation. Factors that can result in errors of the "pulse ox" reading include hypoperfusion of the extremity being used for monitoring, high central venous pressures, and carboxyhemoglobin with carbon monoxide poisoning or methemoglobinemia. For these reasons, pulse oximetry does not replace arterial blood gas sampling for a precise measurement of arterial oxygenation. Neither technique is a substitute for a direct physical examination. Recently, technology advances have allowed for the monitoring of both pulse oximetry and capnography in one device. This is done with the use of microstream capnography via bedside nasal prongs along with pulse oximetry. These modalities together improve the ability to assess the adequacy of ventilation and oxygenation during the perioperative period.

Five-lead electrocardiography allows for the monitoring of the cardiac waveform. The most commonly used leads are II and V5 for diagnosing cardiac arrhythmias and cardiac ischemia.

Noninvasive blood pressure monitoring should be used even when an arterial line is also present postoperatively. The continuous invasive arterial blood pressure monitor may be maintained for 12 to 24 hours postoperatively. This will assist with the monitoring of the patient's arterial blood pressure in a beat-to-beat manner. It may allow for the improved accuracy of diagnosis and better direct the treatment of hypovolemia and hypotension. The arterial line can also be used to draw blood for postoperative laboratory values instead of subjecting the patient to a fresh needlestick.

Whenever there is use of DHA as well as a risk of postoperative bleeding, the maintenance of the intraoperatively placed *Foley catheter* to monitor urine output soon after surgery is useful. A catheter may be helpful to assess the success of fluid resuscitation and for patient comfort. As a baseline, normal hourly urine output for an adult is 0.5 mL/kg/hr; for a child, it is 1 mL/kg/hr.

Measuring the patient's *temperature* both intraoperatively and postoperatively is important. Temperature elevation is a late sign of malignant hyperthermia, which is very rare (i.e., 1 in 50,000 for adults). More commonly, through temperature monitoring, the patient is found to be hypothermic secondary to peripheral vasodilation caused by inhaled anesthetic gases and exposed extremities. Hypothermia can cause the patient to become coagulopathic and to

develop arrhythmias and disorders of the metabolism. Core body temperature can be measured via a temperature-sensing Foley catheter. For this modality, the Foley catheter houses a thermistor at the tip that allows for continuous core-temperature monitoring in a safe and minimally invasive manner.

Sequential compression devices placed on the lower extremities are used intraoperatively and maintained during the postoperative period to limit the formation of deep venous thrombosis. These devices increase the velocity of venous blood return to the heart, thereby decreasing venous stasis. In addition, the mechanical compression results in increased fibrinolytic activity within the vascular system. Stockings should be maintained while the patient is immobilized. On postoperative day 1, the patient should be ambulated with assistance, at which point the compression stockings may be discontinued. Multiple studies have documented that early ambulation improves recovery and results in a decreased risk of deep venous thrombosis, the return of bowel function, and earlier hospital discharge.[6]

Wound Care

Intraoral hygiene with Peridex (chlorhexidine 0.12%) and toothbrushing are performed three to five times daily. This is to decrease the bacterial load and the levels of biofilm in the oral cavity. This is especially important, because there are orthodontic brackets, elastics, splints, and exposed sutures in place near and at the incisions. An active oral hygiene routine is initiated the night after surgery by a health care professional. The patient and his or her family are specifically instructed in the techniques by the day after surgery.

In general, there are no external bandages after orthognathic surgery. The patient is encouraged to shower and shampoo his or her hair and to wash his or her face the day after surgery and at least daily thereafter.

Diet

Initial hydration, electrolyte balance, and nutrition are maintained though intravenous fluids. The night of surgery, oral intake is initiated. The orthognathic patient must tolerate liquids for adequate nutrition and hydration before discharge. Inadequate liquid intake may occasionally result in dehydration and the need for readmission after discharge. Caloric requirements in the healthy, non-hospitalized, and non-stressed individual is 15 to 20 Kcal/kg/day. The caloric need will increase substantially in the postsurgical patient, who is, by definition, under stress. A general guideline of caloric need in the patient after orthognathic surgery is 25 Kcal/kg/day. In addition, protein requirements can be doubled from 1 gm/kg of ideal body weight per day to 2 gm/kg of ideal body weight per day.

Surgical trauma to the lips and cheeks initially results in the diminished ability to achieve full lip seal. Without sufficient lip seal, the ease with which one can swallow liquids or drink from a cup is limited (see Chapter 8). A feeding syringe with a soft wide bore tip is initially used. It is placed in the vestibule between the cheek and the posterior teeth to facilitate the direction and depth of entry of liquids. The fluid bolus is injected, and the patient then seals the lips and swallows. The clear liquid diet is rapidly advanced to full liquids to achieve the needed caloric and nutritional intake. The patient transitions from syringe feeding to the use of a "sippy cup" or a squeezable water bottle and then to drinking from a soft glass as soon as he or she is able.

Nausea and Vomiting Management

Postoperative nausea and vomiting are common anesthetic concerns for the patient. If these conditions are not managed, they may in rare cases result in aspiration.[236,256] Steps taken to decrease their likelihood include gastric suctioning at the end of the procedure before extubation and the use of prophylactic medications. All orthognathic patients are given prophylactic antiemetic therapy. A medication regimen may include the following: glucocorticoids such as dexamethasone at the onset of surgery; ondansetron (Zofran) or granisetron (Kytril) 15 minutes before the conclusion of surgery; metoclopramide (Reglan); and the judicious intake of liquids immediately after extubation. Multimodal antiemetic therapy in the recovery room should be employed for refractory cases of nausea and vomiting; this may include promethazine, compazine, and repeat doses of ondansetron-like agents. Appropriate postoperative patient monitoring and bedside access to a mouth-suctioning device are essential. As long as the patient can maintain protective airway reflexes, episodes of vomiting (with only liquids on the stomach) should have minimal serious sequelae.

Intravenous Fluid Therapy

The preoperative number of hours of "nothing-by-mouth" status, the individual's body weight, and the intraoperative fluids given and lost need to be quantified. This process allows for a precise representation of the patient's fluid balance and ongoing fluid and electrolyte needs. The intravenous fluid needs must account for both sensible and insensible losses. Once maintenance fluids are calculated, a determination of estimated fluid deficit (EFD) should be made. Intraoperative third space loss and blood loss will also be taken into account. Fluid administration needs to be titrated to maintain hydration and desired urine output.

A starting point to estimate maintenance fluid needs after surgery can be given with the use of the following formula:

- 4 mL/kg/hr for the first 10 kg of body weight
- 2 mL/kg/hr for the next 10 kg of body weight
- 1 mL/kg/hr for every 1 kg of body weight above 20 kg

Pain Management

The postoperative orthognathic patient has pain requirements that are similar to those of any other surgical patient. Analgesia is required for the progression of the patient to recovery. Initial postoperative requirements are best maintained with intravenous medication, whether it is administered by the nursing staff or patient controlled. Multiple studies have confirmed that patient-controlled analgesia is safe, effective, and not likely to result in oversedation or respiratory compromise. Intravenous morphine sulfate is routinely used. By the morning after surgery, the patient is transitioned to a liquid form of opioid that taken at 4- to 6-hour intervals as needed for analgesia. It would be unusual for the patient to require or request opioid pain control by the second week after surgery. The transition from intravenous analgesics to an oral form is essential for the patient's discharge and his or her comfortable transition to home care.

Antibiotics

Multiple studies have confirmed the benefit of prophylactic doses of antibiotics for clean-contaminated (Class II) surgery and specifically for orthognathic surgery to decrease wound infection rates. This is typically initiated as a prophylactic dose of the first-generation cephalosporin cefazolin (Ancef); 15 to 20 mg/kg and up to 1 g is given before surgical incision and then every 6 hours. The postoperative course of intravenous antibiotics continues while the patient is still in the hospital. Antibiotics are then given orally upon discharge for a total of no more than a 5-day course. Opinions vary with regard to the extent of use of antibiotics for orthognathic surgery. Bentley and colleagues performed a randomized controlled trial that compared a 1-day regimen with a 5-day regimen of antibiotics for patients who were undergoing orthognathic surgery.[24] A group of 30 patients who were undergoing combined maxillary and mandibular osteotomies was divided equally. Nine out of 15 patients in the 1-day antibiotic group developed postoperative infection, whereas only 1 out of 15 patients in the 5-day antibiotic group did. The authors concluded that a 5-day course of antibiotics is useful to decrease the risk of postoperative infection (see Chapter 16).

Nasal Medications

The use of specific nasal medications after surgery varies by surgeon preference. Many surgeons recommend that, after surgery, several days of an intranasal decongestant be given to open the nasal airway and improve patient comfort. These can include Neo-Synephrine nasal spray, oxymetazoline (Afrin) nasal spray, or a corticosteroid such as Rhinocort. In addition, saline nasal spray improves the humidification of the nasal passages and limits the drying of the nasal mucosa, which may decrease the risk of epistaxis and improve patient comfort. A pure nasal saline spray has the advantage of not having a rebound effect as compared with the pharmacologic agents. This is the requirement that I prefer.

Criteria for Discharge from Hospital

Discharge planning for the orthognathic patient should begin before surgery. There should be ample preoperative communication with the patient and the patient's family or significant others to prepare for the anticipated postoperative care requirements, including medications, diet, oral and body hygiene needs, and activity level. Basic criteria for discharge from the hospital include the following:

- The ability to ingest sufficient liquids orally (i.e., hydration and nutrition)
- The absence of significant fever
- Stable vital signs, including the maintenance of pulse-oximetry documented oxygenation while sleeping
- The absence of continued nausea and vomiting
- Sufficient ambulation to carry out necessary activities of daily living in conjunction with a designated family member, friend, or caregiver
- Adequate pain control with oral analgesia while in the hospital
- The demonstrated ability to achieve required body and oral hygiene

At-Home Convalescence and Outpatient Care After Orthognathic Surgery

When feasible, our preferred outpatient regimen involves weekly office visits for 5 weeks after orthognathic surgery. Although the frequency and duration of routine postoperative office visits will vary from surgeon to surgeon, there is no substitute for a face-to-face discussion and a hands-on examination with the patient to clarify expected progress, to identify potential problems, and to institute timely treatment during the early phases of healing.

Important aspects to monitor early after orthognathic surgery include the following: 1) diet, hydration, and nutrition; 2) oral and body hygiene; 3) physical activity level; 4) emotional well-being; 5) medication use; 6) swelling and wound healing; 7) occlusion and jaw mobility; and 8) adequacy of the oral and nasal airways. The need for physical therapy related to A) lip seal and control exercises, B) sensory retraining exercises, and C) mouth-opening exercises is also assessed. Each of these parameters is addressed during each postoperative office visit. When weekly visits with the surgeon are not geographically convenient, an appropriate clinician closer to home (i.e., an orthodontist and surgeon) can generally be found. This is best supplemented with a phone call and the viewing of e-mailed facial photos sent to the primary surgeon.

Patient Education Materials

Patient Instructions for Orthognathic Surgery

The purpose of these basic instructions is to help you prepare and then recover from your operation with as little discomfort and inconvenience as possible.

Preoperative Instructions

1. During the 2 to 6 weeks before your planned operation, you will return to my office to have current dental and facial records taken; to undergo further head and neck examination; and to allow for the completion of analytic model planning.
2. During the 2 to 6 weeks before your surgery, make arrangements for a physical examination by your family physician that includes necessary laboratory tests to confirm your health. We will provide you with a form for your general physician to complete.
3. Do not take any aspirin, aspirin-like products, or any other medications that may increase bleeding for at least 3 weeks before surgery. Aspirin and aspirin-like products tend to increase bleeding during surgery and bruising postoperatively.
4. If you take or have taken a bisphosphonate medication (e.g., for osteoporosis or osteopenia therapy), you must let us know, because your risk for bone-healing difficulties may be increased.
5. Be sure that you do not smoke and that there are no nicotine products in your bloodstream for at least 3 weeks before and 2 weeks after surgery. Nicotine in the bloodstream jeopardizes wound healing. Smoke that is chronically ingested by the lungs may result in pulmonary complications that will hinder optimal healing.
6. Starting 2 days before surgery, we request that you shampoo your hair and shower at least once per day. The morning of surgery, you may also shower and shampoo your hair again.
7. Do not eat or drink anything after midnight the night before surgery, not even a sip of water. You may brush your teeth, but do not swallow the water.

Postoperative Instructions

1. In general, you will remain in the hospital for 1 or 2 nights during your initial recovery. When you are up and around, drinking enough fluids, and have recovered some of your strength, you will be able to return home to the care of your family.
2. Instructions for a high-protein blenderized (pureed) diet will be discussed with you. In general, the pureed (blenderized) diet will continue for 5 weeks, after which you will gradually return to the eating of regular foods.
3. Exercising your lips (e.g., squeezing them together for closure) during the first several days after surgery is helpful. By doing so, you will more rapidly regain lip control, reduce facial swelling, and find it easier to swallow fluids and control your saliva.
4. You will begin brushing your teeth with a soft toothbrush the day of the operation. We will prescribe a special antimicrobial mouth rinse to reduce the plaque (bacteria) buildup on your teeth.
5. In general, no facial bandages will be used after surgery. You should take a shower and wash your hair and face the day after surgery. Initially, you should have someone close by when you shower, because you may feel unsteady on your feet.
6. It is advisable to sleep with your head in an elevated position (e.g., on two pillows) for at least the first week after surgery. This helps with the natural drainage of fluids in the facial area, and it also helps to decrease swelling.
7. Nasal congestion (i.e., difficulty breathing through the nose) is to be expected immediately after surgery. The congestion generally takes 5 to 7 days to significantly clear. Until then, frequent steamy showers and the use of saline nasal sprays will be helpful. You should avoid blowing your nose after surgery, because doing so may increase the risk of bleeding. When you sneeze, let the air go through your mouth to limit the pressure in your nose.

8. When you get home, you should take the prescribed pain medication as needed. Tylenol may be substituted when your pain has diminished. Avoid taking aspirin or aspirin-like products for an additional 2 weeks after surgery.
9. Your sleep schedule will be irregular during the initial weeks after surgery. You may substitute Benadryl for the pain medication to assist with sleeping, but only after permission is received from your surgeon.
10. You will be given a prescription for an antibiotic. Take it as directed until the supply is exhausted. If you develop a skin rash, diarrhea, or a vaginal infection, stop the medication and contact your surgeon, because these problems may be related to your prescription.
11. Do not drink any beverages that contain alcohol, take any non-prescribed medications, or drive a motorized vehicle until you have been given permission to do so.

Additional Considerations

1. A cold pack placed over the cheek and neck region on each side of the face is useful during the initial 24 to 36 hours after surgery to limit swelling. Nevertheless, you will be swollen, and your skin will be discolored (i.e., black, blue, green, and yellow) for several weeks after surgery. The swelling and discoloration are never the same on the two sides of the face and neck, and they will be seen halfway down your chest. Remember that each side of the face heals somewhat independently. The initial soft-tissue swelling will not influence the final results. Although the swelling decreases significantly by 5 weeks after surgery, 6 to 12 months is required for all swelling to subside.
2. There will be numbness or decreased sensation in specific regions of your mouth and face. This takes some time to get used to, and it will improve considerably. The location of the diminished sensibility of the skin of the face and the mucous membranes of the mouth will be dependent on the specific procedures that are carried out. I will explain this to you in detail at your first consultation and again during office visits before your operation; this information will also be found on your informed consent document.
3. In general, at 5 weeks after surgery, basic healing is sufficient enough for you to gradually return to physical activities such as jogging, bicycling, and aerobics. At this stage, progression to a more regular (chewing) diet is also encouraged.

Physical Therapy after Orthognathic Surgery

1. Early after orthognathic surgery the loss of full lip control/seal results in drooling and difficulty swallowing a bolus of liquid. The lip-control difficulty results from a combination of factors, including A) surgical trauma to the soft tissues of the lips; B) the expected diminished sensation; C) the change in the position of the jaws and teeth; and D) the presence of an intra-oral occlusal splint, elastics and orthodontic lugs. Instituting active lip closure and control exercises beginning the day after surgery will help you to regain lip-seal control for ease of swallowing and managing your saliva.
2. Within the first few weeks after surgery, instructions will be given to help you to achieve the gradual return of normal mouth opening. If the adequate return of mouth opening, speech articulation, and lip control are slow in coming, then the institution of active range-of-motion mouth exercises and speech therapy will be arranged.
3. Some individuals have more difficulty than others adjusting to the expected loss of sensation after a Le Fort I maxillary osteotomy (involving the infra-orbital nerves) or sagittal split ramus osteotomies of the mandible (involving the inferior alveolar nerves). The institution of sensory retraining exercises may be helpful to desensitize you to the altered sensibility and the expected gradual recovery in specific facial and oral locations.

Jeffrey C. Posnick, DMD, MD
Director, Posnick Center for Facial Plastic Surgery

Physical Therapy after Orthognathic Surgery

- Lip Closure/Control Exercises
- Mouth-Opening Exercises
- Sensory Retraining Exercises

LIP CLOSURE/CONTROL EXERCISES

Patient Instructions (to begin the day after surgery)

Keep in mind that many patients with jaw deformities (e.g., long face growth pattern) have never been able to easily obtain full lip seal. Beginning the day of surgery, we encourage active lip closure/control exercises. Initially, the exercises are carried out by the patient under the direction of a health care professional. The patient should look at his or her lips with the use of a handheld mirror. Attempting to squeeze the lips together while viewing them in a mirror generally provides for a successful lip seal within 12 to 24 hours. Until lip seal can be achieved, it is difficult to effectively swallow liquids without drooling a portion of the liquid out of the mouth. After a consistent lip seal is achieved, the ability to swallow saliva and to drink sufficient quantities of liquids for the maintenance of hydration and adequate caloric intake is the norm. With diligent effort, the required level of lip control is generally achievable by 24 to 48 hours after surgery.

MOUTH-OPENING EXERCISES (MODIFIED UNIVERSITY OF NORTH CAROLINA INSTRUCTIONS)

Level I Patient Instructions (2 to 5 weeks after surgery)

Basic Mouth-Opening Exercises

Do these mouth-opening exercises at least three times a day for 2 to 3 minutes each time: once in the morning, once at midday, and again in the evening.

1. First, remove the elastics, and then practice opening your mouth and moving your jaw side to side by just using your jaw muscles.
2. Do not try to open or move beyond what causes only mild discomfort for a short period of time. Do not force the opening if it hurts.
3. Do not do the mouth-opening exercises with your elastics on. Avoid forcefully opening your mouth by straining against the rubber bands that hold your jaws together.

By the end of the third week, you may be able to open your mouth wide enough to get one finger between your front teeth when the elastics are off. By the end of the fifth week, you may be able to get two fingers between your teeth when your elastics are off.

Hold-and-Relax Exercises

If you cannot open your mouth wide enough to get one finger between your front teeth at the end of the third week, start these hold-and-relax exercises.

1. Open your mouth about half of the amount that is comfortable.
2. Fold your hand into a fist, and place your fist under your lower jaw.
3. Using gentle pressure, push up with your fist as if you are trying to close your mouth.
4. Use your jaw muscles to try and open your mouth against the pressure of your fist. Hold this pressure for about 5 seconds, and then remove your fist and close your mouth.
5. Next, spend 30 to 60 seconds moving your jaw side to side and gently stretching and opening your mouth just using your jaw muscles.
6. Repeat the hold-and-relax exercise followed by moving the jaw side to side about five times.

Level II Patient Instructions (5 to 9 weeks after surgery)

Hold-and-Relax Exercise

Do two or three repetitions of the hold-and-relax exercise three times a day: in the morning, at midday, and in the evening. (See the Level I instructions for this exercise.)

Finger-Stretch Exercise

1. Place your middle finger on your lower teeth in the bicuspid region, and place your thumb on your upper teeth in the bicuspid region.
2. Apply pressure with your fingers so that it creates the feeling that you are stretching the jaw muscles. There will be some feeling of tightness or mild discomfort. Hold this for 5 seconds, and then release your fingers and close your mouth.

3. Next, move your jaw side to side and open and close your mouth using your jaw muscles for 30 to 60 seconds.
4. Repeat the finger-stretch exercise but with your fingers on just one side of your mouth at a time. Follow this with the side-to-side exercise.

Do the finger-stretch exercise three times on each side. Each time, try to slightly increase the finger pressure to produce more opening. Producing a feeling of mild discomfort is okay, but do not push enough to create pain.

SENSORY RETRAINING EXERCISES (MODIFIED UNIVERSITY OF NORTH CAROLINA INSTRUCTIONS)

Level I Patient Instructions (2 to 5 weeks after surgery)

Basic Touch Exercises

Do the touch exercises with a brush (i.e., a toothbrush, hairbrush, or makeup brush) at least three times a day for 2 to 3 minutes each time: in the morning, at midday, and in the evening.

1. Start by looking in the mirror, and watch what you are doing with the brush.
2. First, alternate touching and stroking your face with the brush.
3. When you start the exercise, make long strokes that extend from one side of the face to the other.
4. As you do the exercises, vary where you first touch your face before starting the stroke. Do this on different parts of your face (i.e., forehead, cheeks, chin, and upper and lower lip).
5. The brush should be moved at the same rate that you might use your fingertips to touch or examine a piece of cloth.
6. Be sure to move the brush around on both sides of your face. Do not spend all of your time on any one spot.

Additional "Touch" Exercises

Close your eyes, and either touch or stroke your face with the brush. Tell yourself whether you are going to touch or stroke your face with the brush before you do it. If you have someone at home to help you with the exercises, ask him or her to either touch or stroke your face with the brush.

For the first five or so tries, be sure that your partner tells you before the brush touches your face whether it will be a touch or a stroke.

Try keeping your eyes closed, and then let your partner either touch or stroke your face with the brush without telling you what to expect.

If you can easily tell a touch from a stroke, that's great. Remember to decrease the length of the stroke about a quarter of an inch every other day; this will make the task more difficult.

The length of the stroke should result in it being challenging (but not impossible) for you to tell the difference between a touch and a stroke. Remember also to use both side-to-side strokes and up-and-down strokes. If you have difficulty even with long strokes, do not become frustrated. Most individuals sense little change from day to day. It is important that you be patient and persistent.

You can practice the touch exercises as many times during the day as you wish. Looking in the mirror while you practice is very important. If you do not have a brush with you, simply use a light finger touch.

Level II Patient Instructions (5 to 9 weeks after surgery)

Basic Touch Exercises

Do the touch exercises with the brush at least three times a day for 2 to 3 minutes each time: in the morning, at midday, and in the evening. When you begin, look in the mirror and watch the movement.

1. At first, alternate stroking your face side to side or up and down.
2. As you do the exercises, vary where you make the strokes on your face.
3. When you begin the exercises, start the side-to-side exercise on one side of the face, and stroke across the entire region to the other side of the face. For example, move from the bottom of your ear across your upper lip to the bottom of your other ear.
4. When you stroke up and down, start on your neck, and go all the way up and down your face. For example, move from your neck all the way up the side of your face to the forehead, or start just below your eye and stroke down to your neck.
5. While doing the exercise, tell yourself that whatever sensation you feel is normal and expected.

Continued

6. Seeing the orientation of the stroke and the area of the face that you are touching reflected in the mirror and telling yourself that all of the sensations you feel are normal and expected is important.
7. Be sure to move the brush around on both sides of your face. Do not spend all your time on any one spot.

Additional Touch Exercises

Close your eyes. Before you stroke your face with the brush, tell yourself whether you are going to stroke your face side to side or up and down with the brush. If you have someone at home to help you with the exercises, for the first five or so tries, be sure your partner tells you before the brush touches your face whether it will be a side-to-side or up-and-down stroke. Next, try keeping your eyes closed, and let your partner do the exercise again without telling you what to expect.

If you can easily tell an up-and-down stroke from a side-to-side stroke, that's great! Remember to decrease the length of the stroke by about a quarter of an inch every other day; this will make the task more difficult. The length of the stroke should result in it being challenging (but not impossible) for you to tell the difference between an up-and-down stroke and a side-to-side stroke.

If you have difficulty even with long strokes, do not become frustrated. Most patients sense little change from day to day. It is important that you be patient and persistent.

You can practice the touch exercises as many times during the day as you wish. Looking in the mirror while you practice is very important. If you do not have a brush with you, simply use a light finger touch.

Do the touch exercises with the brush at least three times a day for 2 to 3 minutes each time: in the morning, at midday, and in the evening. When you begin, look in the mirror and watch the movement.

Jeffrey C. Posnick, DMD, MD
Director, Posnick Center for Facial Plastic Surgery

References

1. Ablad B: A study of the mechanism of the hemodynamic effects of hydralazine in man. *Acta Pharmacol Toxicol* 20:1, 1963.
2. Acampora G, Shah N, Del Valle O, et al: Myocardial depression by esmolol during nitroprusside hypotension [abstract]. *Anesthesiology* 71:A19, 1989.
3. Adams AP: Techniques of vascular control for deliberate hypotension during anaesthesia. *Br J Anaesth* 47:777–792, 1975.
4. Agnelli G, Berrettini M, de Cunto M, et al: Desmopressin-induced improvement of abnormal coagulation in chronic liver disease [letter]. *Lancet* 1:645, 1983.
5. Ahlering TE, Henderson JB, Skinner DG: Controlled hypotensive anesthesia to reduce blood loss in radical cystectomy for bladder cancer. *J Urol* 129:953–954, 1983.
6. American Society of Anesthesiologists: Practice guidelines for management of the difficult airway. A report by the American Society of Anesthesiologists Task Force on Management of the Difficult Airway. *Anesthesiology* 78:597–602, 1993.
7. Anderson JA: Deliberate hypotensive anesthesia for orthognathic surgery: controlled pharmacologic manipulation of cardiovascular physiology. *Int J Adult Orthodon Orthognath Surg* 1:133–159, 1986.
8. Angaran DM, Schultz NJ, Tschida VH: Esmolol hydrochloride: an ultrashort-acting, B-adrenergic blocking agent. *Clin Pharm* 5:288–303, 1986.
9. Artru AA: Cerebral vascular responses to hypocapnia during nitroglycerin-induced hypotension. *Neurosurgery* 16:468–472, 1985.
10. Artru AA: Cerebral metabolism and EEG during combination of hypocapnia and isoflurane-induced hypotension in dogs. *Anesthesiology* 65:602–608, 1986.
11. Artru AA: Cerebral metabolism and the electroencephalogram during hypocapnia plus hypotension induced by sodium nitroprusside or trimethaphan in dogs. *Neurosurgery* 18:36–44, 1986.
12. Artru AA: Partial preservation of cerebral vascular responsiveness to hypocapnia during isoflurane-induced hypotension in dogs. *Anesth Analg* 65:660–666, 1986.
13. Artru AA, Colley PS: Cerebral blood flow response to hypocapnia during hypotension. *Stroke* 15:878–883, 1984.
14. Artru AA, Wright K, Colley PS: Cerebral effects of hypocapnia plus nitroglycerin-induced hypotension in dogs. *J Neurosurg* 64:924–931, 1986.
15. Ash DC, Mercuri LG: The relationship between blood ordered and blood administered in orthognathic surgery: a retrospective study. *J Oral Maxillofac Surg* 43:944, 1985.
16. Avantika N, Pogrel MA: Preoperative autologous blood donation for oral and maxillofacial surgery: an analysis of 913 patients. *J Oral Maxillofac Surg* 63:347, 2005.
17. Aziz SR, Agnihotri N, Ziccardi VB: Lobar collapse immediately after orthognathic surgery. *J Oral Maxillofac Surg* 68:2335, 2010
18. Bahlman SH, Eger El II, Halsey MJ, et al: The cardiovascular effects of halothane in man during spontaneous ventilation. *Anesthesiology* 36:494–502, 1972.
19. Beadnell SW, Saunderson JR, Sorenson DC: Compartment syndrome following oral and maxillofacial surgery. *J Oral Maxillofac Surg* 46:232, 1988
20. Bemard JM, Pinaud M, Francois T, et al: Deliberate hypotension with nicardipine or nitroprusside during total hip arthroplasty. *Anesth Analg* 73:341–345, 1991.
21. Benoni G, Fredin H: Fibrinolytic inhibition with tranexamic acid reduces blood loss and blood transfusion after knee arthroplasty: a prospective, randomized, double-blind study of 86 patients. *J Bone Joint Surg Br* 78:434, 1996.
22. Benoni G, Lethagen S, Fredin H: The effect of tranexamic acid on local and plasma fibrinolysis during total knee arthroplasty. *Thromb Res* 85:195, 1997.
23. Benoni G, Fredin H, Knebel R, et al: Blood conservation with tranexamic acid in total hip arthroplasty: a randomized, double-blind study in 40 primary operations. *Acta Orthop Scand* 72:442, 2001.

24. Bentley KC, Head TW, Aiello GA: Antibiotic prophylaxis in orthognathic surgery: A 1-day versus 5-day regimen. *J Oral Maxillofac Surg* 57:226, 1999.
25. Bergman S, Hoffman WE, Gans BJ, et al: Blood flow to oral tissues: an experimental study with enflurane, sodium nitroprusside, and nitroglycerin. *J Oral Maxillofac Surg* 40:13–17, 1982.
26. Bernard JM, Pinaud M, Maquin-Mavier I, et al: Hypotensive anesthesia with isoflurane and enflurane during total hip replacement: a comparative study of catecholamine and renin angiotensin responses. *Anesth Analg* 69:467–472, 1989.
27. Bernet F, Carrel T, Marbet G, et al: Reduction of blood loss and transfusion requirements after coronary artery bypass grafting: similar efficacy of tranexamic acid and aprotinin in aspirin-treated patients. *J Card Surg* 14:92, 1999.
28. Berntorp E, Follrud C, Lethagen S: No increased risk of venous thrombosis in women taking tranexamic acid. *Thromb Haemost* 86:714, 2001.
29. Bhavani-Shankar K, Hart NS, Mushlin PS: Negative pressure induced airway and pulmonary injury. *Can J Anaesth* 44:78–81, 1997.
30. Bigoli SJ, Dumont L, Mattys M, et al: A serious anesthetic complication of a Le Fort I osteotomy. *Eur J Anaesthesiol* 16:201, 1999.
31. Blau WS, Kafer ER, Anderson JA: Esmolol is more effective than sodium nitroprusside in reducing blood loss during orthognathic surgery. *Anesth Analg* 75:172–178, 1992.
32. Blinder D, Manor Y, Martinowitz U, et al: Dental extractions in patients maintained on continued oral anticoagulant: comparison of local hemostatic modalities. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 88:137, 1999.
33. Bourreli B, Pinaud M, Passuti N, et al: Additive effects of dihydralazine during enflurane or isoflurane hypotensive anaesthesia for spinal fusion. *Can J Anaesth* 35:242–248, 1988.
34. Brand A: Immunological aspects of blood transfusion. *Blood Rev* 14:130–144, 2000.
35. Bridgens JP, Evans CR, Dobson PM, et al: Intraoperative red blood-cell salvage in revision hip surgery. A case-matched study. *J Bone Joint Surg Am* 89:270, 2007.
36. Brittain RT, Levy GP: A review of the animal pharmacology of labetalol, a combined alpha- and beta-adrenoceptor-blocking drug. *Br J Clin Pharmacol* 3(4 Suppl 3):681–699, 1976.
37. Broccard AF, Liaudet L, Aubert JD, et al: Negative pressure post-tracheal extubation alveolar hemorrhage. *Anesth Analg* 92:273–275, 2001.
38. Bulloch SE, Fridrich KL: Capillary blood flow in the maxilla during hypotensive anesthesia for maxillary surgery. *J Oral Maxillofac Surg* 51(Suppl 3):142–143, 1993.
39. Camarasa MA, Olle G, Serra-Prat M, et al: Efficacy of aminocaproic, tranexamic acids in the control of bleeding during total knee replacement: a randomized clinical trial. *Br J Anaesth* 96:576, 2006.
40. Carswell DJ, Varkey GP, Drake CG: Labetalol for controlled hypotension in surgery for intracranial aneurysm. *Can Anaesth Soc J* 28:505, 1981.
41. Casati V: About dosage schemes and safety of tranexamic acid in cardiac surgery. *Anesthesiology* 99:236, 2003.
42. Casati V, Guzzon D, Oppizzi M, et al: Hemostatic effects of aprotinin, tranexamic acid and epsilon-aminocaproic acid in primary cardiac surgery. *Ann Thorac Surg* 68:2252, 1999.
43. Cascade PN, Alexander GD, Mackie DS: Negative-pressure pulmonary edema after endotracheal intubation. *Radiology* 186:671–675, 1993.
44. Casthely PA, Lear S, Cottrell JE, Lear E: Intrapulmonary shunting during induced hypotension. *Anesth Analg* 61:231–235, 1982.
45. Cavusoglu T, Yazici I, Demirtas Y, et al: A rare complication of nasotracheal intubation: accidental middle turbinectomy. *J Craniofac Surg* 20:566, 2009.
46. Chan W, Smith DE, Ware WH: Effects of hypotensive anesthesia in anterior maxillary surgery. *J Oral Surg* 38:504, 1980.
47. Chebel NA, Ziade D, Achkouty R: Bilateral pneumothorax and pneumomediastinum after treatment with continuous positive airway pressure after orthognathic surgery. *Br J Oral Maxillofac Surg* 48:e14, 2010.
48. Chemello PD, Nelson SR, Wolford LM: Finger injury resulting from pulse oximeter probe during orthognathic surgery. *Oral Surg Oral Med Oral Pathol* 69:161, 1990.
49. Choi SH, Lee WK, Lee KY, et al: Efficacy of remifentanil-induced controlled hypotension for orthognathic two jaw surgery. *Korean J Anesthesiol* 52(1):62–66, 2007.
50. Choi WS, Samman N: Risks and benefits of deliberate hypotension in anaesthesia: a systematic review. *Int J Oral Maxillofac Surg* 37:687–703, 2008.
51. Choi WS, Irwin MG, Samman N: The effect of tranexamic acid on blood loss during orthognathic surgery: a randomized controlled trial. *J Oral Maxillofac Surg* 67:125–133, 2009.
52. Chua W, Chidambaram A, Doyle PT, et al: Voicing concern: an unusual sequelae of orthognathic surgery. *Prog Orthod* 7:220, 2006.
53. Cohen JA, Brecher ME: Preoperative autologous blood donation: benefit or detriment? A mathematical analysis. *Transfusion* 35:640, 1995.
54. Cole PV: The safe use of sodium nitroprusside. *Anaesthesia* 33:473–477, 1978.
55. Cole PV: Sodium nitroprusside. In Hewer CL, Atkinson RS, editors: *Recent advances in anaesthesia and analgesia*, London, 1979, Churchill Livingstone, pp 139–149.
56. Consensus conference: Perioperative red blood cell transfusion. *JAMA* 260:2700, 1988.
57. Cottrell JE, Casthely PA, Brodie JD, et al: Prevention of nitroprusside-induced cyanide toxicity with hydroxocobalamin. *N Engl J Med* 298:809–811, 1978.
58. Cushing H: *Tumors of the nervus acusticus*, Philadelphia, 1917, W.B. Saunders Company.
59. Dalmau A, Sabate A, Acosta F, et al: Tranexamic acid reduces red cell transfusion better than epsilon-aminocaproic acid or placebo in liver transplantation. *Anesth Analg* 91:29, 2000.
60. Davies DW, Kadar D, Steward DJ, Munro IR: A sudden death associated with the use of sodium nitroprusside for induction of hypotension during anaesthesia. *Can Anaesth Soc J* 22:547–552, 1975.
61. Dedrick DF, Mans AM, Campbell PA, et al: Does ATP-induced hypotension cause potentially serious metabolic complications? *Anesthesiology* 57:A66, 1982.
62. Deepika K, Kenaan CA, Barrocas AM, et al: Negative pressure pulmonary edema after acute upper airway obstruction. *J Clin Anesth* 9:403–408, 1997.
63. Del Valle O, Edmondson R, Reinsel R, et al: Esmolol vs nitroprusside for hypotension: dose response during isoflurane anesthesia [abstract]. *Anesthesiology* 72:A143, 1990.
64. Dhariwal DK, Gibbons AJ, Kittur MA, et al: Blood transfusion requirements in bimaxillary osteotomies. *Br J Oral Maxillofac Surg* 42:231, 2004.
65. Diaz JH, Lockhart CH: Hypotensive anaesthesia for craniectomy in infancy. *Br J Anaesth* 51:233–235, 1979.
66. Didier EP, Clagett OT, Theye RA: Cardiac performance during controlled hypotension. *Anesth Analg* 44:379, 1965.
67. Dinmore P: Combined use of trimethaphan and sodium nitroprusside. *Br J Anaesth* 49:1070, 1977.
68. Dodson TB, Neuenschwander MC, Bays RA: Intraoperative assessment of maxillary perfusion during Le Fort I osteotomy. *J Oral Maxillofac Surg* 52:827–831, 1994.
69. Dolman RM, Bentley KC, Head TW, et al: The effects of hypotensive anesthesia on blood loss and operative time during Le Fort I osteotomies. *J Oral Maxillofac Surg* 58:834, 2000.
70. Domen RE: Adverse reactions associated with autologous blood transfusion: evaluation and incidence at a large academic hospital. *Transfusion* 38:296, 1998.
71. Dunn CJ, Goa KL: Tranexamic acid: a review of its use in surgery and other indications. *Drugs* 57:1005, 1999.
72. Eckenhoff JE, Enderby GEH, Larson A, et al: Pulmonary gas exchange during deliberate hypotension. *Br J Anaesth* 35:750–759, 1963.
73. Eckenhoff JE, Rich JC: Clinical experiences with deliberate hypotension. *Anesth Analg* 45:21, 1966.

74. Edmondson R, Del Valle O, Shah N, et al: Esmolol for potentiation of nitroprusside-induced hypotension: impact on the cardiovascular, adrenergic, and random-angiotensin systems in man. *Anesth Analg* 69:202–206, 1989.
75. Edwards DB, Scheffler RB, Jackler I: Postoperative pneumomediastinum and pneumothorax following orthognathic surgery. *J Oral Maxillofac Surg* 44:137, 1986.
76. Eerola R, Eerola M, Kaukinen L, et al: Controlled hypotension and moderate haemodilution in major hip surgery. *Ann Chir Gynaecol* 69:109, 1979.
77. Ekback G, Axelsson K, Ryttberg L, et al: Tranexamic acid reduces blood loss in total hip replacement surgery. *Anesth Analg* 91:1124, 2000.
78. Enderby GEH: Controlled circulation with hypotensive drugs and posture to reduce bleeding in surgery: preliminary results with pentamethonium iodide. *Lancet* 1:1145–1147, 1950.
79. Enderby GEH: Pentolinium tartrate in controlled hypotension. *Lancet* 2:1097–1098, 1954.
80. Enderby GEH: A report of mortality and morbidity following 9107 hypotensive anaesthetics. *Br J Anaesth* 33:109–113, 1961.
81. Enderby GEH: Some observations on the practice of deliberate hypotension. *Br J Anaesth* 47:743–744, 1975.
82. Endrich M, Franke N, Peter K, Messmer K: Induced hypotension: action of sodium nitroprusside and nitroglycerin on the microcirculation. *Anesthesiology* 66:605–613, 1987.
83. Enlund M, Mentell O, Engström, et al: Occurrence of adenylate kinase in cerebrospinal fluid after isoflurane anaesthesia and orthognathic surgery. *Ups J Med Sci* 101:97, 1996.
84. Enlund MG, Ahlstedt BL, Andersson LG, et al: Induced hypotension may influence blood loss in orthognathic surgery, but it is not crucial. *Scand J Plast Reconstr Surg Hand Surg* 31:311, 1997.
85. Eriksson O, Kjellman H, Pilbrant A, et al: Pharmacokinetics of tranexamic acid after intravenous administration to normal volunteers. *Eur J Clin Pharmacol* 7:375, 1974.
86. Etchason J, Petz L, Keeler E, et al: The cost effectiveness of preoperative autologous blood donations. *N Engl J Med* 332:719, 1995.
87. Fahmy NM: Indications and contraindications for deliberate hypotension with a review of its cardiovascular effects. *Int Anesthesiol Clin* 17:175–187, 1979.
88. Fahmy NR: Nitroglycerin as a hypotensive drug during general anesthesia. *Anesthesiology* 49:17–20, 1978.
89. Fahmy NR: Nitroprusside versus nitroprusside-trimethaphan mixture for induced hypotension: hemodynamic effects and cyanide release. *Clin Pharmacol Ther* 37:264–270, 1985.
90. Farah GJ, de Moraes M, Filho LI, et al: Induced hypotension in orthognathic surgery: a comparative study of 2 pharmacological protocols. *J Oral Maxillofac Surg* 66:2261–2269, 2008.
91. Feagan B: The efficacy, safety and acceptance of autologous blood donation as a blood conservation strategy. *Vox Sang* 83 Suppl 1:11–12, 2002.
92. Flood TR, Ilankovan V, Moos KF, El-Attar A: Cross-match requirements in orthognathic surgery: an audit. *Br J Oral Maxillofac Surg* 28:292, 1990.
93. Fong J, Gurewitsch ED, Kang HJ, et al: An analysis of transfusion practice and the role of intraoperative red blood cell salvage during cesarean delivery. *Anesth Analg* 104:666, 2007.
94. Fordyce AM, Telfer MR, Stassen LF: Cross-matched blood for major head and neck surgery: an analysis of requirements. *Br J Oral Maxillofac Surg* 36:103–106, 1998.
95. Fridrich KL: Anesthetic techniques to reduce blood loss and transfusion therapy. *Oral Maxillofac Surg Clin North Am* 4:863–864, 1992.
96. Fridrich KL: Induced hypotensive anesthesia of adolescent orthognathic surgery patients [discussion]. *J Oral Maxillofac Surg* 54:683, 1996.
97. Fromme GA, MacKenzie RA, Gould AB, et al: Controlled hypotension for orthognathic surgery. *Anesth Analg* 65:683–686, 1986.
98. Fukunaga AF, Facke WE, Bloor BC: Hypotensive effects of adenosine and adenosine triphosphate compared to sodium nitroprusside. *Anesth Analg* 61:273–278, 1982.
99. Fukunaga AF, Ikeda K, Matsuda L: ATP-induced hypotensive anesthesia during surgery. *Anesthesiology* 57:A65, 1982.
100. Gallagher DM, Milliken RA: Induced hypotension for orthognathic surgery. *J Oral Surg* 37:47–51, 1979.
101. Gallandat Huet RCG, Siemons AW, Baus D, et al: A novel hydroxyethyl starch for effective perioperative plasma volume substitution in cardiac surgery. *Can J Anesth* 47:1207, 2000.
102. Gardner WJ: The control of bleeding during operation by induced hypotension. *JAMA* 132:572–574, 1946.
103. Gibbons AJ, Dhariwal DK, Benton A, et al: Blood usage in maxillofacial surgery. *Br J Oral Maxillofac Surg* 40:350, 2002.
104. Gillon G, Greenburg AG: Transfusions: infectious complications. *Complications in Surgery* 11:19, 1992.
105. Goldman M, Savard R, Long A, et al: Declining value of preoperative autologous donation. *Transfusion* 41:819, 2002.
106. Golia JK, Woo R, Farole A, Seltzer J: Nitroglycerin-controlled circulation in orthognathic surgery. *J Oral Maxillofac Surg* 43:342–345, 1985.
107. Gong SG, Krishnan V, Waack D: Blood transfusions in bimaxillary orthognathic surgery: are they necessary? *Int J Adult Orthodon Orthognath Surg* 17:314, 2002.
108. Goodnaugh LT: Risks of blood transfusion. *Crit Care Med* 31(Suppl):678, 2003.
109. Goodson ML, Manemi R, Paterson AW: Pneumothorax after orthognathic surgery. *Br J Oral Maxillofac Surg* 48:180, 2010.
110. Goto F, Otani E, Kato S, Fujita T: Prostaglandin El as a hypotensive drug during general anaesthesia. *Anaesthesia* 37:530–535, 1982.
111. Grace AH: Prostatectomy under hypotensive anaesthesia. *Proc R Soc Med* 54:1130–1132, 1961.
112. Grime PD, Tyler C: An obstructed airway: cuff herniation during nasotracheal anesthesia for a bimaxillary osteotomy. *Br J Oral Maxillofac Surg* 29:14, 1991.
113. Gruvstad M, Kebbon L, Lof BA: Changes in mental function after induced hypotension. *Acta Psych Scand* 37(Suppl 163):1–112, 1962.
114. Guay J, Reinberg C, Poitras B, et al: A trial of desmopressin to reduce blood loss in patients undergoing spinal fusion for the idiopathic scoliosis. *Anesth Analg* 75:405, 1992.
115. Guyuron B, Vaughan C, Schlecter B: The role of DDAVP (desmopressin) in orthognathic surgery. *Ann Plast Surg* 37:516, 1996.
116. Hackmann T, Friesen M, Allen S, et al: Clonidine facilitates controlled hypotension in adolescent children. *Anesth Analg* 96:976, 2003.
117. Harp JR, Wollman H: Cerebral metabolic effects of hyperventilation and deliberate hypotension. *Br J Anaesth* 45:256–262, 1973.
118. Hegtverdt AK, Collins ML, White RP, Turvey TA: Minimizing the risk of transfusion in orthognathic surgery: use of predeposited autologous blood. *Int J Adult Orthodon Orthognath Surg* 2:185, 1987.
119. Hewitt PB, Lord PW, Thornton HL: Propranolol in hypotensive anaesthesia. *Anaesthesia* 22:82–90, 1967.
120. Hiippala ST, Strid LJ, Wennerstrand MI, et al: Tranexamic acid radically decreases blood loss and transfusions associated with total knee arthroplasty. *Anesth Analg* 84:839, 1997.
121. Hines R, Barash PG: Infusion of sodium nitroprusside induces platelet dysfunction in vitro. *Anesthesiology* 70:611–615, 1989.
122. Ho KM, Ismail H: Use of intravenous tranexamic acid to reduce allogeneic blood transfusion in total hip and knee arthroplasty: a meta-analysis. *Anaesth Intensive Care* 31:529, 2003.
123. Holdgaard HO, Pedersen J, Schurizek BA, et al: Complications and late sequelae following nasotracheal intubation. *Acta Anaesthesiol Scand* 37:475, 1993.
124. Holmes JR, Hensinger RN, Wojtys EW: Postoperative pulmonary edema in young, athletic adults. *Am J Sports Med* 19:365–371, 1991.

125. Horrow JC, van Riper DF, Strong MD, et al: Hemostatic effects of tranexamic acid and desmopressin during cardiac surgery. *Circulation* 84:2063, 1991.
126. Horrow JC, Van Riper DF, Strong MD, et al: The dose-response relationship of tranexamic acid. *Anesthesiology* 82:383, 1995.
127. Hughes C, Johnson C, Irvine G: A technique to reduce airway risk in patients undergoing maxillofacial surgery. *Anaesthesia* 56:1127, 2001.
128. Hunter AR: *Neurosurgical anaesthesia*, Oxford, 1964, Blackwell Scientific Publications, p 147.
129. Husted H, Blond L, Sonne-Holm S, et al: Tranexamic acid reduces blood loss and blood transfusions in primary total hip arthroplasty: a prospective randomized double-blind study in 40 patients. *Acta Orthop Scand* 74:665, 2003.
130. Hynes M, Calder P, Scott G: The use of tranexamic acid to reduce blood loss during total knee arthroplasty. *Knee* 10:375, 2003.
131. Ickx BE, van der Linden PJ, Melot C, et al: Comparison of the effects of aprotinin and tranexamic acid on blood loss and red blood cell transfusion requirements during the late stages of liver transplantation. *Transfusion* 46:595, 2006.
132. Ido K, Neo M, Asada Y, et al: Reduction of blood loss using tranexamic acid in total knee and hip arthroplasties. *Arch Orthop Trauma Surg* 120:518, 2000.
133. Ishikawa T, Funatsu N, Okamoto K, et al: Blood-brain barrier function following drug-induced hypotension in the dog. *Anesthesiology* 59:526–531, 1983.
134. Joint statement on acquired immune deficiency syndrome (AIDS) related to transfusion. *Transfusion* 23:87, 1983.
135. Jordan WS, Graves CL, Boyd WA, et al: Cardiovascular effects of three techniques for inducing hypotension during anesthesia. *Anesth Analg* 50:1059–1068, 1971.
136. Jovic MD, Calija BM, Radomir BJ, et al: The use of acute normovolemic hemodilution in patients undergoing cardiac surgery. *Cardiovasc Surg* 11:201, 2003.
137. Kademani D, Voiner JL, Quinn PD: Acute hypertensive crisis resulting in pulmonary edema and myocardial ischemia during orthognathic surgery. *J Oral Maxillofac Surg* 62:240, 2004.
138. Karnezis TA, Stulberg SD, Vixon RL, et al: The hemostatic effect of desmopressin on patients who had total joint arthroplasty: a double-blind randomized trial. *J Bone Joint Surg* 76:1545, 1994.
139. Kassell NF, Boarini DJ, Olin JJ, Sprowell JA: Cerebral and systemic circulatory effects of arterial hypotension induced by adenosine. *J Neurosurg* 58:69–76, 1983.
140. Keats AS: The ASA classification of physical status: a recapitulation. *Anesthesiology* 49:233, 1978.
141. Keiser GJ, Bozentka NE, Gold BD: Laryngeal granuloma: a complication of prolonged endotracheal intubation. *Anesth Prog* 38:232, 1991.
142. Kessler P, Hegewald J, Adler W, et al: Is there a need for autogenous blood donation in orthognathic surgery? *Plast Reconstr Surg* 117:571, 2006.
143. Kien ND, White DA, Reitan JA, Eisele JH, Jr: Cardiovascular function during controlled hypotension induced by adenosine triphosphate or sodium nitroprusside in the anesthetized dog. *Anesth Analg* 66:103–110, 1987.
144. Kim HJ, Kim JT, Kim HS, et al: A comparison of Glide Scope videolaryngoscopy and direct laryngoscopy for nasotracheal intubation in children. *Paediatr Anaesth* 21:417, 2011
145. Kitaguchi K, Nakajima T, Takaki O, et al: The change in cerebral blood flow during hypotensive anesthesia induced by prostaglandin El. *Masui* 41:766–771, 1992.
146. Cha KJ, Lee SC: Thermosoftening treatment of the nasotracheal tube before intubation can reduce epistaxis and nasal damage. *Anesth Analg* 91:698, 2000.
147. Kohler M, Hellestern P, Miyashita C, et al: Comparative study of intranasal, subcutaneous and intravenous administration of desamino-arginine vasoprassine (DDAVP). *Thromb Haemost* 55:108, 1986.
148. Kretschmer W, Koster U, Dietz K, et al: Factors for intraoperative blood loss in bimaxillary osteotomies. *J Oral Maxillofac Surg* 66:1399, 2008.
149. Kurian A, Ward-Booth P: Blood transfusion and orthognathic surgery—a thing of the past? *Br J Oral Maxillofac Surg* 42:369, 2004.
150. Lagerkranser M, Andreen M, Irestedt L, Sollevi A: Controlled hypotension with the combination of dipyridamole and adenosine. *Anaesthesia* 37(Suppl):303, 1982.
151. Lam AM: Induced hypotension. *Can Anaesth Soc J* 31:S56, 1984.
152. Laureano-Filho JR, Godoy F, O'Ryan F: Orthodontic bracket lost in the airway during orthognathic surgery. *Am J Orthod Dentofacial Orthop* 134:288, 2008.
153. Laureano-Filho JR, de Oliveiro Neto PJ, Duarte N, et al: Successful management of malignant hyperthermia during orthognathic surgery: a case report. *J Oral Maxillofac Surg* 66:1485, 2008.
154. Lessard MR, Trepanier CA, Baribault JP, et al: Isoflurane-induced hypotension in orthognathic surgery. *Anesth Analg* 69:379–383, 1989.
155. Litfle DM: Induced hypotension during anesthesia and surgery. *Anesthesiology* 16:320–332, 1955.
156. Lupori JP, Van Sickels JE, Holmgreen WC: Outpatient orthognathic surgery: review of 205 cases. *J Oral Maxillofac Surg* 155:558, 1997.
157. Mackie D: Hypotensive anesthesia and orthognathic surgery. *Anesth Analg* 76:667, 1993.
158. Macnab MSP, Manninen PH, Lam AM, Gelb AW: The stress response to induced hypotension for cerebral aneurysm surgery: a comparison of two hypotensive techniques. *Can J Anaesth* 35:111–115, 1988.
159. Macrae WR, Owen M: Severe metabolic acidosis following hypotension induced with sodium nitroprusside. *Br J Anaesth* 46:795–797, 1974.
160. Macrae WR, Wildsmith JAW, Dale BAB: Induced hypotension with a mixture of sodium nitroprusside and trimethaphan camsylate. *Anaesthesia* 36:312–315, 1981.
161. Madsen JB, Cold GE, Hansen ES, et al: Cerebral blood flow and metabolism during isoflurane-induced hypotension in patients subjected to surgery for cerebral aneurysms. *Br J Anaesth* 59:1204–1207, 1987.
162 Madsen DE, Ingerslev J, Sidelmann JJ, et al: Intraoperative blood loss during orthognathic surgery is predicted by thromboelastography. *J Oral Maxillofac Surg* 70:e547–e552, 2012.
163. Magness A, Yashon D, Locke G, et al: Cerebral function during trimethaphan-induced hypotension. *Neurology* 23:506–509, 1973.
164. Mallampati SR, Gatt SP, Gugino LD, et al: A clinical sign to predict difficult tracheal intubation: a prospective study. *Can Anaesth Soc J* 32(4):429–434, 1985.
165. Manola M, De Luca E, Moscillo L, Mastella A: Using remifentanil and sufentanil in functional endoscopic sinus surgery to improve surgical conditions. *ORL J Otorhinolaryngol Relat Spec* 67:83–86, 2005.
166. Marchal JM, Gomez-Luque A, Martos-Crespo F, et al: Clonidine decreases intraoperative bleeding in middle ear microsurgery. *Anesthesiol Scand* 45:627, 2001.
167. Marciani RD, Dickson LG: Autologous transfusion in orthognathic surgery. *J Oral Maxillofac Surg* 43:201, 1985.
168. McNeill TW, DeWald RL, Kuo KN, et al: Controlled hypotensive anesthesia in scoliosis surgery. *J Bone Joint Surg* 56:1167–1172, 1974.
169. McNulty S, Sharifi-Azad S, Farole A: Induced hypotension with labetalol for orthognathic surgery. *J Oral Maxillofac Surg* 45:309–311, 1987.
170. Mead JH, Anthony CD, Sattler M: Hemotherapy in elective surgery: an incidence report, review of the literature, and alternatives for guideline appraisal. *Am J Clin Pathol* 74:223, 1980.
171. Melissari E, Scully MF, Paes T, et al: The influence of DDAVP infusion on the coagulation and fibrinolytic response to surgery. *Thromb Haemost* 55:54, 1986.
172. Meoli AL, Rosen CL, Kristo D, et al: Report of the AASM clinical practice review committee. Upper airway management of the adult patient with obstructive sleep apnea in the perioperative period—avoiding complications. *Sleep* 26(8):1060–1065, 2003.

173. Messmer KF: Acceptable haematocrit levels in surgical patients. *World J Surg* 11:41–46, 1987.
174. Michenfelder JD, Theye RA: Canine systemic and cerebral effects of hypotension induced by hemorrhage, trimethaphan, halothane, or nitroprusside. *Anesthesiology* 46:188–195, 1977.
175. Moenning JE, Bussard DA, Lapp TH, et al: Average blood loss and the risk of requiring perioperative blood transfusion in 506 orthognathic surgical procedures. *J Oral Maxillofac Surg* 53:880, 1995.
176. Monaghan A, Hindle I: Malignant hyperpyrexia in oral surgery: case report and literature review. *Br J Oral Maxillofac Surg* 32:190, 1994.
177. Moor AC, Dubbleman TM, VanSteveninck J, Brand A: Transfusion-transmitted diseases: risks, prevention and perspectives. *Eur J Haematol* 62:1–18, 1999.
178. Moraca P, Bitte EM, Hale DE, et al: Clinical evaluation of sodium nitroprusside as a hypotensive agent. *Anesthesiology* 23:193–199, 1962.
179. Murphy MA: Bilateral posterior ischemic optic neuropathy after lumbar spine surgery. *Ophthalmology* 110:1454–1457, 2003.
180. Murray D: Acute normovolemic hemodilution. *Eur Spine J* 13(Suppl):S72, 2004.
181. Murtagh GP: Controlled hypotension with halothane. *Anaesthesia* 15:235–244, 1960.
182. Nakazawa K, Taneyama C, Benson KT, et al: Mixtures of sodium nitroprusside and trimethaphan for induction of hypotension. *Anesth Analg* 73:59–63, 1991.
183. Nanni V, Sachs S: Mediastinal emphysema following Le Fort I osteotomy: report of a case. *Oral Surg Oral Med Oral Pathol* 62:508, 1986.
184. Nath A, Pogrel MA: Preoperative autologous blood donation for oral and maxillofacial surgery: an analysis of 913 patients. *J Oral Maxillofac Surg* 63:347, 2005.
185. National Heart, Lung, and Blood Institute Expert Panel on the use of Autologous Blood: Transfusion alert: use of autologous blood. *Transfusion* 3:703–711, 1995.
186. Neilipovitz DT: Tranexamic acid for major spinal surgery. *Eur Spine J* 13(Suppl):S62, 2004.
187. Neilipovitz DT, Murto K, Hall L, et al: A randomised trial of tranexamic acid to reduce blood transfusion for scoliosis surgery. *Anesth Analg* 93:82, 2001.
188. Newman B, Gelb AW, Lam AM: The effect of isoflurane-induced hypotension on cerebral blood flow and cerebral metabolic rate for oxygen in humans. *Anesthesiology* 64:307–310, 1986.
189. Nkenke E, Kessler P, Wiltfang J, et al: Hemoglobin value reduction and necessity of transfusion in bimaxillary orthognathic surgery. *J Oral Maxillofac Surg* 63:623, 2005.
190. Omstein E, Matteo RS, Weinstein JA, Schwartz AE: A randomized controlled trial of esmolol for deliberate hypotension. *Anesthesiology* 67:A423, 1987.
191. Ornsiein E, Matteo RS, Weinstein JA, Schwartz AE: A controlled trial of esmolol for the induction of deliberate hypotension. *J Clin Anesth* 1:31–35, 1988.
192. O'Ryan F, Ebker BN: Prolonged apnea after orthognathic surgery due to atypical cholinesterase. *Int J Oral Surg* 10:338, 1981.
193. Owall A, Lagerkranser M, Sollevi A: Effects of adenosine-induced hypotension on myocardial hemodynamics and metabolism during cerebral aneurysm surgery. *Anesth Analg* 67:228–232, 1988.
194. Pagar DM, Kupperman AW, Stern M: Cutting of nasoendotracheal tube: an unusual complication of maxillary osteotomies. *J Oral Surg* 36:314, 1978.
195. Pasch T, Huk W: Cerebral complications following induced hypotension. *Eur J Anaesthesiol* 3:299, 1986.
196. Patel NJ, Patel BS, Paskin S, Laufer S: Induced moderate hypotensive anesthesia for spinal fusion and Harrington rod instrumentation. *J Bone Joint Surg* 67:1384–1387, 1985.
197. Petrozza PH: Induced hypotension. *Int Anesthesiol Clin* 28:223–229, 1990.
198. Pfleiderer T: Na-nitroprussid. A very potent platelet disaggregating substance. *Acta Univ Carol Med Monogr* 53:247, 1972.
199. Piecuch JF, West RA: Spontaneous pneumomediastinum associated with orthognathic surgery: a case report. *Oral Surg Oral Med Oral Pathol* 48:506, 1979.
200. Pineiro-Aguilar A, Somoza-Martin M et al: Blood loss in orthognathic surgery: a systematic review. *J Oral Maxillofac Surg* 69:885–892, 2011.
201. Posnick JC, Fantuzzo JJ, Troost T: Simultaneous intranasal procedures to improve chronic obstructive nasal breathing in patients undergoing maxillary (Le Fort I) osteotomy. *J Oral Maxillofac Surg* 65:2273, 2007.
202. Posnick JC, Rabinovich A, Richardson DT: Blood replacement practices for complex orthognathic surgery: a single surgeon's experience. *J Oral Maxillofac Surg* 68:54–59, 2010.
203. Practice guidelines for management of the difficult airway: an updated report by the American Society of Anesthesiologists Task Force on Management of the Difficult Airway. *Anesthesiology* 98:1269–1277, 2003.
204. Praveen K, Narayanan V, Muthusekhar MR, Baig MF: Hypotensive anaesthesia and blood loss in orthognathic surgery: a clinical study. *Br J Oral Maxillofac Surg* 39:138, 2001.
205. Precious DS, Bosco DA, Splinter W, Muir J: Induced hypotensive anesthesia for adolescent orthognathic surgery patients. *J Oral Maxillofac Surg* 51(Suppl 3):94–95, 1993.
206. Precious DS, Splinter W, Bosco D: Induced hypotensive anaesthesia for adolescent orthognathic surgery patients. *J Oral Maxillofac Surg* 54:680–683, 1996.
207 Prior S, Heaton J, Jatana KR, Rashid RG: Parker flex-tip and standard-tip endotracheal tubes: a comparison during nasotracheal intubation. *Anesth Prog* 57:18, 2010.
208. Prys-Roberts C, Lloyd JW, Fisher A, et al: Deliberate profound hypotension induced with halothane: studies of haemodynamics and pulmonary gas exchange. *Br J Anaesth* 46:105–116, 1974.
209. Prys-Roberts C, Millard RK: Self-tuning adaptive control of induced hypotension in humans: a comparison of isoflurane and sodium nitroprusside. *J Clin Monit* 6:236–240, 1990.
210. Puelacher W, Hinteregger G, Nussbaumer W, et al: Preoperative autologous blood donation in orthognathic surgery: a follow-up study of 179 patients. *J Craniomaxillofac Surg* 26:121, 1998.
211. Purdham RS: Reduced blood loss with hemodynamic stability during controlled hypotensive anesthesia for Le Fort I maxillary osteotomy using high-dose fentanyl: a retrospective study. *CRNA* 7:33, 1996.
212. Ramstrom G, Sindet-Pedersen S, Hall G, et al: Prevention of postsurgical bleeding in oral surgery using tranexamic acid without dose modification of oral anticoagulants. *J Oral Maxillofac Surg* 51:1211, 1993.
213. Regan F, Taylor C: Recent developments blood transfusion medicine. *BMJ* 318:1435, 2002.
214. Reid RW, Zimmerman AA, Laussen PC, et al: The efficacy of tranexamic acid versus placebo in decreasing blood loss in pediatric patients undergoing repeat cardiac surgery. *Anesth Analg* 84:990, 1997.
215. Renner S, Howantiz PL: Preoperative autologous blood donation in 612 hospitals. *Arch Pathol Lab Med* 116:613, 1992.
216. Rodrigo C: Induced hypotension during anesthesia with special reference to orthognathic surgery. *Anesth Prog* 42:41–58, 1995.
217. Rodrigo C: Anesthetic considerations for orthognathic surgery with evaluation of difficult intubation and technique for hypotensive anesthesia. *Anesth Prog* 47:151, 2000.
218. Rohling RG, Haers PE, Zimmermann AP, et al: Multimodal strategy for reduction of homologous transfusions in cranio-maxillofacial surgery. *Int J Oral Maxillofac Surg* 28:137–142, 1999.
219. Rollason WN, Hough JM: A study of hypotensive anaesthesia in the elderly. *Br J Anaesth* 32:276–285, 1960.
220. Rollason WN, Hough JM: A re-examination of some electrocardiographic studies during hypotensive anaesthesia. *Br J Anaesth* 41:985–986, 1969.
221. Rollason WN, Robertson GS, Cordiner CM, Hall DJ: A comparison of mental function in relation to hypotensive and normotensive anaesthesia in the elderly. *Br J Anaesth* 43:561–566, 1971.

222. Ruggeri ZM, Mannucci PM, Lombardi R: Multimeric composition of factor VIII/von Willebrand factor following administration of DDAVP: implication for pathophysiology and therapy of von Willebrand's disease subtypes. *Blood* 59:1272, 1982.
223. Rummasak D, Apipan B, Kaewpradup P: Factors that determine intraoperative blood loss in bimaxillary osteotomies and the need for preoperative blood preparation. *J Oral Maxillofac Surg* 69:e456–e460, 2011.
224. Saamivaara L, Brander P: Comparison of three hypotensive anaesthetic methods for middle ear microsurgery. *Acta Anaesthesiol Scand* 28:435–442, 1984.
225. Salzman EW, Weinstein MJ, Weintraub RM, et al: Treatment with desmopressin acetate to reduce blood loss after cardiac surgery: a double-blind randomised trial. *N Engl J Med* 314:1402, 1986.
226. Samman N, Cheung LK, Tong ACK, et al: Blood loss and transfusion requirements in orthognathic surgery. *J Oral Maxillofac Surg* 54:21, 1996.
227. Schaberg SJ, Kelly JF, Terry B, et al: Blood loss and hypotensive anesthesia in oral-facial corrective surgery. *J Oral Surg* 34:147–156, 1976.
228. Schendel S, Powell N, Jacobson R: Maxillary, mandibular and chin advancement: Treatment planning based on airway anatomy in obstructive sleep apnea. *J Oral Maxillofac Surg* 69:663–676, 2011.
229. Schindler I, Andel H, Leber J, Kimla T: Moderate induced hypotension provides satisfactory operating conditions in maxillofacial surgery. *Acta Anaesthesiol Scand* 38:384–387, 1994.
230. Sculco PT, Galliana J: Blood management experience: relationship between autologous blood donation and transfusion in orthopedic surgery. *Orthopedics* 22(Suppl):s129, 1999.
231. Segal JB, Blasco-Colmenares E, Norris EJ, et al: Preoperative acute normovolemic hemodilution: a meta-analysis. *Transfusion* 44:632, 2004.
232. Senghore N, Harris M: The effect of tranexamic acid (cyclokapron) on blood loss after third molar extraction under a day case general anaesthetic. *Br Dent J* 186:634, 1999.
233. Sethna NF, Zurakowski D, Brustowicz RM, et al: Tranexamic acid reduces intraoperative blood loss in pediatric patients undergoing scoliosis surgery. *Anesthesiology* 102:727, 2005.
234. Shah N, DelValle O, et al: Esmolol or sodium nitroprusside for induced hypotension during Isoflurane anesthesia. *Vasc Endovascular Surg* 27:681–687, 1993.
235. Shay Bess R, Lenke L, Bridwell K, et al: Wasting of preoperatively donated autologous blood in the surgical treatment of adolescent idiopathic scoliosis. *Spine* 31:2375, 2006.
236. Silva AC, O'Ryan F, Poor DB: Postoperative nausea and vomiting (PONV) after orthognathic surgery: a retrospective study and literature review. *J Oral Maxillofac Surg* 64:1385, 2006.
237. Simpson DA, Ireland J: Hypotensive and normotensive anaesthesia in total hip replacement: a comparative study. *Br J Clin Pract* 37:16–18, 1983.
238. Sindet-Pedersen S, Ramstrom G, Bernvil S, et al: Hemostatic effect of tranexamic acid mouthwash in anticoagulant-treated patients undergoing oral surgery. *N Engl J Med* 320:840, 1989.
239. Sivarajan M, Amory DW, Everett GB, Buffington C: Blood pressure, not cardiac output, determines blood loss during induced hypotension. *Anesth Analg* 59:203–206, 1980.
240. Slater JL: Autologous transfusion: its role in transfusion medicine in Canada. *Ann RCPSC* 21:209, 1988.
241. Sollevi A: Hypotensive anesthesia and blood loss. *Acta Anaesthesiol Scand* 32(Suppl 89):39–43, 1988.
242. Sollevi A, Lagerkranser M, Irestedt L, et al: Controlled hypotension with adenosine in cerebral aneurysm surgery. *Anesthesiology* 61:400–405, 1984.
243. Sood S, Jayalaxmi TS, Vijayaraghavan S, Nundy S: Use of sodium nitroprusside induced hypotensive anaesthesia for reducing blood loss in patients undergoing lienorenal shunts for portal hypertension. *Br J Surg* 74:1036–1038, 1987.
244. Stead SW, Bloor BC: The effects of captopril on the renin-angiotensin system and the sympathetic nervous system during sodium nitroprusside-induced hypotension in the halothane-anesthetized rabbit. *J Cardiovasc Pharmacol* 15:465–471, 1990.
245. Stewart A, Newman L, Sneddon K, et al: Aprotinin reduces blood loss and the need for transfusion in orthognathic surgery. *Br J Oral Maxillofac Surg* 39:365, 2001.
246. St-Hilaire H, Montazem AH, Diamond J: Pneumomediastinum after orthognathic surgery. *J Oral Maxillofac Surg* 62:892, 2004.
247. Stramer SL: Current risks of transfusion-transmitted agents. *Arch Pathol Lab Med* 131:702, 2007.
248. Strickland SM, Westrich GH: Spontaneous compartment syndrome occurring postoperatively in 2 oral surgery patients. *J Oral Maxillofac Surg* 58:814, 2000.
249. Strunn L: Organ perfusion during controlled hypotension. *Br J Anaesth* 47:793–798, 1975.
250. Sudhindran S: Perioperative blood transfusion: a plea for guidelines. *Ann R Coll Surg Engl* 79:299–302, 1997.
251. Suttner SW, Piper SN, Lang K, et al: Cerebral effects and blood sparing efficiency of sodium nitroprusside-induced hypotension alone and in combination with acute normovolemic hemodilution. *Br J Anaesth* 87:699, 2001.
252. Tanaka N, Sakahashi H, Sato E, et al: Timing of the administration of tranexamic acid for maximum reduction in blood loss in arthroplasty of the knee. *J Bone Joint Surg Br* 83:702, 2001.
253. Teeples TJ, Rallis DJ, Rieck KL, et al: Lower extremity compartment syndrome associated with hypotensive anesthesia for orthognathic surgery: a case report and review of the disease. *J Oral Maxillofac Surg* 68:1166, 2010.
254. Thiagarajamurthy S, Levine A, Dunning J: Does prophylactic tranexamic acid safely reduce bleeding without increasing thrombotic complications in patients undergoing cardiac surgery? *Interact Cardiovasc Thorac Surg* 3:489, 2004.
255. Thompson GE, Miller RD, Stevens WC, Murray WR: Hypotensive anesthesia for total hip arthroplasty: a study of blood loss and organ function (brain, heart, liver, and kidney). *Anesthesiology* 48:91–96, 1978.
256. Thompson HJ: The management of post-operative nausea and vomiting. *J Adv Nurs* 29:1130, 1999.
257. Tobias JD: Nicardipine for controlled hypotension during orthognathic surgery. *Plast Reconstr Surg* 99:1539–1543, 1997.
258. Tough ICK: Induced hypotension and post operative bleeding. *Anaesthesia* 15:154–157, 1960.
259. Ueki K, Marukawa K, Shimada M, et al: The assessment of blood loss in orthognathic surgery for prognathia. *J Oral Maxillofac Surg* 63:350, 2005.
260. Vamvakas EC: Meta-analysis of randomized controlled trials comparing the risk of postoperative infection between recipients of allogenic and autologous blood transfusion. *Vox Sang* 83:339, 2002.
261. Van de Perre JPA, Stoelinga PJW, Blijdorp PA, et al: Perioperative morbidity in maxillofacial orthopaedic surgery: a retrospective study. *J Craniomaxillofac Surg* 24:263, 1996.
262. Vazeery AK, Lunde O: Controlled hypotension in hip joint surgery. An assessment of surgical hemorrhage during sodium nitroprusside infusion. *Acta Orthop Scand* 50(4):433–441, 1979.
263. Venugoplan SR, Nanda V, Turkistani K, et al: Discharge patterns of orthognathic surgeries in the United States. *J Oral Maxillofac Surg* 70:e77–e86, 2012.
264. Wagner JD, Moore DL: Preoperative laboratory testing for the oral and maxillofacial surgery patient. *J Oral Maxillofac Surg* 49:177, 1991.
265. Wang HH, Liu LMP, Katz RL: A comparison of the cardiovascular effects of sodium nitroprusside and trimethaphan. *Anesthesiology* 46:40–48, 1977.
266. Warner WA, Shumrick DA, Caffrey JA: Clinical investigation of prolonged induced hypotension in head and neck surgery. *Br J Anaesth* 42:39–44, 1970.
267. Washburn MC, Hyer RL: Deliberate hypotension for elective major maxillofacial surgery: a balanced halothane and morphine technique. *J Maxillofac Surg* 10:50–55, 1982.

268. Weerasinghe JU, Gunawardana RH: Delayed tracheal extubation in oral and maxillofacial surgery. *Br J Oral Maxillofac Surg* 37:152, 1999.
269. Wildsmith JAW, Drummond GB, MacRae WR: Blood-gas changes during induced hypotension with sodium nitroprusside. *Br J Anaesth* 47:1205–1210, 1975.
270. Wildsmith JAW, Sinclair CJ, Thom J, et al: Hemodynamic effects of induced hypotension with a nitroprusside-trimethaphan mixture. *Br J Anaesth* 55:381–389, 1983.
271. Wilson ME, Ellis FR: Predicting malignant hyperpyrexia. *Br J Anaesth* 51:66, 1979.
272. Wood M, Hyman S, Wood AJJ: A clinical study of sensitivity to sodium nitroprusside during controlled hypotensive anesthesia in young and elderly patients. *Anesth Analg* 66:132, 1987.
273. Yamada S, Brauer F, Knierim D, et al: Safety limits of controlled hypotension in humans. *Acta Neurochir Suppl* 42:14–17, 1988.
274. Yu CN, Chow TK, Kwan AS, et al: Intra-operative blood loss and operating time in orthognathic surgery using induced hypotensive general anesthesia: prospective study. *Hong Kong Med J* 6:307, 2000.
275. Yun KI, Lee JA, Park JU: Intubation granuloma: report of a case. *J Oral Maxillofac Surg* 66:1263, 2008.
276. Zellin G, Rasmusson L, Palsson J, et al: Evaluation of hemorrhage depressors on blood loss during orthognathic surgery: a retrospective study. *J Oral Maxillofac Surg* 62:662, 2004.
277. Zonis Z, Seear M, Reichert C, et al: The effect of preoperative tranexamic acid on blood loss after cardiac operations in children. *J Thorac Cardiovasc Surg* 111:982, 1996.
278. Zulian MA, Chisum JW, Mosby EL, et al: Extubation criteria for oral and maxillofacial surgery patients. *J Oral Maxillofac Surg* 47:616, 1989.

12 Orthognathic Surgery Treatment Planning

JEFFREY C. POSNICK, DMD, MD

Part I: The Importance of Proportions and Symmetry to Establish Ideal Facial Aesthetics
- Fundamental Concepts
- Evolution in Thinking
- Current Philosophy

Part II: Clinical Evaluation of the Patient with a Dentofacial Deformity
- Background
- Global Head and Neck Assessment: Seven Critical Aspects
- Detailed Maxillomandibular Surgical Planning: Eight Critical, Quantitative, To-the-Millimeter Decisions
- Conclusions

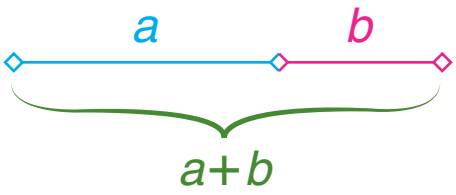
Part I: The Importance of Proportions and Symmetry to Establish Ideal Facial Aesthetics

Fundamental Concepts

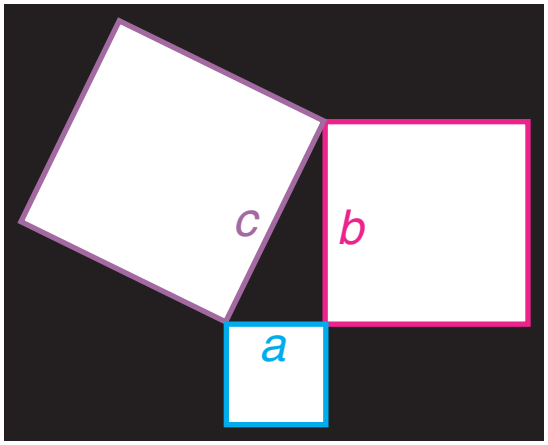
We use the word *proportion* to describe the comparative relationship between parts of things in an attempt to describe harmonious and disharmonious relationships between different parts of a whole.[13,14,49,92] The first clear and still unsurpassed definition of what has become known as the "golden ratio" was put into words by Euclid of Alexandria around 300 B.C.[5,29,42-44,61] Euclid is best known as the founder of geometry, which is a branch of mathematics that involves the use of deductive reasoning.[68] In the most pure way, Euclid defined a harmonious proportion derived from the simple division (sectioning) of a line into what he called its "extreme and mean ratio."[61] In Euclid's own words, "[a] straight line is said to have been cut in extreme and mean ratio when, as the whole line is to the greater segment, so is the greater to the lesser."[98,99] (Fig 12-1) This ratio was defined by Euclid as the *"golden ratio."* Interestingly, this ratio represents a never-ending (irrational) number 1.6180339887…. The common symbol for the golden ratio is the Greek letter tau (τ), the meaning of which is "the cut" or "the section." It was later renamed phi (Ψ) after the Greek sculptor Phidias, who was said to have employed it[61]; this is described in more detail later in this chapter.

A definition of the word *beauty* can be found in Webster's unabridged dictionary as "the quality which makes an object seem pleasing or satisfying in a certain way."[63] The golden ratio is said to be an important feature that is found in many classically beautiful works of art, architectural structures, objects designed for human use, the music of the great composers, and most of what is found in nature.[17,18,20,45,49,57,58,67,68] "Perfect" proportions are found throughout the art world and throughout the centuries. Objects of "classic" beauty and "attractive" forms found in nature consistently reveal the golden ratio.[40] However, an important question remains: "Does form really follow function?" Richard Buckminster Fuller (1895-1983), a great American architect and writer, once stated, "When I am working on a problem, I never think about beauty. I think only of how to solve the problem. But when I finish, if the solution is not beautiful, I know it is wrong."[61]

In their simplest form, attractive facial images—whether in drawings, in photographs, or in reality—seem to incorporate a *consistent symmetry of proportion*. It has been stated that most people have a common appreciation of what is attractive when they visualize it even if they cannot quite explain why. This has been demonstrated across cultures and throughout the ages, and it has been relatively consistent in art and photography.[6,19,27,32,48,53,54,63,77,81,80] A strict mathematical formula may not be required to define classic attractiveness, but the question remains: "Can the golden ratio (a mathematical formula) be found in these commonly agreed upon beautiful images?"



• **Figure 12-1** Golden ratio (section) of a straight line.



• **Figure 12-2** Pythagorean theorem as described through a triangle.

Evolution in Thinking

Pythagoras (570 B.C.-522 B.C.) coined the terms *philosophy,* defined as "a love of wisdom," and *mathematics,* defined as "that which is learned."[47] He described a *philosopher* as someone who "gives himself up to discovering the meaning and purpose of life itself—to uncover the secrets of nature."[100,42,61] Pythagoras and his followers (i.e., Pythagoreans) are best known for their role in the development of mathematics and the application of mathematic principles to the concept of *order in the world*. The Pythagorean theorem states that, in a right-angle triangle, the square of the hypotenuse (i.e., the side opposite the right angle) is equal to the sum of the squares of the other two sides; in other words, $a^2 + b^2 = c^2$ (Fig. 12-2).[9]

According to legend, Pythagoras discovered that musical notes could be translated into an orderly mathematical equation by observing a blacksmith at work. He thought that the sounds emanating from the blacksmith's anvils, when they were struck, were both beautiful and harmonious. By studying the blacksmith's tools, Pythagoras discovered that the emanating sounds were "simple ratios of each other and that one was half the size of the first and so on."[61,100] He then constructed metal bells with each sized in equal ratio to the next. When he struck the proportionately sized bells, harmonious tunes resulted in the same way that he had observed with the anvils. The harmony that resulted when striking the proportionately sized bells or anvils, according to Pythagoras, was beautiful.

• **Figure 12-3** *Doryphoros* by Polykleitos. *From http://en.wikipedia.org/wiki/File:Doryphoros_MAN_Napoli_Inv6011-2.jpg Photograph by Marie-Lan Nguyen. Creative Commons License: Attribution.*

Pythagoreans also believed that "harmony existed with the spheres of the planets and stars according to a similar strict mathematical equation and thus produced a similar "planetary symphony."[61,100] Pythagoras later discovered flaws in the purity of mathematics. He was the first to write of having knowledge of a number that is "neither a whole—nor a ratio of two whole numbers."[61,100] This discovery was said to have overwhelmed his perspective of a world based on the intrinsic properties of whole numbers and their ratios. He could not find a role for irrational numbers in this world.[44] Pythagoreans believed that the existence of such irrational numbers was so horrific that such numbers represented some sort of cosmic mistake and thus should be suppressed and kept secret.

Pythagoras and the Pythagorean school made a great impact in various fields, from sculpture to philosophy.[55] Polykleitos (450 B.C.) is a vivid example of the positive influence of Pythagoras' ideas. Polykleitos consciously created a new approach to sculpture that is immortalized in his treatise *Kanon* and visually depicted in his design of the male nude (also known as *Kanon*), which exemplified his aesthetic theories regarding the mathematical basis of artistic perfection.[69] His general artistic aims were clarity, balance, and completeness. The *Kanon*'s basic principles are represented in Polykleitos' best-known bronze sculptures, including *Doryphoros* (Fig 12-3); *Diadumenos* (Fig 12-4); and *Astragalizontes.* These sculptures are said to have a precise commensurability (i.e., "symmetria") of all of the parts with one another. The idea of "symmetria" comes from the Hippocratic principles of "equilibrium" and "rhythmos."[100] Polykleitos also said that "perfection comes about little by little through many numbers."[61,100] This meant that

• **Figure 12-4** *Diadumenos* by Polykleitos. *Image Copyright © The Metropolitan Museum of Art, Image source: Art Resource, NY.*

a statue should be composed of clearly definable parts, all of which are related to one another through a system of ideal mathematical proportions and balance. This was expressed in terms of the ratios established by Pythagoras, including in the perfect intervals of the musical scale: 1:2 (octave), 2:3 (harmonic fifth), and 3:4 (harmonic fourth).

The Polykleitos school continued after Polykleitos' death and included at least 20 sculptors and artisans through early 200 B.C. They were defined by their adherence to the *Kanon* and its *principles of balance and definition* (i.e., clarity in the separation and relationship of the parts to the whole). Polykleitos' students would think beyond sculpture in bronze; this included the work of Polykleitos' son, Polykleitos the Younger, whose architectural design of the great theater of Epidaurus was remarkable for its harmony and balance (Fig. 12-5).[100] Within the theater, the place for spectators was divided into two tiers: the first one had 34 rows of places, and the second one had 21 rows (i.e., 34/21 = 1.61…). The angle between the theatron and the scene divides the circumference of the basis of the amphitheater in a ratio of 1.618…. Interestingly, both of these meet the standards of the golden section, which was soon to be precisely and clearly defined by Euclid, as described later in this chapter.

Polykleitos' greatest work is said to be the sculpture *Doryphoros* (i.e., "spear-bearer"), which is considered one of the best-known sculptures of the classic era.[100] During the second century A.D., Galen would write about *Doryphoros* as the perfect visual expression of Greece's search for harmony and beauty, which is rendered in the perfectly proportioned sculpted male nude. Galen stated the following:

• **Figure 12-5** Theater of Epidaurus in Epidaurus, Greece. Copyright 2013 BootsnAll Travel Network, LLC. All Rights Reserved. http://www.greecelogue.com/the-ancient-theater-of-epidaurus.html.

> *Chrysippos [or Doryphoros, the spear-bearer] holds beauty to consist not in the commensurability or "symmetria" [i.e., proportions] of the constituent elements [of the body], but in the commensurability of the parts, such as that of finger to finger, and of all the fingers to the palm and wrist, and of those of the forearm, and of the forearm to the upper arm, and in fact, of everything to everything else, just as it is written in the Canon of Polyclitus [Polykleitos]. For having taught us in that work all the proportions of the body, Polyclitus supported his treatise with a work: he made a statue according to the tenets of his treatise and called the statue, like the work, the "Canon" [i.e.,* Kanon*].*[61,100]

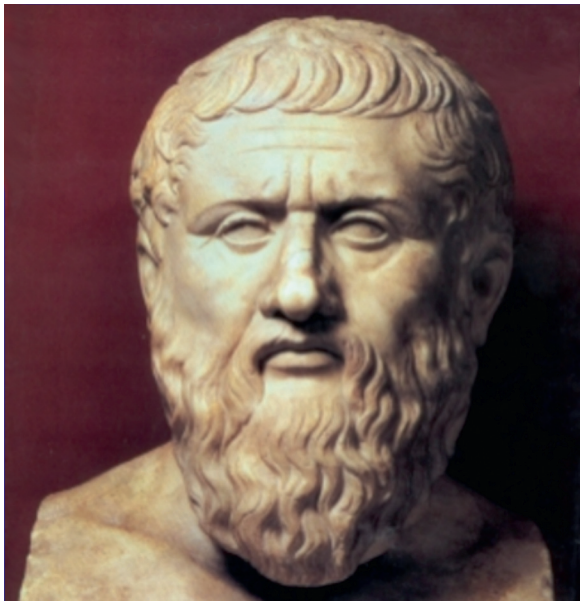
A contemporary of Polykleitos was Phidias (480 B.C.),[100] who is regarded as one of the greatest sculptors of bronze, gold, and ivory from Greek antiquity. Phidias' statue of *Zeus at Olympia* was considered one of the seven manmade wonders of the Ancient World (Fig. 12-6). He also created the statue of the *Goddess Athena* on the Athenian Acropolis (Fig. 12-7) as well as the *Athena Promachos* (i.e., a bronze statue of Athena) adjacent to the entrance of the Acropolis in Athens (Fig. 12-8). The influence of Pythagorean mathematics, the *Kanon* (i.e., the relationship of the parts to the whole), and the golden ratio (although not spoken of as a specific number) are clearly represented in all of Phidias' works.

Pythagoras' writings also influenced Plato (428 B.C.-337 B.C.), who is considered one of the most extraordinary minds of Ancient Greece and of Western civilization in general.[100] His text, the *Republic,* states that mathematics is an absolute must for the education of all state leaders and philosophers (Fig. 12-9). Plato was intrigued by the *five regular polyhedrons* that are now considered the *Platonic solids* (Fig. 12-10). He used their design to emphasize the importance of proportions for the achievement of "ideal" beauty. His thinking must have also been greatly influenced by Polykleitos' sculptures and written words in the *Kanon.* Plato is quoted as follows:

• **Figure 12-6** Statue of Zeus at Olympia by Phidias.

• **Figure 12-8** Statue of the Goddess Athena at the entrance of the Athenian Acropolis by Phidias. *From http://en.wikipedia.org/wiki/File:NAMA_Ath%C3%A9na_Varvakeion.jpg Photograph by Marsyas. Creative Commons License: Attribution.*

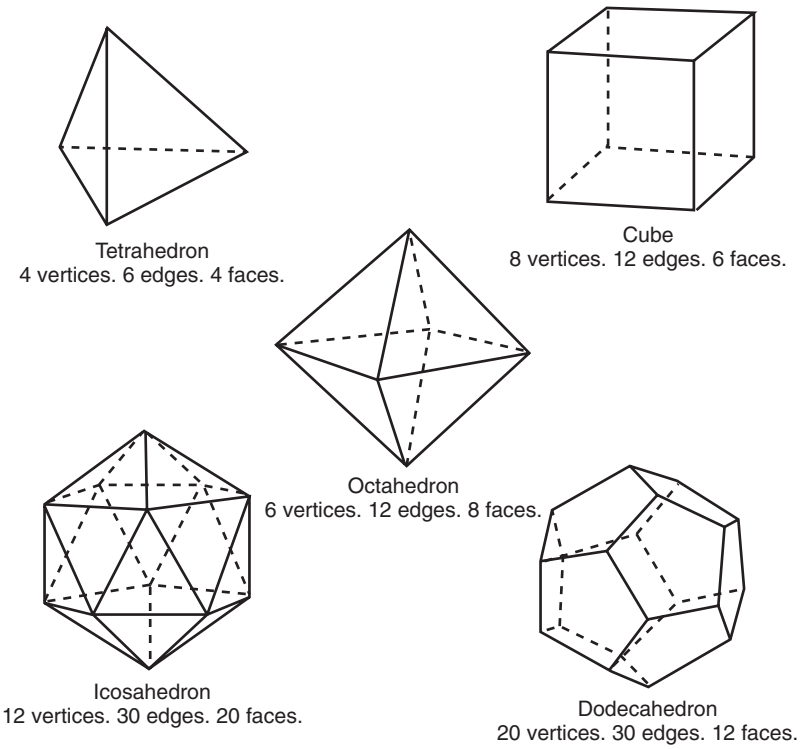
• **Figure 12-7** Statue of the Goddess Athena on Athenian Acropolis by Phidias.



• **Figure 12-9** Bust sculpture of Plato.

Two parts or values cannot be satisfactorily connected among themselves without a third part; the most beautiful link is that, which together with two initial values gives the perfect unit. It is reached in the best way by proportion [analogy], in which among three numbers, planes or bodies, the mean one so concerns to the second one, as the first one to the mean one, and also the second one to the mean one as the mean one to the first one. This implies, that the mean one can exchange the first one and the second one, the first one and the second one can exchange the mean one and all things together thus makes an indissolvable unit.[61,100]

Plato's favored student Aristotle (384 B.C.-?) was able to take Pythagorean ideas and the *Kanon* of Polykleitos a step further forward when he wrote that *the main requirements of beauty include order, proportionality, and control of sizes* (i.e., the relationship of the parts to each other).[100] *Order*



• **Figure 12-10** The five Platonic solids described by Euclid.

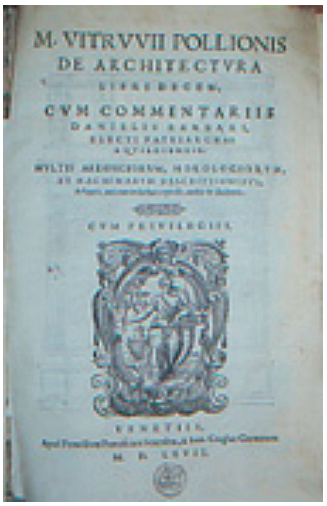
was said to arise "when between parts of the whole there are definite ratios in proportions."[61,100] In music, for example, Aristotle recognized the octave as the most beautiful consonance when taking into consideration that the number of oscillations between the basic tone and the octave can be expressed by the first numbers of a natural series: 1 : 2. He also had the opinion that *rhythmic relations* of a poetic verse should be based on small numerical ratios to reach a beautiful impression.[41] Like Plato, Aristotle believed that *beauty was commensurate of the separate parts to the whole*.

The young Alexander the Great of Macedonia (Fig. 12-11) succeeded to the throne after his father's assassination (336 B.C.). He went on to conquer most of Asia Minor, Syria, Egypt, and Babylon, and he then founded the City of Alexandria near the mouth of the Nile. Alexandria developed into the intellectual center of the hemisphere, and it included a library that is said to have held more than 700,000 books. The first group of scholars at the center included the young *Euclid* (300 B.C.), who authored a complete text about the history of mathematics that included many of his own original ideas (Fig. 12-12). The *Elements* was a 13-volume work on geometry and number theory.[43] Each book contained a number of theorems (i.e., from 10 to 100) that follow a series of definitions. Scholars believe that the *Elements* is primarily a collection of theorems described by other mathematicians that was then supplemented by at least some original work by Euclid. Proclus, a Greek mathematician who lived several centuries after Euclid, wrote the following in his commentary about the *Elements*: "Euclid, who put together the *Elements*, collecting many of Eudoxus' theorems, perfecting many of

• **Figure 12-11** Sculpture of Alexander the Great of Macedonia. *From http://en.wikipedia.org/wiki.File:Alexander1256.jpg*

Theaetetus, and also bringing to irrefutable demonstration the things which were only somewhat loosely proven by his predecessors."[5] For example, Pythagoras was probably the source of most of Books 1 and 2; the ideas of Hippocrates were found in Book 3, and those of Eudoxus were in Book 5; Books 4, 6, 11, and 12 probably came from other Pythagorean or Athenian mathematicians. Euclid is credited with replacing Fallacious' proofs with his own more rigorous versions. Most of the definitions, postulates, and axioms within the *Elements* date back to Plato. Euclid also authored

• **Figure 12-12** The 13-volume *Elements* text written by Euclid.



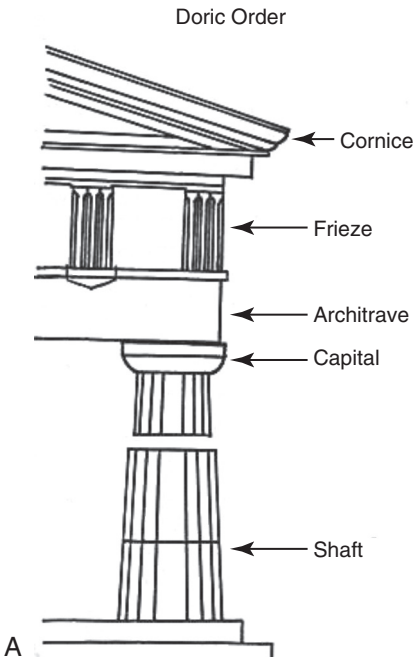
• **Figure 12-13** The 10-volume *De Architectura* text written by Marcus Vitruvius Polio.

almost a dozen other books that covered topics from music to mechanics and optics. His book *Optics* contains some of the earliest studies of perspective. Interestingly, until the 20th century, Euclid's *Elements* remained second only to the Bible with regard to the number of books sold worldwide. Euclid's textbook, through various translations, has remained actively referenced for more than 2300 years.

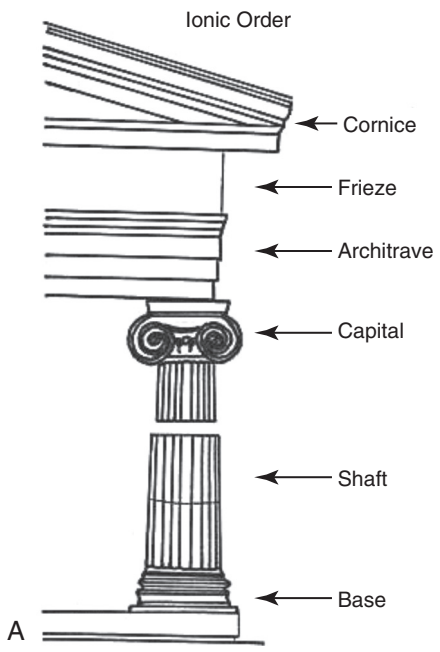
The golden ratio or golden section appears in the *Elements,* primarily in relation to proportions in Book 6: *On Divisions of Figures.*[43] Book 6 survives only partially in an Arabic translation, and it discusses the division of geometric figures into two or more parts or into parts in given ratios. It is likely that Euclid's primary interest in the golden ratio was as a means of geometric interpretation of the construction of the five Platonic solids. This discussion is also found in Book 13, which constructs the five regular Platonic solids that are inscribed within a sphere. Euclid calculated the ratio of their edges to the radius of the sphere and proved that there are no other regular solids to be found. Curiosity about the structure and design of these Platonic Solids were of much interest to Ancient Greek intellectuals.[47] In addition, throughout the *Elements,* Euclid described several theorems and their proofs, which also employed the golden ratio. Although no documentation has been found to confirm Euclid's knowledge of Polykleitos' *Kanon* or Aristotle's stated *requirements of beauty (i.e., order, proportionality, and control of sizes),* it is almost inconceivable that these ideas did not influence him.

Marcus Vitruvius Polio (80 B.C.-15 B.C.) came two centuries after Euclid; he was a Roman writer, architect, and engineer. At one point, he even served as Chief of Artillery for the Roman Army. His written work *De Architectura,* a 10-volume treatise on architecture, is not just remarkable for its originality and contents but also for being the only surviving major book about architecture from classic antiquity (Fig. 12-13). This treatise indicates a mastery of architecture and engineering, and it crystallizes the importance of precise proportions as a fundamental component of beauty in the human form. Interestingly, in the records that remain, no mention is made by Vitruvius of Pythagoras, Polykleitos, or Euclid. This lack of acknowledgement by Vitruvius is striking in its absence; thus, it likely represents lost documents rather than a lack of awareness.

Fortunately, Vitruvius' treatise would later be rediscovered by the Florentine humanist Poggio Bracciolini (1414). It was Leon Battista Alberti (1404-1472) who then revitalized Vitruvius' works by writing a widely read seminal treatise on architecture, *De Re Aedificatoria,* which was published in 1450.[35] During the High Italian Renaissance, this volume had a great influence on artists, scholars, and architects, including Leonardo da Vinci (1452-1519) and Michelangelo (1475-1564).[72] Vitruvius is most remembered for his basic assertion that a structure must exhibit the three qualities of *firmitas, utilitas,* and *venustas:* it must be strong (durable), useful, and beautiful. According to Vitruvius, architecture is an imitation of nature: "As birds and bees built their nests, so humans construct housing, from natural materials that give them shelter against the elements."[61] When perfecting the art of building, ancient greeks designed and defined the three architectural orders: *Doric* (Fig. 12-14), *Ionic* (Fig. 12-15), and *Corinthian* (Fig. 12-16).[100] These architectural orders gave the Ancient Greek buildings a sense of proportion and continuity that was believed to be as important as the proportions of the greatest known work of art: the human body. This led Vitruvius to define the proportions of the human body in words that would later be illustrated by Leonardo da Vinci's *Vitruvian Man* (1437; Fig. 12-17). The human body as described by Vitruvius was inscribed in a circle and in a square. These are considered the fundamental geometric patterns of the cosmic order.[102]



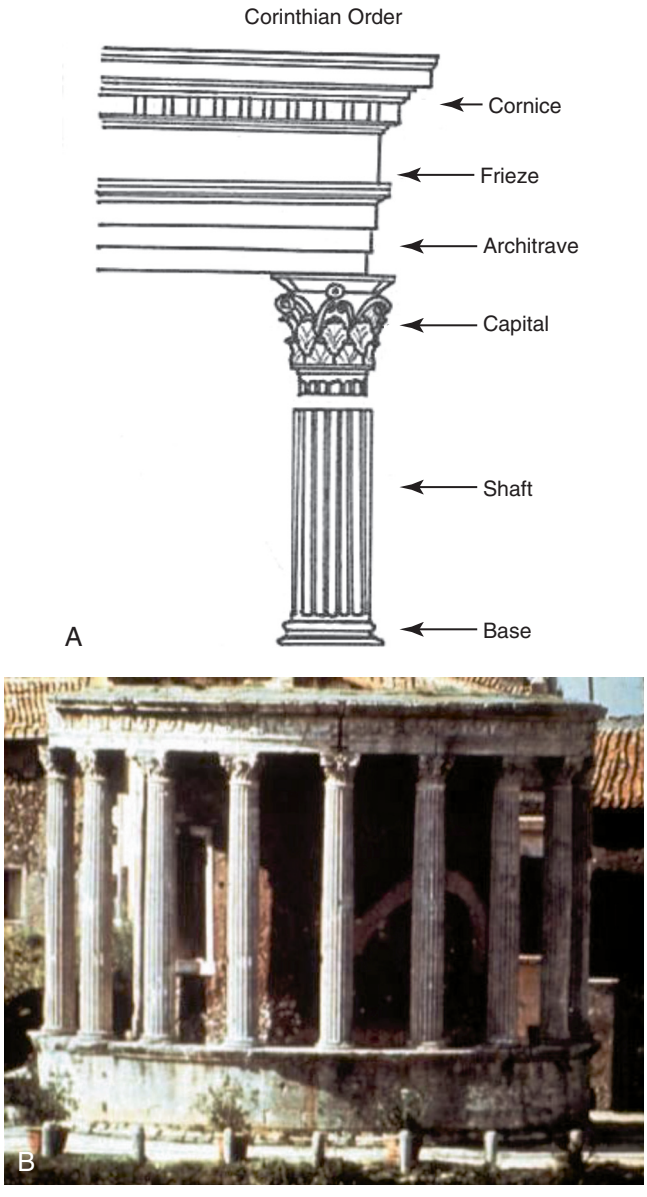
• **Figure 12-14 A,** Illustrations of Doric architecture. **B,** Doric architecture as demonstrated by the Parthenon from Ancient Greece. *Courtesy Charlotte-Mecklenburg Historic Landmarks Commission.* http://www.cmhpf.org/kids/dictioary/classicalorders.html



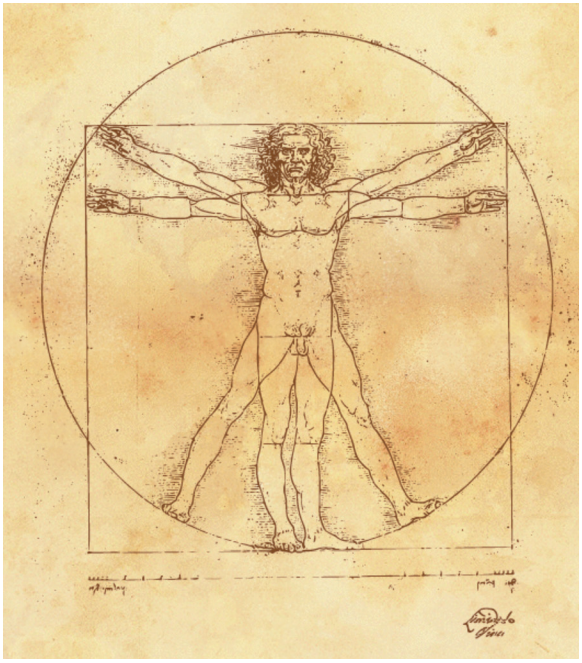
• **Figure 12-15 A,** Illustrations of Ionic architecture. **B,** Ionic architecture as demonstrated by the Temple of Athena Nike from Ancient Greece. *Courtesy Charlotte-Mecklenburg Historic Landmarks Commission.* http://www.cmhpf.org/kids/dictioary/classicalorders.html

After the destruction of the great Alexandrian library—first by Roman, then by Christian, and finally by Muslim groups—the center of study came to an end (529 A.D.). With the ascendancy of Islam and the initial attempts of Islamic groups to destroy everything "non-Islam," many of the earlier Greek and Roman literary works were lost as the Arab world became the dominant center for intellectual study. Abu Kmil Shuja (850-930), who was known as *al-Hasib al-Misri* (translated as "The Calculator from Egypt"), shared his rediscovery of Euclid's works through Arabic translations.

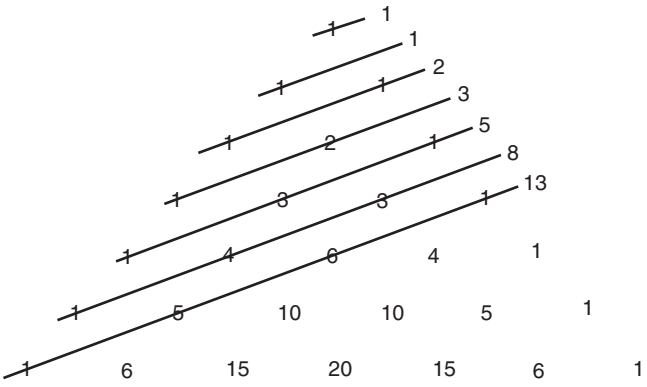
Abu Kmil's books would later serve as a basis for Leonardo of Pisa's further understanding of Euclid's geometric principles. Leonardo of Pisa (1170-1250) was also known as Fibonacci, Leonardo Bonacci, Leonardo Pisano Bogollo, and Leonardo Fibonacci; he was considered the most talented mathematician of the late middle ages.[25] He is best remembered by the modern world for spreading knowledge of the Hindu-Arabic numeral system (i.e., digits 0 through 9) in Europe; this was primarily done through publication during the earlier 13th century of the *Book of Calculations* or *Liber Abaci*.[97] His second contribution was the recognition of a number sequence that became known as the *Fibonacci numbers* (Fig 12-18). Within the *Liber Abaci*, Fibonacci posed and solved a classic problem involving the growth of a hypothetical population of rabbits that was based on idealized assumptions (Fig 12-19). The solution was a sequence of numbers that was later named after Fibonacci. Although this number sequence was known to Indian mathematicians as early as the sixth century, it was Fibonacci's *Liber Abaci* that introduced it to the West.[101] In the sequence of the Fibonacci numbers, each number is the sum of the previous



• **Figure 12-16 A,** Illustrations of Corinthian architecture. **B,** Corinthian architecture as demonstrated by the Temple of the Sybil from Ancient Greece. Courtesy Charlotte-Mecklenburg Historic Landmarks Commission. http://www.cmhpf.org/kids/dictionary/classicalorders.html.



• **Figure 12-17** *Vitruvian Man* by Leonardo da Vinci. This idea was described by Marcus Vitruvius Polio.



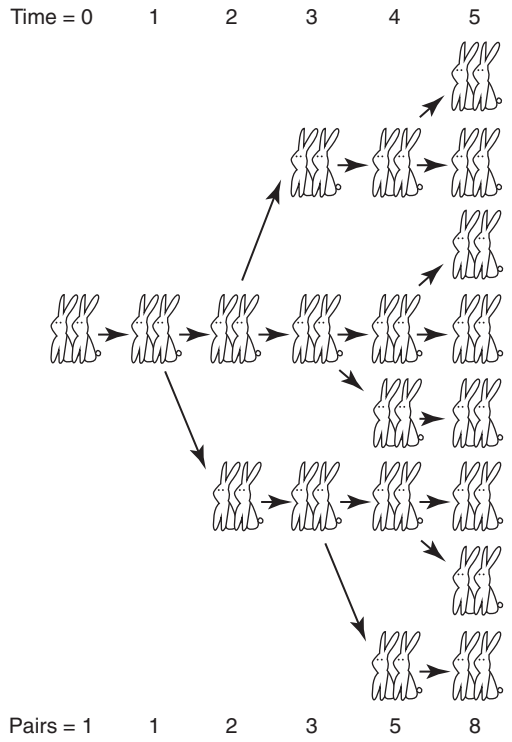
• **Figure 12-18** Fibonacci numbers.

two numbers, starting with 0 and 1. Thus, the sequence begins 0, 1, 1, 2, 3, 5, 8, 13, 21, 34, 55, 89, 144, and so on. Interestingly, the higher up in the sequence, the closer two consecutive numbers in the sequence—when divided by each other—will approach the golden ratio of approximately 1 : 1.618 or 0.618 : 1. The sequence of the Fibonacci numbers and their relative ratios have been found with consistency in nature, art, and classic objects of beauty. They would soon be recognized and used by the Renaissance painters.

The Fibonacci numbers and the golden ratio are found in the most basic forms in nature.[25] For example, spirals arise from a property of growth called "self-similarity" (i.e., the tendency to grow in size but to maintain the same shape). This is seen in the simplest of natural forms: the *shell of the chambered nautilus*. As the nautilus outgrows each chamber, it builds a new chamber for itself, but this always has the same shape (Fig. 12-20). If the nautilus lives a long life, it will spiral around and around, growing even larger but always looking exactly the same at every scale. Interestingly, by using the Fibonacci sequence of numbers, you can build a nautilus that is based on squares by starting with a square of size 1 and successively building new "rooms" with sizes that correspond with the Fibonacci sequence.[39] Running through the centers of the squares, a smooth curve can be drawn that corresponds to the nautilus spiral (see Fig. 12-20, *B*). This self-similar curve, which keeps its shape at every scale, is called *equiangular* because a radial line from the center always makes the same angle to the curve. This curve was also known to Archimedes of Ancient

Greece, who—philosophically and religiously—thought of this curve as both spiraling inward forever as well as spiraling outward forever. Euclid's golden ratio mathematically describes this finding both in nature and in the Fibonacci numbers.

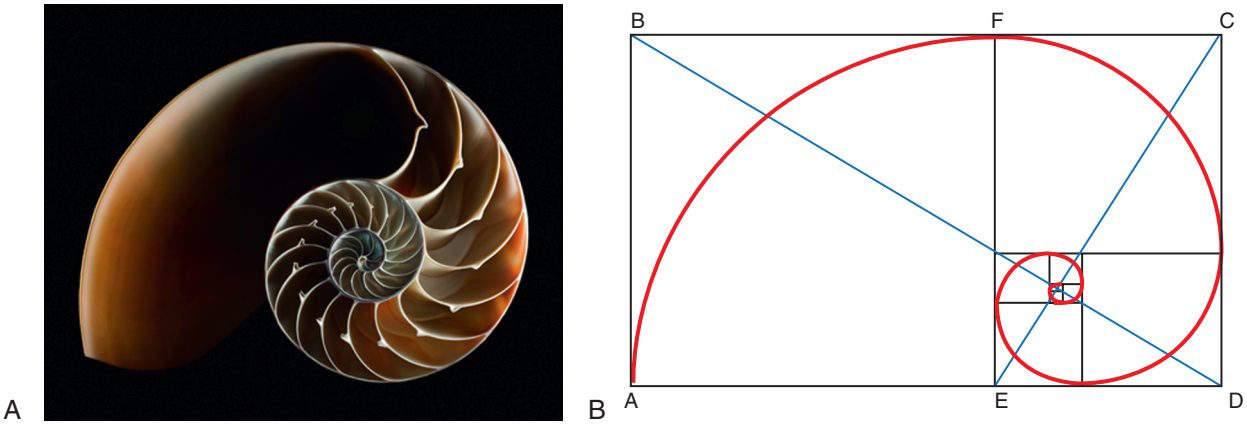
Euclid's ideas have had an enormous impact throughout the centuries,[5] first on the medieval Italian philosopher St. Thomas Aquinas (1225-1274) and then on the Renaissance Italian architect L.B. Alberti (1404-1472).[72] Aquinas captured the fundamental relationship between beauty and mathematics when he stated that *"humans seem to react with a sense of pleasure to forms that possess certain symmetries [proportions] and obey certain mathematical rules."*[35,61]



• **Figure 12-19** Fibonacci numbers as described through breeding rabbits. The sequence 1, 1, 2, 3, 5, 8, 13, 21, 34, 55, 89, 144, 233, and so on—in which each term (starting with the third) is equal to the sum of the two preceding terms—was appropriately dubbed the *Fibonacci numbers* during the 19th century by the French mathematician Edouard Lucas (1842-1891).

Leon Battista Alberti was an author, artist, architecture, poet, priest, linguist, philosopher, cryptographer, and general Renaissance humanist. Alberti regarded mathematics (as summarized by Euclid) to be the common ground of the arts and the sciences. For Alberti, *beauty was "the harmony of all parts in relation to one another."* He stated that "this concord is realized in a particular number, proportion and arrangement demanded by harmony."[35,61] Alberti's thoughts on harmony were referenced back to Pythagoras and Euclid, but he set them in a fresh context. He studied Ancient Roman sites, ruins, and objects and made detailed observations that are cataloged in his *Ten Books of Architecture (De Re Aedificatoria),* which was published in 1450 A.D. His writings were patterned after the *De Architectura* by Vitruvius (46 B.C.-30 B.C.) and covered a wide range of subjects, from history to town planning to engineering to the philosophy of beauty.

Three Renaissance genius mathematicians and artists were clearly influenced by Euclid's ideas[72]: Piero della Francesca (1415-1492), Leonardo da Vinci (1452-1519), and Luca Pacioli (1446-1517). Piero della Francesca was known as a mathematician, a geometer, and an artist. His painting was characterized by its serene humanism and its use of geometric forms, particularly in relation to perspective and foreshortening (Fig. 12-21). It is known that he was at least acquainted with Leon Battista Alberti's mathematical and architectural work and that he would also have a significant influence on Luca Pacioli's thinking.[71] Three important mathematical treatises were written by Piero: *Abacus Treatise (Trattato d'Abaco)*; *Short Book on the Five Regular Solids (Libellius de Quinque Corporibus Regularibus)*; and *Perspective on Painting (De Prospectiva Pingendi)*. The subjects covered in these writings include mathematics, algebra, geometry, and innovative work on both solid geometry and perspective. Much of Piero's work would later be rewritten



• **Figure 12-20 A,** Photograph of the chambered nautilus, which demonstrates structural anatomy. **B,** The golden ratio as found in the spiral structural anatomy of the nautilus.

• **Figure 12-21** *Madonna and Child with Saints* by Piero della Francesca.

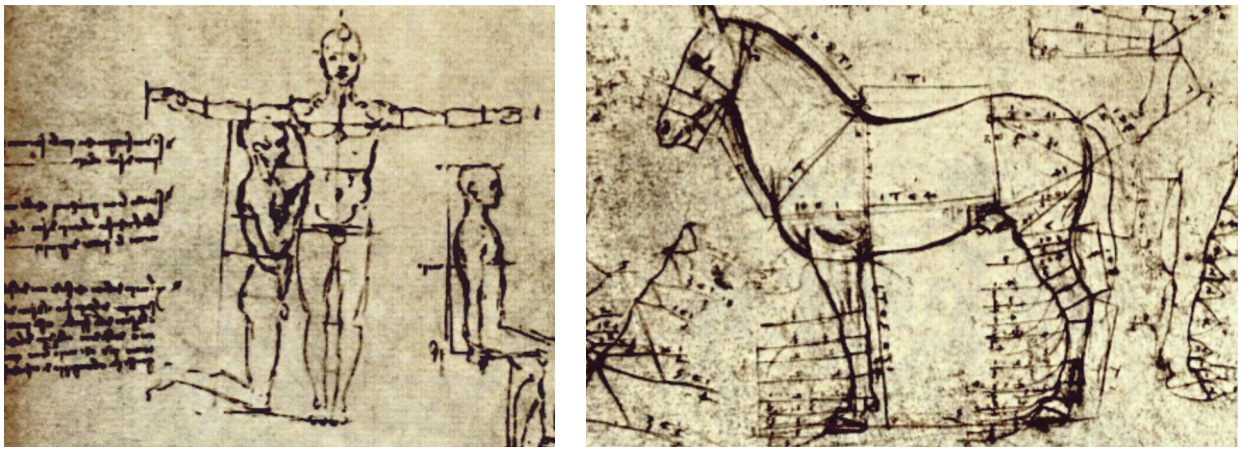
• **Figure 12-22** Self-portrait by Pacioli.

by others, including Luca Pacioli.[71] Piero's work on solid geometry appears in Pacioli's *De Divina Proportione*, which is believed to have been illustrated by Leonardo da Vinci. Piero's *Perspective on Painting (De Prospectiva Pingendi)* clearly refers to Euclid's *Elements* and *Optics*. Piero used Euclid's written works to demonstrate that, to achieve perspective in a painting, the artist must rely on the scientific basis of visual perception. Piero's treatise *De Prospectiva Pingendi* became the standard manual of the day for artists who attempted to paint plain figures and solids.

Luca Pacioli (Fra Luca Bartolomeo de Pacioli) lived from 1446 to 1517. He was a Franciscan friar and an Italian mathematician.[71] He later collaborated with Leonardo da Vinci, and he also made seminal contributions to the field that is now known as accounting. In 1494, his first book—*Summa di Aritmetica, Geometria, Proporzioni e Proporzionalità* was published in Venice. This text compiled the knowledge of the time related to arithmetic, algebra, geometry, and trigonometry. Pacioli identified Fibonacci (Leonardo of Pisa) as the primary source for his basic facts. Several years later, he met, collaborated with, lived with, and taught mathematics to Leonardo da Vinci. Pacioli also completed the three-volume treatise *The Divine Proportion (De Divina Proportione),* which applies to geometrical proportions, letters, and architecture constructions. The original manuscript, which was written around 1496 to 1498, is preserved in Milan; it was published in Venice in 1509.[71] Within the text, Pacioli lists five reasons why a more appropriate name for Euclid's golden ratio would be *"the divine proportion."* References and analogies to the church and godliness clarify Pacioli's *belief that the divine proportion is spiritual and universal*. He was also interested in the process of painting, and he emphasized his desire to reveal the secret to the creation of harmonic forms through the use of the golden ratio (or the divine proportion). Pacioli used the square and the compass (which are clearly visible in the background of his well-known self-portrait) to both analyze the works of others and to help artists achieve divine proportions in their paintings (Fig. 12-22). It is believed that Pacioli invited Leonardo da Vinci to provide 60 illustrations, primarily of solid objects, to clarify the importance of the divine proportion. (Fig. 12-23). Leonardo's drawings are the first illustrations of geometric figures in the dual state of skeletonic (vacuum) and solid (solidum) (Fig. 12-24). This allowed for a distinct interpretation between front and back. Pacioli wrote of da Vinci's contributions to society by stating that "he is the most excellent painter in perspective, architect, musician and man endowed with all virtues."[71] Pacioli's text largely restates the earlier published work of the Roman architect Marcus Vitruvius Polio.[5] Within the text, Pacioli states the following:

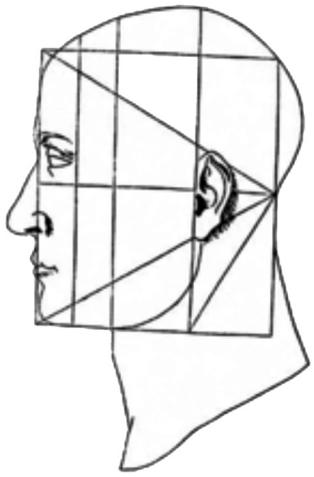
> *"The ancients, having taken into consideration the rigorous construction of the human body, elaborated all their works, especially their holy temples, according to these proportions; for they found here the two principal figures without which no project is possible: the perfection of the circle, the principle of all regular bodies, and the equilateral square."*

It is clear from a woodcut sketch included in *De Divina Proportione* (1509) that Luca Pacioli understood the importance of the golden ratio as it applies to the human face (Fig. 12-25). It is also believed that, at the request of Pacioli, Leonardo da Vinci took Vitruvius' written words and drew them out in a diagram that he called *Vitruvian Man* (1487; see Fig 12-17).[61] The image depicts a male figure in two superimposed positions with his arms and legs apart and simultaneously inscribed in a circle and a square. The



• **Figure 12-23** Drawings by Leonardo da Vinci confirming the golden ratio found in humans and other mammals. *From Royal Collection Trust/©Her Majesty Queen Elizabeth II 2013.*

• **Figure 12-24** *Virgin of the Rocks* by Leonardo da Vinci, confirming the golden ratio. *From Stamperia d'Arte Fratelli Alinari. Bibliotecca Reale, Turin, Italy. Photo Credit: Alinari/Art Resource, NY.*



• **Figure 12-25** *Woodcut Sketch* by Leonardo da Vinci, confirming the golden ratio.

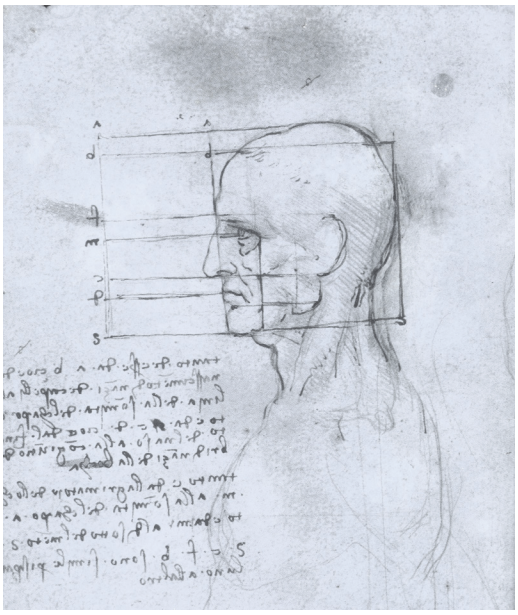
illustrated interpretation of Vitruvius' words is sometimes also called the *Canon of Proportions;* the original drawing is stored in the Galleria dell'Accademia in Venice, Italy. It is known that *Vitruvius described the human figure as being the principal source of correct proportions and symmetry among the classic orders of architecture*. da Vinci's *Vitruvian Man* beautifully demonstrates the blend of art and science envisioned by intellectuals during the renaissance, and it provides a clear understanding of the artist's keen interest in the proportions of the human form. Like Plato, da Vinci believed the workings of the human body to be an analogy of the workings of the universe. According to Leonardo himself, in a note that accompanied the drawing, "it was made as a study of the proportions of the male human body as described by Vitruvius" ("Palmus autem habet quattuor digitos").[61,72] Vitruvius' words, which Leonardo attempted to draw out in pen and ink on paper, are found in Vitruvius' *De Architectura* 3.1.2-3, which reads as follows:

> *For the human body is so designed by nature that the face, from the chin to the top of the forehead and the lowest roots of the hair, is a tenth part of the whole height; the open hand from the wrist to the tip of the middle finger is just the same; the head from the chin to the crown is an eighth, and with the neck and shoulder from the top of the breasts to the lowest roots of the hair is a sixth; from the middle of the breasts to the summit of the crown is a fourth. If we take the height of the face itself, the distance from the bottom of the chin to the underside of the nostrils is one-third of it; the nose from the underside of the nostrils to a line between the eyebrows is the same; from there to the lowest roots of the hair is also a third, comprising the forehead. The length of the foot is one-sixth of the height of the body; of the forearm, one-fourth; and the breadth of the breasts is also one-fourth. The other members, too, have their own symmetrical proportions,*

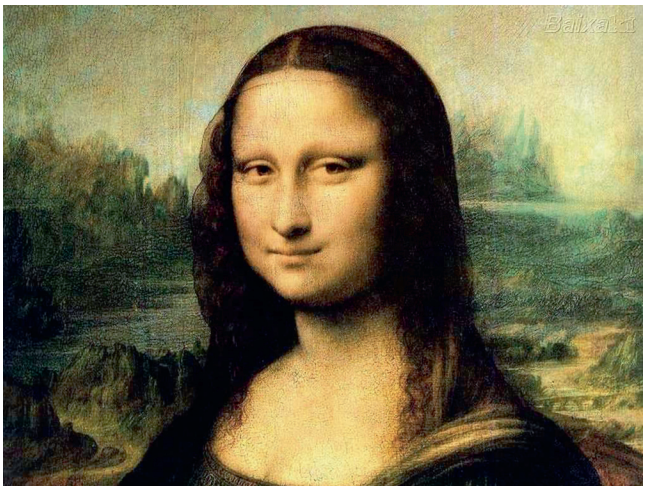
and it was by employing them that the famous painters and sculptors of antiquity attained to great and endless renown. Similarly, in the members of a temple, there ought to be the greatest harmony in the symmetrical relations of the different parts to the general magnitude of the whole. Then again, in the human body, the central point is naturally the navel. For if a man is placed flat on his back, with his hands and feet extended, and a pair of compasses centered at the navel, the fingers and the toes of his two hands and feet will touch the circumference of a circle described therefrom. Just as the human body yields a circular outline, so too a square figure may be found from it. For if we measure the distance from the soles of the feet to the top of the head, and then apply that measure to the outstretched arms, the breadth will be found to be the same as the height, as in the case of plain surfaces which are perfectly squared.[61,72]

For some scholars and artists of the High Italian Renaissance, the golden ratio was elevated from a simple mathematical formula to become a way of explaining natural phenomena and a tool to be used in the painting of objects and human forms.[66,93] By the mid 1400s, advances in technology allowed artists to use lenses, concave mirrors, and other methods to help them to create realistic-looking paintings. Many of today's critics believe that, simply by using their innate aesthetic sense, talented artists, architects and designers of that time period created art, built structures, and designed objects that closely approximate the golden ratio.[46] Skeptics often claim that the most attractive and harmonious objects were methodically designed by their artists to adhere to the golden ratio measurements; they also believe that many of the great artists' designs were simply superimposed on laid-out golden ratios. Interestingly, many other scholars deny that the Greeks (e.g., Euclid) made any direct aesthetic associations with the golden ratio. Thus, some questions remain unanswered: *Throughout antiquity and during the Renaissance, did artists consciously use the golden ratio as part of their technique? Did they model their work on precise mathematical dimensions, or was their aesthetic and artistic sense the guiding force that inevitably closely approximated the golden ratio?*

The analysis of Leonardo's drawing entitled *A Head of an Old Man* (Fig. 12-26) leaves little doubt that he was, at the very least, intrigued by various proportions of the face. This drawing shows how Leonardo used standard rectangles to determine dimensions in his paintings. Because classic art requires "harmony" and because we know that Leonardo collaborated with Pacioli and that he was believed to be the illustrator of *Divine Proportions*, Leonardo's use of the golden ratio in his paintings would be an obvious extension of his mathematic knowledge and artistic principles. Most of today's art critics place Leonardo's *Mona Lisa* on a short list of the most beautiful faces ever painted (Fig. 12-27). Debate continues regarding Leonardo's conscious use of the golden ratio in this well-known work. Interestingly, da Vinci is said to have revised the *Mona Lisa* for more than 10 years before his death without ever fully finishing the work to his own satisfaction.

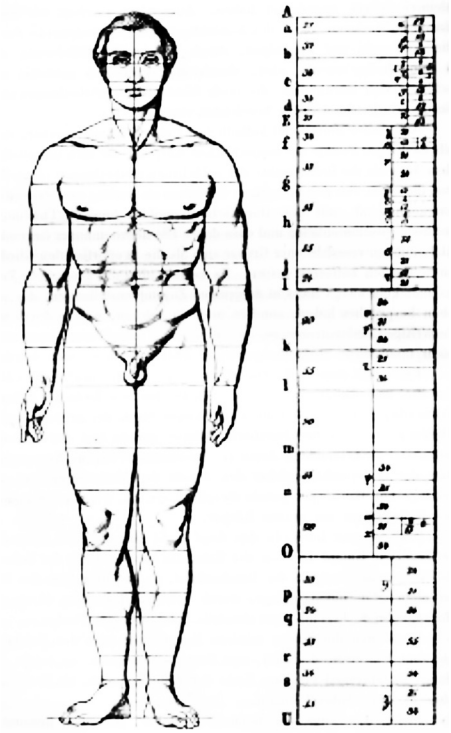


• **Figure 12-26** *Head of an Old Man* by Leonard da Vinci, confirming the golden ratio. *From Royal Library, Windsor Castle, Windsor, Great Britain. Photo Credit: Alinari/Art Resource, NY.*



• **Figure 12-27** *Mona Lisa* by Leonardo da Vinci.

After Leonardo, the golden ratio as an artistic principle likely fell from favor. To my knowledge, no prominent philosopher or artist was known to have actively used the golden ratio again until Adolf Zeising (1810-1876).[105] Zeising was formally trained in psychology, mathematics, and philosophy. He found the golden ratio expressed in all things, including in the skeletons of animals and the branching of their veins and nerves (Fig. 12-28). In connection with his theory involving golden-ratio–based human body proportions, Zeising (1854) wrote what he called the "universal law of proportions," "in which is contained the ground-principle of all formative striving for beauty and completeness in the realms of both nature and art, and

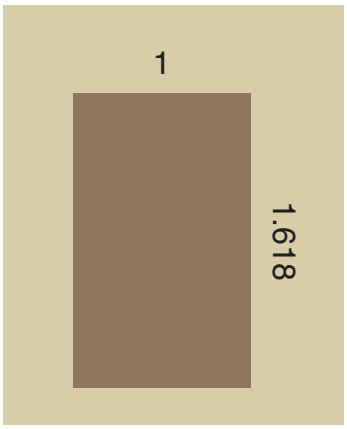


• **Figure 12-28** *Human Ratio* by Zeising, confirming the golden ratio.

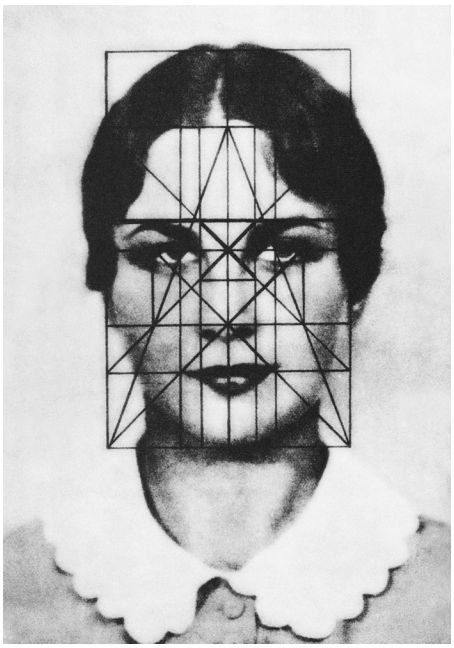
which permeates, as a paramount spiritual ideal, all structures, forms and proportions, whether cosmic or individual, organic or inorganic, acoustic or optical; which finds its fullest realization, however, in the human form."[34]

The famous German psychologist, Gustav Fechner, was inspired by Adolf Zeising's book *Der Goldene Schnitt* and began a series of inquiries to see if the golden rectangle had psychological aesthetic impact.[34] A golden rectangle is one with side lengths that reflect the golden ratio. A distinctive feature of this shape is that, when a square section is removed, the remainder creates another golden rectangle. In other words, it has the same proportions as the first. Another section can be removed infinitely, with the same results. Fechner's work was published in 1876. He made thousands of measurements of commonly seen rectangles (e.g., writing pads, books, playing cards, windows) and found that most were close to phi (Fig. 12-29). He also tested people's preferences and found that most people prefer the shape of the golden rectangle. Fechner's experiments were repeated and validated by others, including Witmar (1894), Lalo (1908), and Thorndike (1917).[34]

By the late 1800s, knowledge of and the conscious use of the golden ratio by painters, architects, and designers was well documented.[34] There are clear examples where these professionals have modeled their end products to conform with Euclid's golden ratio. In 1946 in *The Geometry of Art and Life,* Matila Ghyka presented an analysis of the face of the tennis champion Helen Wills.[34] He made several measurements of Wills' face, all of which were shown to be consistent with the divine proportion (Fig. 12-30). More recent examples include Salvador Dali's use of the golden



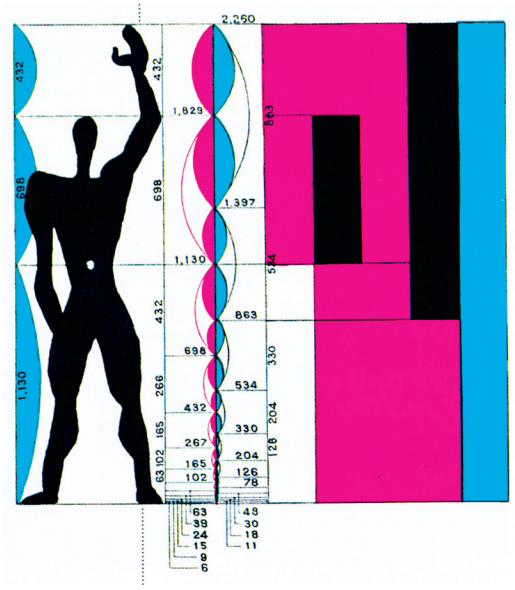
• **Figure 12-29** *Ideal Rectangle* by Fechner, confirming the golden ratio.



• **Figure 12-30** Facial proportions of Helen Wills by Matila Ghyk, confirming the golden ratio. From Matila Ghyk: *The Geometry of Art and Life,* 1946, Kessinger Publishing, Oct. 30, 2004, p. 102.

ratio in his masterpiece *The Sacrament of the Last Supper* (1955; Fig. 12-31) and the experimental piece of music *Binary Universe* (2006) by Brian Transeau. A strong advocate for the conscious application of the golden ratio to art, the design of objects, and architecture was *Le Corbusier* (1887-1965).[32,59] His legacy remains one of the most influential in modern architecture. He developed an intense interest in proportional systems and their role in all forms of aesthetics. Le Corbusier's fascination with the importance of the golden ratio in aesthetics centered around his belief that basic forms and structures are the underpinning of natural phenomenon. He developed what he called the *Modulor* in the tradition of Vitruvius with the intention of improving both the appearance and function of all manmade objects and architecture (Fig. 12-32).[59] He was also a strong believer in the idea that ideal harmony in music (as stated

• **Figure 12-31** *The Sacrament of the Last Supper* by Salvador Dali, confirming the golden ratio. *Copyright Salvador Dali, Fundacio Gala-Salvador Dali, Artists Rights Society (ARS), New York, 2013.*

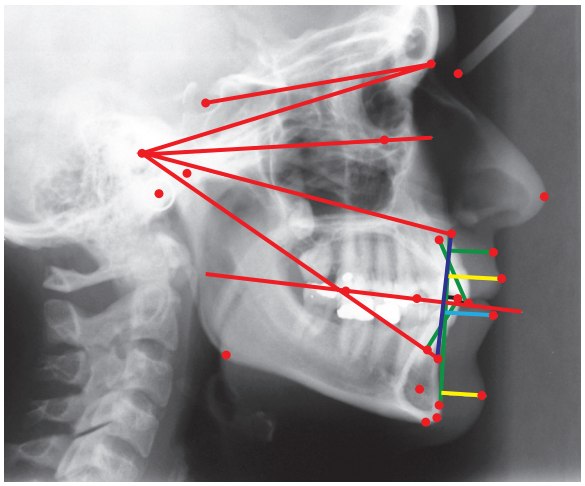


• **Figure 12-32** *Modular Man* by Le Corbusier, confirming the golden ratio. *Copyright 2013 Artists Rights Society (ARS), New York/ADAGP, Paris/F.L.C.*

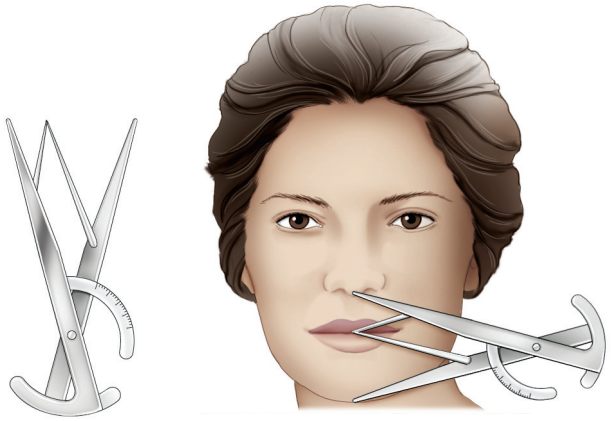
by Pythagoras) could only be achieved through the application of the golden ratio to numbers.

Current Philosophy

From Pythagoras, Polykleitos, Plato, Aristotle, Euclid, Vitruvius, Galen, Fibonacci, Piero, da Vinci, and Pacioli as well as the more recent Le Corbusier and all of their disciples for more than 2.5 millennia, we have learned that the proportions of the parts to the whole affect the observers' impression of beauty and attractiveness.[1,3,7,23,26,29,36,56,62,82,96] From the premier artists, architects, and designers throughout the centuries, we know that mirror-image symmetry and the harmony of proportions matter when judging facial aesthetics. They have also shown by example that the precise—even to the millimeter—achievement of an exacting mathematical ratio is not sufficient or even preferred for



• **Figure 12-33** Skeletal proportions according to cephalometric analysis.



• **Figure 12-34** Human face with use of the Rickett's divider, confirming the golden ratio.

the creation of the most important end products; rather, an approximation of the golden ratio is.

In the realm of facial aesthetics and reconstructive surgery, clinicians from all involved fields have from time to time attempted to match facial aesthetics to a strict mathematical model (e.g., normative cephalometric measurements) without always considering individual biologic variations and aesthetic nuances* (Fig. 12-33). When doing so, they have often fallen short of their objective. In 1984, Ricketts reported about a significant number of golden proportions when analyzing the facial skeleton and overlying soft tissues.[83-91] He was a strong advocate of the use of these divine proportions as a guide for planning the correction of dentofacial deformities via orthognathic surgery (Fig. 12-34). The validity of Ricketts' approach to facial analysis for the correction of jaw deformities has been questioned by some while supported by most.[8,12,15,30,50,73,74,75,76,94] For the clinician who is asked to correct a congenital or developmental craniofacial or

*2, 4, 10, 11, 16, 21, 22, 24, 28, 31, 33, 37, 38, 51, 52, 60, 64, 65, 70, 78, 79, 95, 103, 104, 108, 153, 177, 200, 241, 246

maxillofacial deformity, attention is directed to both form and function. As R. Buckminster Fuller stated, "rarely are the two objectives divergent."[61] For the achievement of a harmonious face, attention is directed toward the practical objectives of improving the upper airway and the occlusion, which will affect speech, swallowing, chewing, and breathing. Facial aesthetic perfection is not a specific millimeter distance or degree of angulation but rather a visual impression at conversational distance in three-dimensional life. At the same time, neither divine proportions nor biologic realities and the patient's personal preferences can be ignored throughout the process.

The rediscovery of the importance of achieving an approximation of Euclidean proportions and mirror-image symmetry as a treatment objective in the correction of dentofacial deformities remains an essential step in the education of all students of the human face.

Part II: Clinical Evaluation of the Patient with a Dentofacial Deformity

Background

In the correction of a dentofacial deformity, the clinician's aesthetic sense and knowledge of patient-specific biologic and psychosocial factors must be condensed into precise millimeter measurements and angular changes that are then accomplished on the surgically sectioned (osteotomized) maxilla, mandible, and chin region with a goal of establishing *Euclidean facial proportions* and *improved function*.* Although there is no singular normative table of numbers or a formula that can be relied on to direct the surgeon to the *golden ratio,* a methodical approach is nevertheless required. The clinician's vision is based on 1) fundamental aesthetic principles 2) the biologic realities of the tissues 3) patient-specific dysfunctions (e.g., chewing, swallowing, speech articulation, breathing, lip closure/posture) and 4) individual treatment preferences (e.g., timing, techniques, extent of intervention).† It is expected that a degree of uneasiness about the end result will be felt by both the operating surgeon as well as the patient and his or her family during this process. With clinician experience, this creative tension for the surgeon and the uncertainty of the patient and his or her family are reduced; however, these can never be fully alleviated before surgery.

At the time of the preoperative, direct, face-to-face planning examination of the patient by the operating surgeon, several evaluations will have already been carried out.[118,136,193,199,197,247] A review of relevant medical data in combination with facial photographic, radiographic, and dental analysis should be fresh in the surgeon's mind. The patient's facial structures are examined while he or she is upright and sitting in the *natural head position* (NHP). This is considered the most rational, physiologic, and anatomic orientation of the head and neck for the evaluation of the face, jaws, and teeth.* This is generally not the head and neck position in which the patient with a dentofacial deformity will naturally relax. Rather, the clinician must place the patient in the NHP. The teeth are also placed in *centric relation or with a normal freeway space,* and the *lips are relaxed* (i.e., not pursed together). The clinician then observes the patient three dimensionally at different angles in both repose and with dynamic facial expressions. *It is from the NHP that the surgeon makes crucial decisions regarding what facial features are abnormal and the extent of required surgical reorientation*.

Global Head and Neck Assessment: Seven Critical Aspects

1. *Quality of the overlying soft-tissue envelope of the head and neck.* The surgeon must recognize distortions of the soft-tissue envelope that will self-correct after the skeleton is reconstructed versus intrinsic soft-tissue malformations and deformities that will not (e.g., hypoplasia, hyperplasia, effects of trauma and aging).
2. *Symmetry and harmony of the upper facial skeleton (i.e., cranial vault, orbits, nasofrontal process, and zygomatic arches).* Although most individuals with dentofacial deformities have relatively normal upper facial skeletons, some do not (i.e., those with hemifacial microsomia or Treacher Collins syndrome).
3. *Morphology of unique aesthetic units of the face (e.g., external nose, external ears, periorbital soft tissues).*
4. *History of temporomandibular joint symptoms and signs and the documentation of any limitations in the mandibular range of motion.*
5. *History of cervical spine symptoms and signs and the documentation of the safe neck range of motion.*
6. *Symmetry and harmony of the lower facial skeleton (i.e., maxilla, mandible, and chin region).* The patient with a dentofacial deformity will have significant variations from normal in this region of the craniofacial skeleton.
7. *Dental rehabilitative needs (i.e., restorative and periodontal) as well as orthodontic aspects.*

Evaluation of the Overlying Soft-Tissue Envelope

An individual with a routine developmental jaw disproportion will generally have a normal overlying soft-tissue envelope that is simply distorted by the underlying skeletal disharmony. Alternatively, normal variations in the

*107, 111, 112, 122, 125, 138, 139, 161, 162, 166, 206, 207, 210, 215, 219, 223, 228, 229, 240, 248, 249

†114, 115, 116, 123, 127, 128, 129, 143, 144-150, 158, 174, 186, 189-191, 201-204, 208, 211, 222

*24, 124, 126, 133-135, 151, 168, 172, 171, 177, 181, 180, 184, 185, 225-227, 234, 238, 239, 243

thickness and quality of the skin, density of sweat glands, fat distribution, and degree of pigmentation are to be expected.

An example of soft-tissue distortion (not deformity) is seen with maxillomandibular deficiency (see Chapter 23). The impression of "chubby cheeks" and excess fat in the neck is typical. With maxillomandibular (skeletal) advancement and vertical lengthening, the normal facial contours and curvatures will be restored.

There are exceptions with which the soft-tissue envelope is directly affected by a malformation (e.g., Treacher Collins syndrome, hemifacial microsomia, cleft lip, hemifacial hypertrophy), previous trauma (e.g., burns, lacerations), or scarring after infection. When a jaw deformity is associated with a syndrome (see Chapters 27 through 31), clefting (see Chapters 32, 33, and 34), or a soft-tissue traumatic deformity (see Chapter 35), the soft-tissue envelope may require special consideration.

Evaluation of the Upper Facial Skeleton

For most individuals who present with dentofacial deformities, the upper facial skeleton (i.e. cranial vault, orbits, and zygomas) will be reasonably symmetric and proportionate. There are exceptions that result from 1) congenital anomalies (e.g., craniofacial microsomia, Treacher Collins syndrome, Apert syndrome, Crouzon syndrome), 2) traumatic events (e.g., molding during infancy, torticollis, previous facial bone fractures with malunion), and 3) tumors (e.g., fibrous dysplasia, neurofibromatosis).

With routine dentofacial deformities, minor upper face skeletal variations from normal are expected; they may include differences in the convexity of the forehead (i.e., the anterior cranial vault) or the prominence of the supraorbital ridge (i.e., anterior frontal sinus wall bulging). Minor mirror-image asymmetries of the upper face are also common (e.g., external ear position, eyebrow height, cranial nerve VII dysfunction). When present, facial variations and asymmetries should be documented through medical-quality photographs and then discussed with the patient and his or her family from the outset.

In the presence of a dentofacial deformity the cheeks and cheekbones may appear bigger or smaller than they actually are. The correction of the jaw deformity toward Euclidian proportions is likely to change the observer's impression. An example of this phenomenon is seen in the patient with maxillary deficiency in combination with relative mandibular excess (Fig. 12-35, *K* & *L*). When the maxilla is advanced and rotated clockwise in conjunction with mandibular ramus osteotomies, the apparent flat cheekbones, deficient infraorbital rims, and enlarged nose typically disappear (see Chapter 20).

Evaluation of Unique (Discrete) Facial Aesthetic Units

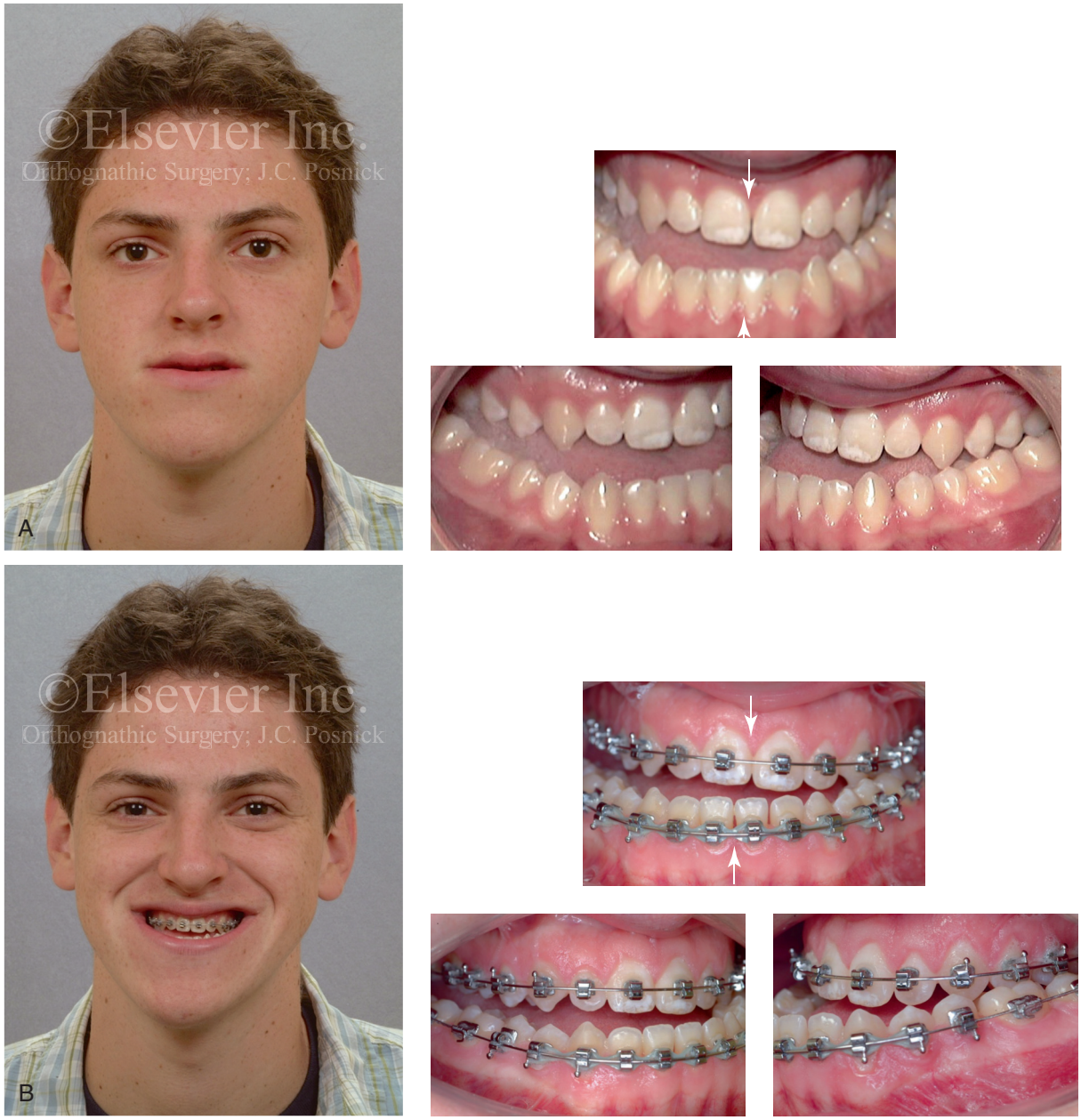
There are discrete aesthetic units of the face that stand out and that should be independently considered, including the periorbital (eyelid) regions, the external ears, and the nose.

The *periorbital (adnexal) tissues* may be of special concern as a result of 1) congenital hypoplasia (e.g., Treacher Collins syndrome, hemifacial microsomia, upper eyelid ptosis), 2) previous trauma (e.g., medial or lateral canthal injury, nasolacrimal apparatus injury, upper or lower eyelid injury), or 3) the normal aging process (e.g., descent, deflation, ptosis). The individual's eyelid appearance may either add a sparkle to or be a distracting focal point of the face. For example, distortions of the lower eyelids are generally seen in patients with dentofacial deformities that are associated with maxillary deficiency and relative mandibular excess (i.e., negative vector relationship of the eyeballs and lower lids). This negative vector relationship affects the aesthetic appearance of the midface, which only worsens with age (see Chapter 25 and Fig. 12-35, *D*). Maxillary horizontal advancement and clockwise rotation in conjunction with mandibular ramus osteotomies corrects the skeleton and has positive effects on the soft tissues of the eyelids and the midface contours (see Chapter 20 and Fig. 12-35, *K* & *L*).

The *external ears* display considerable variations in size, shape, extent of protuberance, and asymmetry (see Chapter 39). Forensic pathologists consider details of auricular morphology to be as unique to the individual as that individual's fingerprints. In association with specific syndromes (e.g., hemifacial microsomia, Treacher Collins syndrome), there may be auricular hypoplasia or a malformation that ranges from total microtia to cupping to just an abnormal location of the ears on the side of the face (see Chapters 27 and 28). Significant disproportion of the external ears, as a standalone and distinct feature, can be a distracting aesthetic aspect in the presence of an otherwise attractive face. An external ear set-back procedure may be carried out as part of the orthognathic surgery or later as a separate operation (see Chapter 39).

The *external nose* will display a wide range of aesthetic variation due to unique bony, cartilaginous, and soft-tissue anatomy (see Chapter 38). In the presence of a dentofacial deformity, the middle third of the central face (i.e., the nose) may appear bigger or smaller than its actual size (Fig. 12-35, *L*). The correction of the presenting jaw discrepancy toward Euclidean proportions will improve harmony between the upper, middle, and lower thirds of the face. After this is accomplished, a more accurate assessment of the actual nasal aesthetic unit can be made. Disproportion of the nose as a standalone aesthetic unit may nevertheless exist and benefit from alteration (see Chapter 38). Dysfunction of the nose characterized by chronic obstructive nasal breathing and inadequate sinus drainage should also be recognized and dealt with at the appropriate time (see Chapter 10). Clarification of these issues with the patient and his or her family at the first consultation visit as well as additional discussion at the time of orthognathic planning and again after surgical healing is essential to achieve patient satisfaction (see Chapter 7).

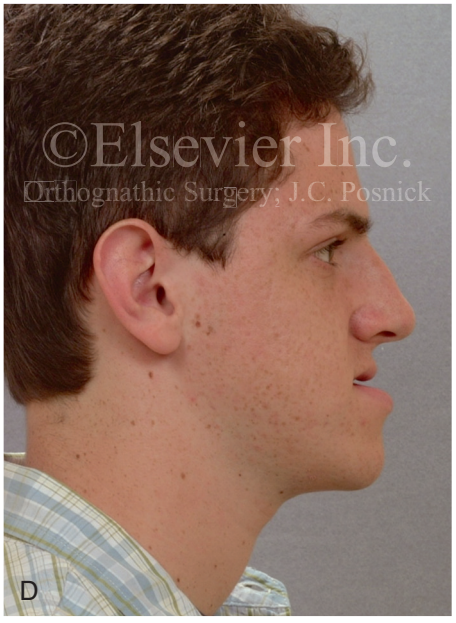
Text continued on p. 360



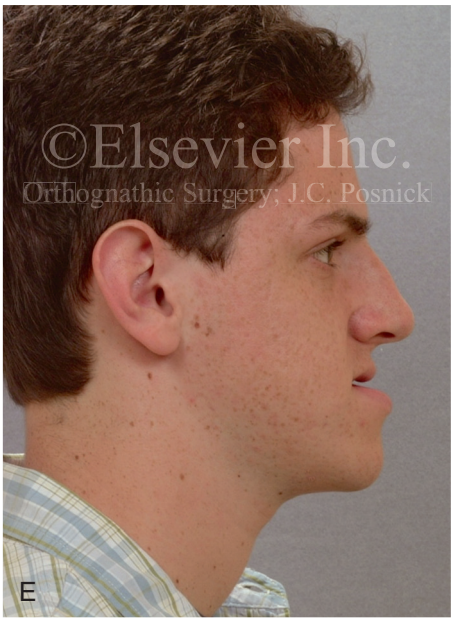
• **Figure 12-35** A 17-year-old boy with a diagnosis of left hemimandibular elongation. This dentofacial deformity affects the patient's chewing, swallowing, speech articulation, lip closure/position, and breathing. He has normal cervical spine function and neck range of motion. He has no temporomandibular disorder, and he has a full range of mandibular motion. The upper facial skeleton (i.e., the cranial vault, the orbits, and the zygoma) is symmetric and with normal proportions. The facial soft-tissue envelope is distorted by the maxillomandibular deformity but without malformation. During his early teenage years, the patient underwent an initial phase of orthodontics without success. He was referred for surgical evaluation and then underwent a combined orthodontic and surgical approach. With the relief of dental compensation, his surgery included a maxillary Le Fort I osteotomy in segments (horizontal advancement, cant correction, vertical change, arch expansion, and clockwise rotation); bilateral sagittal split ramus osteotomies (cant correction and mandibular straightening); osseous genioplasty (straightening and minimal advancement); and septoplasty/inferior turbinate reduction. **A,** Frontal facial view in repose before treatment. Occlusal views before orthodontic retreatment are also shown. **B,** Frontal view with smile before surgery. Occlusal views with orthodontic (dental) decompensation in progress.

Continued

Maxillary Surgical Plan		
Frontal View:		
Maxillary midline	No change	
Cant correction	4 mm	−2 mm (L) +2 mm (R)
Anterior vertical	+2 mm	

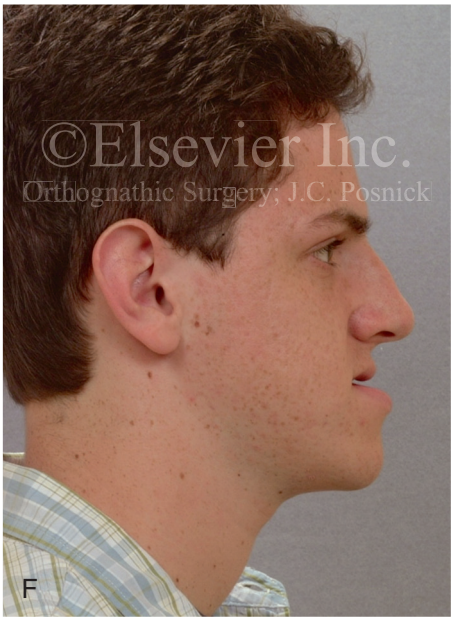


• **Figure 12-35, cont'd C,** The planned surgical repositioning of the maxilla is clarified during the preoperative direct visual examination. Frontal view changes will include no maxillary dental midline change; 4-mm cant correction (up 2 mm at the left first molar and down 2 mm at the right first molar); and a 2-mm vertical lengthening at the incisors. **D,** Profile facial view before treatment. Lateral cephalometric radiograph before treatment.



Maxillary Surgical Plan	
Profile View:	
Anterior vertical	+2 mm (lengthen)
Posterior vertical	0 mm
Horizontal	+8 mm (advance)

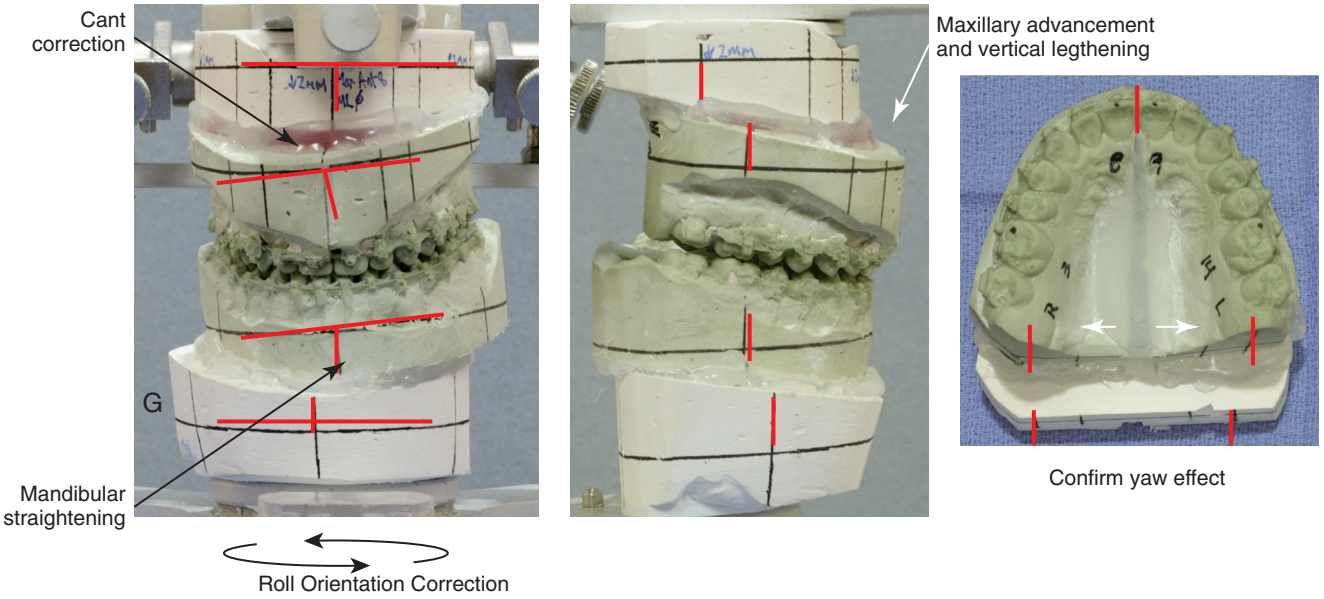
Maxillary clockwise rotation (2 mm) to enhance profile aesthetics



Maxillary Surgical Change	
Horizontal change at incisors	+8
Vertical change at incisors	+2
Midline change at incisors	0
Cant correction -*TOTAL*	4
↓ +2 on right ↑ −2 on left	
Maxillary plane change -*TOTAL* (Clockwise) Counterclockwise	2
Vertical change at T#3 (right) molar (Base + Cant + Plane)	+2 +2 −2 = +2
Vertical change at T#14 (left) molar (Base + Cant + Plane)	+2 −2 −2 = −2

• **Figure 12-35, cont'd E,** The planned maxillary repositioning is clarified during the preoperative direct visual examination. Profile view changes will include vertical lengthening of the incisors (2 mm); horizontal advancement at the incisors (8 mm); and maxillary clockwise rotation (2 mm). **F,** The surgical plan for the repositioning of the maxilla is indicated on the standard orthognathic surgery model-planning data sheet. The data is then relied on to reorient the maxilla on the articulator with use of the Erickson Model Table. This is accomplished before splint construction.

Continued



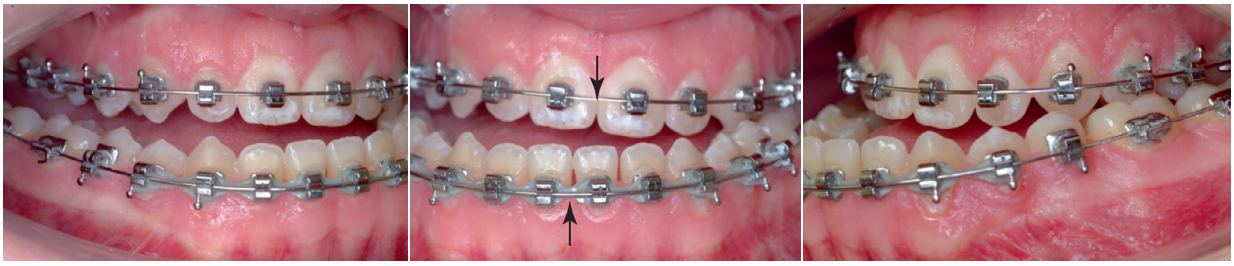
• **Figure 12-35, cont'd G,** Articulated dental casts indicate analytic model planning (see Chapter 13). **H,** Illustration of roll, yaw, and pitch orientation. Consideration of reorienting the maxilla in accordance with these parameters is an essential part of planning. **H** *from Ackerman JL, Proffit WR, Sarver DM, et al: Pitch, roll, and yaw: describing the spatial orientation of dentofacial traits,* Am J Orthod Dentofacial Orthop *131:305–310, 2007.*

• **Figure 12-35, cont'd I,** Frontal views in repose before and after reconstruction. **J,** Frontal views with smile before and after reconstruction.

Continued

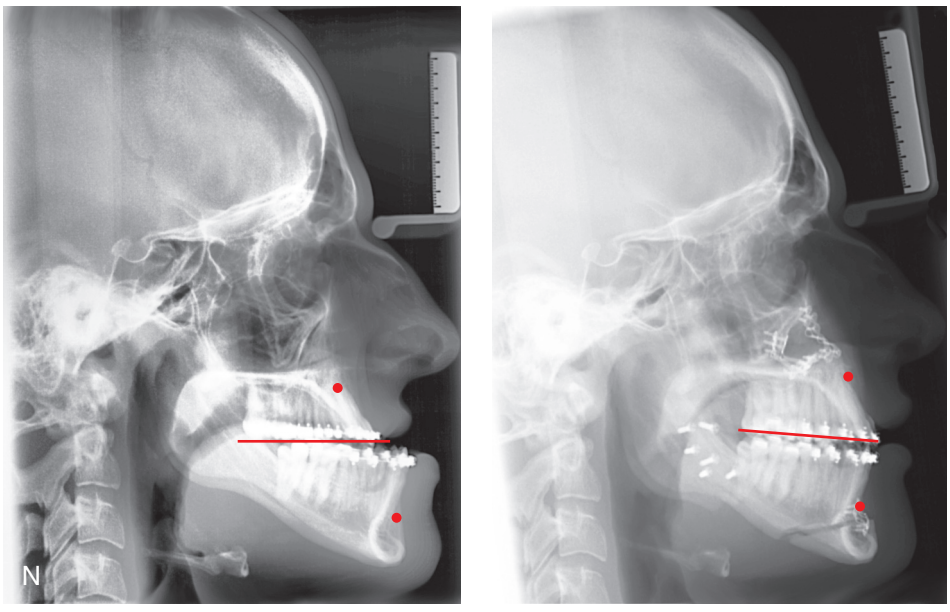
• **Figure 12-35, cont'd K,** Oblique facial views before and after reconstruction. **L,** Profile views before and after reconstruction.

Prior to retreatment



Pre surgery

4 years after treatment



• **Figure 12-35, cont'd M,** Occlusal views before retreatment, with orthodontic decompensation in progress, and 4 years after treatment. **N,** Lateral cephalometric radiographs before and after surgery.

Assessment for Temporomandibular Disorder and Current Mandibular Range of Motion

An accurate history of any past and current temporomandibular disorder symptoms and signs should be documented, including any previous therapy; subjective complaints; joint noise; mandibular range of motion; any centric relation–centric occlusion discrepancy; masticatory muscle tenderness to palpation; and current radiographic findings. Any active temporomandibular disorder will require treatment to diminish symptoms and to achieve adequate range of motion before orthodontic treatment is initiated (see Chapter 9).

Assessment of the Cervical Spine and Current Neck Range of Motion

An accurate history of past and current cervical spine symptoms and signs should be documented. Individuals with a dentofacial deformity are at higher-than-average risk for cervical spine anomalies. If the suspicion of cervical spine dysfunction exists, consultation with an appropriate specialist (i.e., neurologist, orthopedic surgeon, neurosurgeon) and the obtaining of focused imaging studies (i.e., magnetic resonance imaging, computed tomography scanning, plain radiographs) should be carried out. Confirming the safe range of motion of the neck with the patient awake in the office setting and understanding any limitations is important for safe orthognathic surgery and recovery.

Evaluation of the Lower Facial Skeleton

Confirmation that the upper facial skeleton and the soft-tissue envelope of the face do not require surgical alteration and that the neck and mandible have an intact safe range of motion without significant limitations or discomforts is essential before focusing on detailed maxillomandibular surgical planning.[142]

Qualitative decisions concerning which components of the maxillomandibular skeleton are asymmetric or disproportionate and thus require surgical repositioning will have already been discussed with the patient, his or her family, and other treating clinicians before the initiation of treatment. These details should be reconfirmed by the orthognathic surgeon during the immediate preoperative planning visit.[120,121,130,131,132,152,153,155,170]

Assuming that both the maxilla and the mandible are part of the dentofacial deformity that requires reconstruction, quantitative (to the millimeter) decisions of the preferred aesthetic and best functional repositioning of the upper jaw (at the time of the Le Fort I osteotomy) must first be made.* When thinking through the details of maxillary surgical repositioning, it is understood that the "new" upper jaw position (in two-jaw surgery) will serve as the platform on which the mandible is to be placed. Where the lower jaw ends up is important and will be entirely dependent on the new surgical position of the maxilla and the orthodontic alignment of the teeth.[159,160,187,195,196,198,205,212,213,214,216-221,235-237,240]

Evaluation of Overall Dental Rehabilitation Needs

Recognizing when a patient who is seeking orthognathic surgery has additional dental needs beyond standard orthodontics is essential to the achievement of a favorable outcome. The need for *periodontal evaluation and treatment* as well as the need for *complex dental restorative work* should be considered for every patient before the initiation of a coordinated orthodontic and jaw surgical approach. This is especially true for the individual who has suffered dental and periodontal sequelae from a longstanding uncorrected dentofacial deformity and who may have undergone camouflage treatment in an attempt to neutralize the occlusion (e.g., orthodontics, occlusal equilibration, restorative dentistry) (see Chapters 5 & 6).

Detailed Maxillomandibular Surgical Planning: Eight Critical, Quantitative, To-the-Millimeter Decisions

1. Assess the current and preferred position of the *maxillary dental midline* with reference to the upper face (i.e., yaw orientation). Anticipate any potential yaw effect of the new surgical maxillary orientation on the mandible and the midface (i.e., the cheeks) (see Fig. 12-35, *B*).
2. Assess for the presence of any *maxillary cant* (i.e., roll orientation) with reference to the upper face. Determine the preferred upper jaw repositioning for cant correction (see Fig 12-35, *B*).
3. Assess the current and preferred *vertical position of the maxillary incisor crown* with reference to the upper face. Consider the upper-lip–incisor relationship in both repose and smile (see Fig 12-35, *A* and *B*).
4. Assess the current and preferred *horizontal position of the maxillary incisor crown* with reference to the upper face (see Fig 12-35, *D*).
5. Assess the current and preferred *A-point to B-point relationship of the jaws* (i.e., pitch orientation). When the face is viewed in profile in the NHP, consider the need to differentially alter the vertical maxillary height at the first molars and the incisors (i.e., maxillary plane change) to achieve specific objectives (see Fig 12-35, *D, E,* and *F*).
6. Assess the *baseline pyriform rim, floor of nose, and anterior nasal spine morphology.* Anticipate the location of each after midface reorientation, including any planned maxillary plane change. Consider the advantages of the recontouring of the pyriform rims, the floor of the nose,

*106, 109, 110, 113, 117, 119, 137, 140, 141, 156, 157, 163-165, 167-169, 170, 175, 176, 179, 182, 183, 188, 194, 224, 230-233, 242, 244, 245

and the anterior nasal spine to achieve airway and aesthetic objectives (see Fig 12-35, *D, E,* and *F*).

7. Assess the *baseline chin morphology.* Anticipate the location of the pogonion after jaw reorientation that may also include mandibular plane change (i.e. pitch orientation). Consider the advantages of chin reshaping (i.e., osseous genioplasty) to achieve the preferred horizontal and vertical aesthetics and shape of the chin (see Fig 12-35, *D, E,* and *F*).
8. Reconfirm the advantage of and *need for the segmentation of the maxilla* to achieve the preferred arch form and occlusion with the mandible (see Fig 12-35, *G*).

Assess the Current and Preferred Position of the Maxillary Dental Midline (i.e., Yaw Orientation) with Reference to the Upper Face

After the decision has been made to surgically reposition the maxilla and the mandible (i.e., two-jaw surgery), no additional risk is encountered by making a change in the *maxillary dental midline position*. In fact, the surgeon must make a conscious decision to either leave the midline as it is or to adjust it. During the immediate preoperative, direct, face-to-face examination, the operating surgeon should consider the preferred location of the maxillary dental midline.[10,117,120,167,176,183] This is a clinical observation when viewing the patient at conversational distance and closer up. It is measured in millimeters of variation of the current maxillary dental midline position (i.e., the point between the incisors at the incisal edge) in comparison with the baseline upper facial midline.

Ideally, the midline between the maxillary central incisors should coincide, within 2 mm, with the facial midline. When the upper face has satisfactory basic symmetry, the decision of where to position the maxillary dental midline is generally straightforward (see Fig. 12-35, *C*). In the presence of upper face asymmetry—especially in combination with canting of the maxilla and asymmetry or disproportion of the mandible (see Chapter 28)—the decision of where to reposition the maxillary midline becomes more complex.

When the surgeon determines the preferred (new) location of the maxillary dental midline, the *yaw effect of the midline change* must also be taken into account (see Fig. 12-35, *G*). For example, the maxilla may be either bodily moved to one side to correct the incisor midline, or the maxillary dental midline maybe corrected while leaving the buttress regions (i.e., side to side) essentially unchanged. This would require a *twisting* of the maxilla rather than a *bodily* movement so that the posterior maxilla moves slightly anterior on one side while the other side moves slightly posterior. This is a similar movement to the twisting open of a lid on a jar. As stated, a twisting movement of the maxilla around the vertical axis of rotation (i.e., yaw orientation) results in an incisor midline shift and a simultaneous anterior-posterior shift at the molars. When twisting the maxilla, the resulting posterior shift will cause a relative Class III on one side and a Class II molar relationship on the other side. Alternatively, a lateral (side-to-side) shift of the maxilla would cause a different change in the yaw orientation (see Fig. 12-35, *H*).

Establishing the correct maxillary dental midline position and yaw orientation is especially of concern in cases that present with moderate to severe three-dimensional facial asymmetry of multiple bones (i.e., hemifacial microsomia). A three-dimensional computed tomography scan (i.e., from the top of the skull through bottom of the chin) can be helpful to appreciate where specific mirror-image dysmorphology exists and how best to manage it (see Chapter 27 & 28).

During orthognathic surgery, the introduction of yaw effects may later be visualized as unwanted facial asymmetry in the following areas:

- The *maxillary incisor midline* as compared with the upper facial midline
- The *cheek region,* with fullness on one side and deficiency on the other
- The *angle of the mandible region,* with fullness on one side and deficiency on the other

Assess for the Presence of Any Maxillary Cant (i.e., Roll Orientation) with Reference to the Upper Face

When the decision has been made to surgically reposition both the maxilla and the mandible (i.e., two-jaw surgery), no additional risk is encountered when correcting any observed *cant (i.e., roll orientation)* of the maxilla. The operating surgeon is forced to make a conscious decision to either leave the roll orientation (cant) as is or to alter it and, if so, by how much. Studies confirm that a 3-degree occlusal cant is perceived as a noticeable asymmetry by 50% of both professionals and lay people, whereas a 4-degree cant is noticeable by 90% of professionals and lay people.

During the preoperative direct planning examination, the clinician should make an observation when viewing the patient face to face of whether the maxilla is vertically even (i.e., from side to side) or, if not, of how much of a deviation (cant) is present.[109,117,141,157,192,231] What is generally considered a maxillary cant (i.e., vertical height discrepancies of the maxilla from side to side) may also be viewed from the perspective of the *aesthetic line of the dentition* or at the *functional line of the occlusion* (see Fig. 12-35, *J*). Maxillary cant considerations take into account the upper face (i.e., the structures above the maxilla). When the upper face is basically symmetric, the interpupillary line may be used as an accurate baseline reference (see Fig. 12-35, *B* and *C*).

The presurgical planning visit is a good opportunity for the surgeon to clarify and discuss directly with the patient and his or her family both major and minor presenting facial asymmetries. This can be done by viewing a medical-quality frontal facial photograph or while the patient is viewing himself or herself with a handheld mirror. Typical

minor asymmetries include differences in the height (position) and shape of the external ears, the eyebrows, and the lateral canthi as well as asymmetries in cheek and lip (i.e., soft-tissues) elevation during smiling.

The quantifying of any presenting maxillary cant and the extent of correction planned (to the millimeter) may be complicated by upper facial skeletal asymmetries; other directional abnormalities of the maxilla (e.g., transverse, horizontal, or dental midline to facial midline); and any asymmetry or disproportion of the mandible. When present, these compounding factors make it difficult to accurately predict the most aesthetically pleasing outcome.

Thought is simultaneously given to the *yaw orientation* (i.e., rotation around the Y axis) of the presenting maxillary deformity and the yaw effect at the level of the mandibular angles and the midface (cheek) regions anticipated as a result of the (planned) maxillary change (see Fig. 12-35, *G*). In complex facial asymmetry cases, the clinician's ability to simultaneously analyze the presenting yaw and roll orientations and the subsequent aesthetic changes of the planned correction can be augmented with the use of craniofacial three-dimensional computed tomography scan imaging.

The clinicians' assessment of the extent of maxillary cant and the desired correction may be facilitated by the following:

1. Use of a *Fox plane* during the direct visual examination. The placement of a Fox plane directly over the occlusal surfaces of the maxillary dentition is helpful to judge the presence and extent of maxillary cant as compared with the upper face (i.e., the interpupillary reference line). Soft wax can be added to the deficient side (i.e., wax can be placed in between the Fox plane and the occlusal surfaces of teeth) until the cant is corrected. The millimeter amount of wax buildup (e.g., at the first molar) is a measure of the cant. When using a Fox plane, the clinician must also consider the patient's maxillary arch form. Often, two separate dentoalveolar planes (i.e., in the presence of a steep curve of Spee) have been accentuated by the orthodontic mechanics and will be managed with segmental osteotomies. How the Fox plane is seated to the patient's maxillary occlusal plane is important when assessing for a cant.
2. Comparison measurements from a *posterior-anterior cephalometric radiograph* (left side to right side)
   - Incisal edge to nasofrontal suture (millimeter distance, left and right sides)
   - Canine edge to nasofrontal suture (millimeter distance, left and right sides)
   - First molar edge to frontozygomatic suture (millimeter distance, left and right sides)
3. Comparison measurements from a *three-dimensional craniofacial computed tomography scan* (left side to right side)
   - Incisal edge to nasofrontal suture (millimeter distance, left and right sides)
   - Canine edge to nasofrontal suture (millimeter distance, left and right sides)
   - First molar edge to frontozygomatic suture (millimeter distance, left side and right side)
4. Comparison measurements taken directly from the *anthropometric (facial) surface* (left side to right side)
   - Incisal edge to medial canthus (millimeter distance, left and right sides)
   - Canine edge to medial canthus (millimeter distance, left and right sides)
   - First molar edge to lateral canthus (millimeter distance, left and right sides)

Assess the Current and Preferred Vertical Position of the Maxillary Incisor Crown with Reference to the Upper Face

The relationship between the upper lip and the maxillary incisors represents a key facial aesthetic landmark. Ideally, with the upper lip at rest, several millimeters (i.e., 1 mm to 3 mm) of the lower aspect to the maxillary incisor crowns should be seen in frontal view. With a broad smile, the upper lip elevates and, ideally, the entire maxillary incisor crown and at least a few millimeters of the gingiva will be seen. This will vary when evaluating a *posed smile* (as for a photograph) versus a *dynamic smile* (as seen when laughing).* The *aesthetic line of the dentition* follows the surfaces of the maxillary anterior and posterior teeth. The *functional line of occlusion* takes into account arch form, arch width, and symmetry. In the presence of malocclusion—as a result of distortions in arch width, curve of Spee, and overall arch form—the functional line of occlusion may not be even or on a single plane (see Fig 12-35, *J*). With many of the typical developmental jaw deformities, the *lip-to-tooth relationship* (i.e., the aesthetic line and the functional line) will show variation from ideal. Affected individuals will benefit from differential surgical repositioning of the anterior and posterior maxilla (i.e., intrusion or lengthening) as part of the surgical correction not only to positively influence the occlusion itself but also to influence the aesthetic line of the dentition and the functional line of occlusion.

Through a Le Fort I osteotomy with vertical intrusion, less gingiva with smile and incisor crown height in repose will be seen. With surgical (vertical) lengthening of the upper jaw during the Le Fort I osteotomy, more dental show and gingival exposure are to be expected (see Fig 12-35, *I* and *J*). Unfortunately, even with a pure vertical vector change of the maxilla, the upper lip soft-tissue response is generally less than a one-to-one relationship and not entirely predictable. Soft-tissue predictions become even less accurate in combination with other upper jaw vector changes (i.e., horizontal, and transverse); in the presence of maxillary asymmetries (i.e., cant and dental midline shifts); when other procedures are simultaneously carried out (e.g., mandible and chin osteotomies, pyriform rim/nasal floor/

*References 106, 110, 130, 131, 137, 140, 152, 173, 179, 194, 209, 218, 220, 230, 233, 244, 250

anterior nasal spine recontouring); and in the presence of a hypotonic upper lip (e.g., longstanding skeletal Class III malocclusion) or an immobile scarred upper lip (e.g., fibrosis after cleft lip repair).

Nevertheless, with thoughtful planning, a favorable aesthetic outcome involving the relationship of the *incisor vertical position to the upper lip* should be achieved for the vast majority of orthognathic patients (see Fig. 12-35, *I* and *J*). As a general rule, it is best to prevent too little maxillary incisor and gingival show in relationship to the upper lip, because this will result in an edentulous, aged, or sad look. Remember that, with age, the upper lip lengthens as a result of a loss of muscle tone. As long as excess lip separation at rest and mentalis strain with closure are avoided, erring on the side of more rather than less incisor show is generally the preferred aesthetic objective.

NOTE: I prefer to make final decisions regarding the exact aesthetic vertical position of the maxillary incisors during operation after the maxilla is secured to the mandible through the intermediate splint. At this point, with the condyles seated in the terminal hinge position, all other vector changes for the maxilla are predetermined. The maxillomandibular complex is rotated, the vertical height is measured (i.e., medial canthus to incisor height), and the final vertical incisor position is determined.

Assess the Current and Preferred Horizontal Position of the Maxillary Incisor Crown with Reference to the Upper Face

The "ideal" aesthetic horizontal (sagittal) position of the maxilla is not an exact millimeter number but rather a narrow range of what will be considered proportionate within the overall face. For each specific developmental jaw growth pattern (e.g., long face or short face growth pattern, maxillary deficiency with relative mandibular excess, cleft maxilla), the needed directional horizontal surgical change of the maxilla should be clear at the initial consultation. This will be reconfirmed during the preoperative planning examination by the operating surgeon. The preferred *horizontal (millimeter) change at the maxillary incisors* can be gauged through a combination of analyses:

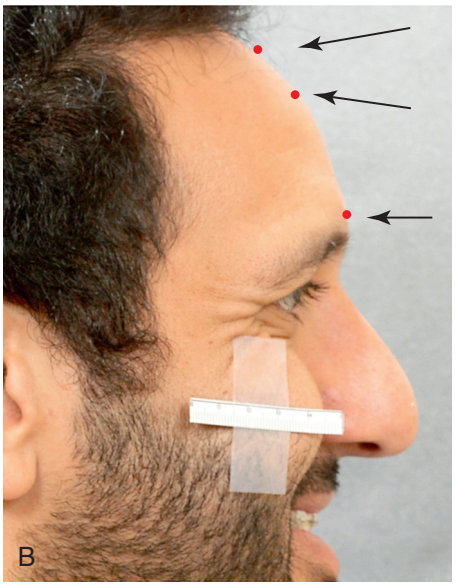
1. *Cephalometric analysis.* A lateral cephalogram of the patient is taken in (or adjusted to) the NHP, in centric relation, and with the lips relaxed.
2. *Andrews' analysis.* A lateral facial photograph of the patient is taken in (or adjusted to) the NHP, in centric relation, with a broad smile (i.e., exposing the maxillary incisors) and with the exposure of the forehead skin (i.e., with the hair pulled back).
3. *Anthropometric surface analysis.* A direct visual examination of the patient's face is made by viewing him or her in the NHP from various perspectives, both in repose and with a smile.

COMMENT: Normative cephalometric measurements (e.g., the sella-nasion-A point angle) generally underestimate but may also overestimate the preferred horizontal aesthetic projection of the maxilla and therefore cannot be relied on. Alternatively, an Andrews' analysis of how much to advance the maxilla in the sagittal plane can be a useful aide to the clinician when evaluating a patient with a dentofacial deformity. Spending time with an experienced orthognathic surgeon is a valuable way for a clinician in training to gain the hands-on knowledge required to make consistent favorable aesthetic judgments of the preferred three-dimensional reorientation of the maxilla during the operation.

As stated, *Andrews' analysis* can be a helpful tool to assess the preferred horizontal (sagittal) projection of the maxilla in the patient with a dentofacial deformity. This author's application of Andrews' "Element II of Orofacial Harmony" is illustrated in Figure 12-36. A step-by-step outline of how to do so is also given here:

- View a photograph of the individual's head and neck in profile. The photograph is best taken in the NHP and with a broad smile so that the labial surfaces of the maxillary incisors are fully visualized. The forehead skin up to the hairline must also be exposed, so the hair should be pulled back, if necessary. There should be a calibration scale on the facial photograph to confirm that it is life size (i.e., 1 to 1) so that accurate measurements can be made.
- Identify the *trichion:* This is the junction of the forehead skin and the anterior hairline. It is considered the most superior aspect of the forehead when the forehead contour is relatively flat.
- Identify the *superion:* This is typically considered the most superior aspect of the clinical forehead. This is the point used when the forehead is either rounded or angular in contour.
- Identify the *glabella:* This is the most anterior projection of the lower forehead.
- Draw a line that connects the superion and the glabella (assuming that the patient has an angular or rounded forehead).
- Identify the midpoint between the superion and the glabella: This is called the *forehead anterior point* (FA point).
- Locate and drop a vertical line through the FA point. This line should be perpendicular to the floor when viewing the patient's photograph in the NHP. This is called the *forehead anterior limit line* (FALL).
- Measure the angle at the superior forehead between these two lines. This angle is a measure of *forehead inclination*.

Face in profile (NHP)

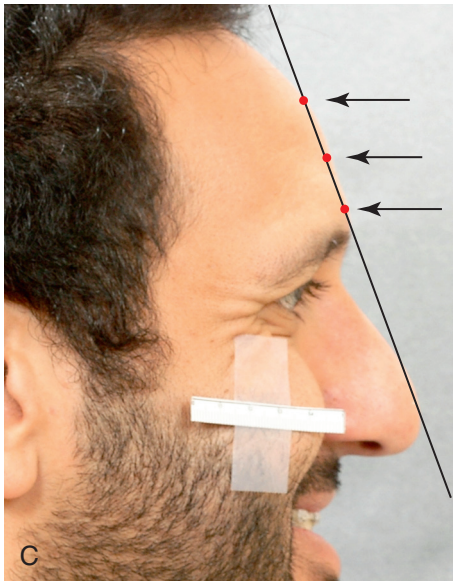


Trichion
(Junction of anterior hairline and forehead)

Superion
(Superior limit of clinical forehead)

Glabella
(Most anterior projection of forehead)

Calibration for 1 to 1 measurements

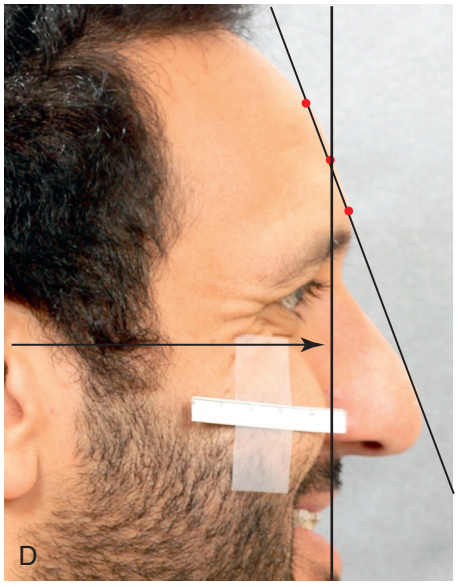


Superion

Midpoint
(FA point)

Glabella

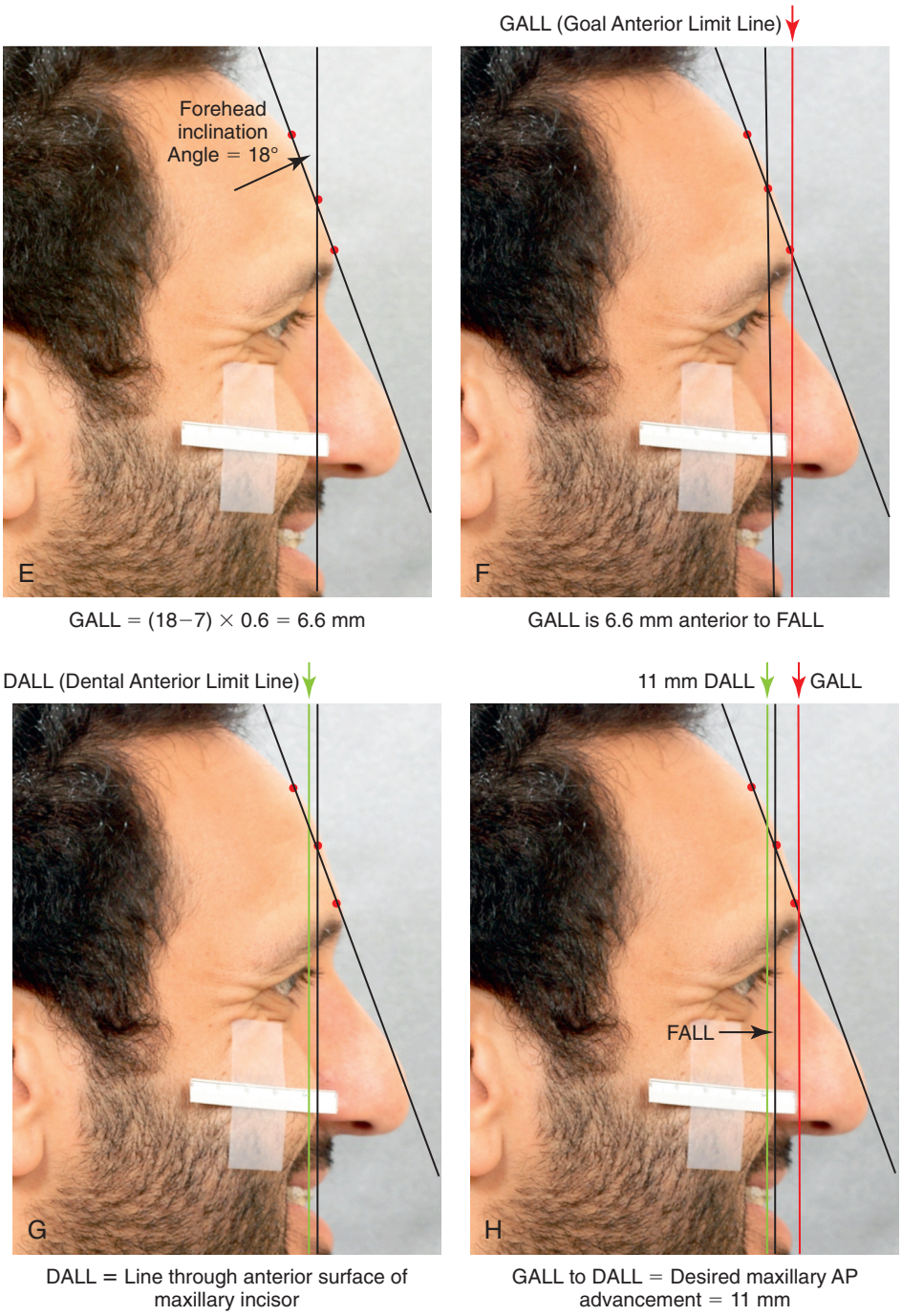
Line connecting the 2 points identify midpoint (called FA point)



FALL
(Forehead Anterior Limit Line)

Line through FA point and perpendicular to floor with patient in NHP

• **Figure 12-36** Patient viewed in profile in the natural head position (NHP). He is shown as a case example to demonstrate application of Andrews' "Element II of Orofacial Harmony." (See text for explanation.)



• **Figure 12-36, cont'd**

- Locate and drop a second vertical line that is anterior and parallel to the FALL. This second line is called the *goal anterior limit line* (GALL). The location of this line is measured in millimeters anterior to the FALL in accordance with the following standard formula: (Forehead inclination angle − 7) × 0.6 = _____ mm.
- Locate and drop a third vertical line that is parallel to both the FALL and the GALL. This line is called the *dental anterior limit line* (DALL). The line runs through the anterior surface of the maxillary incisors. The difference (in mm) between the GALL and the DALL is the "recommending" distance that the maxilla should be horizontally (sagittally) advanced to improve midface aesthetics in the patient with a maxillary deficiency. This is assuming that Andrews' Element I is correct (i.e., that

NOTE: If the GALL is anterior to the glabella, then locate and drop a fourth vertical line (through the glabella) that is parallel to the others. This represents the maximum horizontal position for the incisor to achieve preferred midface aesthetics (i.e., rather than the GALL).

the proper arch shape-positioning of the maxillary teeth [roots] over basal bone is correct).

Additional Considerations to Andrews' Standard Analysis

- I have found that Andrews' analysis for the preferred maxillary incisor position (i.e., Element II) is best suited for Caucasian patients. When applying Andrews' formula to Asian, African, or Indian morphology, caution is warranted. Facial variations in forehead, nasal, and midface morphology are characteristically influenced by *race*.
- I have found that variations in *skin thickness* can have a significant effect on the preferred aesthetic sagittal position of the maxilla. This is an additional factor that should be taken into account when reorienting the maxilla in conjunction with a Le Fort I osteotomy. For example, the individual with a thick soft-tissue envelope (e.g., a patient with obstructive sleep apnea and an elevated body mass index) may handle a greater horizontal advancement than Andrews' analysis indicates. The opposite may be true for the individual with a thin soft-tissue envelope.
- Andrews points out that midface aesthetics in profile are highly dependent on the *ideal positioning of the maxillary teeth* within the arch (i.e., Element I). An example of this is seen in the patient with a maxillary deficient and relative mandibular excess growth pattern. If maxillary bicuspid extractions are not carried out (when required) to provide space for the orthodontic retraction of the incisors toward a normal inclination, then midface aesthetics will be negatively affected. Unless *Element I* has first been achieved, the procumbent maxillary incisors will negatively affect the nasolabial angle in repose, the draping of the upper lip with a smile, and the baseline nasal base aesthetics.

The achievement of *a favorable smile* is influenced by several factors, including but not limited to: 1) the quality of the perioral soft tissues 2) the vertical position of the maxilla 3) the horizontal position of the maxilla 4) the maxillary arch width and 5) the inclination of the anterior teeth within the arch (see Fig. 35, *I* and *J*). The *smile arc* is analyzed in the frontal view by evaluating parallel lines that represent the lower lip and the upper dentition. The ideal smile arc (i.e., the consonant smile line) is characterized by the curvature of the lower lip line parallel to the curvature of the maxillary incisal edges. A *consonant smile line* is felt to project a happy and youthful appearance. The *buccal corridor* is the space that is created between the buccal surfaces of the posterior teeth and the inner aspect of the oral commissure when the individual is smiling. Those with excessive buccal corridors (i.e., negative space) are generally rated as having less attractive smiles than those with smaller buccal corridors. When the maxilla is narrow and horizontally retrusive, the visual appearance of dark buccal corridors reduces the attractiveness of the smile. Smile aesthetics are enhanced when the *display zone* is filled with the maxillary teeth. This concept is best appreciated when viewing photographs of the smile before and after orthognathic surgery for an individual with maxillary deficiency in combination with relative mandibular excess who presents with a Class III negative overjet malocclusion (see Fig. 12-35, *I* and *J*). The improvement of the *smile aesthetics* in the patient with a maxillary deficiency are often accomplished through a combination of maxillary arch expansion, horizontal advancement, and the adjustment of the vertical height.

NOTE: Decisions regarding where to position the maxillary incisor vertically and horizontally at the time of Le Fort I osteotomy will greatly affect smile aesthetics.

When determining the preferred horizontal (surgical) repositioning of the maxilla, an important facial aesthetic consideration is the interrelationship of the anterior maxillary wall (i.e., above the alveolar process), the ocular globes, and the lower eyelids (see Fig. 12-35, *D*). Horizontal advancement and clockwise rotation of the upper jaw (after Le Fort I osteotomy) for individuals who present with maxillary deficiency and relative mandibular excess are typically required to achieve and maintain favorable periorbital aesthetics (i.e., *a positive vector relationship;* see Chapter 20).

William Hogarth was an artist during the mid 1700s who published an analysis of facial beauty.[188] He documented the aesthetic importance of establishing an unbroken convex contour from the lower eyelid to the cheek (see Fig 12-35, *L*). He felt strongly that this convex shape, also called the *midface ogee curve,* was fundamental to a youthful and attractive midface. Dr. John Little further characterized the ideal midface ogee curve when viewing the individual at conversational distance.[170] This can also be appreciated on a medical-quality, high-resolution, oblique facial photograph. Little suggested that the surgeon use the midface ogee curve as an aesthetic reference standard when attempting to rejuvenate an aging face.

A lack of this youthful cheek region is often seen in individuals with either a long face Class II mandibular deficiency anterior open-bite growth pattern (see Chapter 21) or a maxillary deficiency relative mandibular excess growth pattern (see Fig. 12-35, *D*). Projecting the midface forward and appropriately rotating the maxillary plane (i.e., pitch orientation) as part of the orthognathic correction is often useful to recreate the *natural attractive curvatures and*

NOTE: Some cosmetic surgeons attempt to camouflage the individual's maxillary deficiency either with the excess injection of subcutaneous soft-tissue fillers (e.g., fat grafts, collagen) or with the placement of hard implants (e.g., Silastic, porous polyethylene) rather than using an orthognathic approach (see Chapter 40). I believe that a surgical camouflage approach is counterproductive in most cases; it will result in a less favorable facial appearance as compared with a biologic orthognathic approach, and it runs the risk of leaving the patient with an unnatural and "operated" look.

contours of the otherwise deficient midface (see Fig. 12-35, *L*). This generally requires *counterclockwise rotation* for the long face growth pattern and *clockwise rotation* for the maxillary deficient relative mandibular excess growth pattern.

Assess the Current and Preferred A-Point to B-Point Relationship of the Jaws (i.e., Pitch Orientation)

The facial aesthetic advantage of achieving an ideal *A-point to B-point relationship* has been discussed by contemporary orthodontists and orthognathic surgeons.[109,161,162,187,198,205,212-217] The individual who presents with a dentofacial deformity will frequently have a disharmonious A-point to B-point relationship, which results in negative influences on facial aesthetics (see Fig. 12-35, *D*). This deviation from ideal, when viewed in profile and in the NHP, tends to be predictable in accordance with the specific pattern of developmental jaw deformity (e.g., long face growth pattern, maxillary deficiency with relative mandibular excess, primary mandibular deficiency).

For most patients with dentofacial deformities, the achievement of preferred facial aesthetics requires the planned operative rotation of the maxillary and mandibular planes (i.e., *pitch orientation*) to correct A-point to B-point disproportion (see Fig. 12-35, *L* and *N*). This may require *clockwise* or *counterclockwise rotation* of the *maxillary and mandibular planes,* but it rarely requires a true lengthening or shortening of the posterior facial height.[198,205,212-217]

When using sagittal split ramus osteotomies, the unchanged proximal segment of the mandible establishes the posterior facial height, whereas any plane change carried out occurs in the distal segment. *The height of the proximal segment (i.e., the posterior facial height) does not change with either clockwise or counterclockwise rotation of the distal segment in conjunction with a sagittally split ramus osteotomy* (see Chapter 15).

When establishing the preferred A-point to B-point relationship and achieving full midface projection and ideal anterior vertical height through orthognathic techniques, the upper airway will also improve.

The assessment of the profile aesthetics should be made with the patient in the Natural Head Position with the teeth in centric relation and then with the normal freeway space. The lips are first relaxed (i.e., not pursed together) and then also assessed with full smile (see Fig. 12-35 *A, B,* and *D*). Individuals who present with a jaw discrepancy frequently have an altered mode of respiration and tend to rest their heads in positions that an experienced evaluator will view as involving either neck extension or flexion or a head tilt. The adjustment of the individual's head position by the clinician to the NHP is required to accurately assess the profile.

Oftentimes, the patient's lateral facial photograph and lateral cephalometric radiograph are not taken with the patient's head in the NHP. If the profile photograph, the three-dimensional computed tomography scan, and the lateral cephalometric radiograph are to be used for facial aesthetic analysis, then the image should first be rotated to establish NHP (see Fig. 12-35, *D*). From this position, a perpendicular line may be dropped (e.g., from the nasion, the soft-tissue glabella or from subnasion) as a vertical reference line from which to assess the A-point to B-point relationship, the horizontal jaw projection, and the effects on the overlying soft tissues. The clinician should then consider the need for operative rotation (i.e. pitch alteration) of the maxillary and mandibular planes as part of the orthognathic correction. This is to bring the A-point just in front of the B-point and to bring both of them in correct proximity to the vertical reference line (which is perpendicular to the NHP) to achieve enhanced facial projection.

The establishment of a favorable A-point to B-point relationship is an important part of approximating *Euclidian proportions* and of recreating the *natural attractive curvatures and contours of the face* (see Chapter 40). Achieving the preferred A-point and B-point relationship will influence the relative position of the skeletal structures above (i.e., the anterior wall of the maxilla, the pyriform rims, and the anterior nasal spine) and below (i.e., the pogonion) (see Fig. 12-35, *L* and *N*). The position of these superior and inferior skeletal structures will then be reflected through the overlying facial soft-tissue envelope. This includes the soft tissues of 1) the *lower eyelids* (i.e., improvements in the tear trough and the negative vector); 2) the *cheeks* (i.e., improvements in the nasolabial folds); 3) the *nose* (i.e., improvements in depressed nasal sills and a plunging tip); 4) the *lips* (i.e., improvements in turned-down oral commissures and marionette lines); 5) the *chin* (i.e., the prevention of ptosis); and 6) the *neck* (i.e., improvements in an obtuse neck angle and a double chin).

The surgical repositioning of the jaws to improve the A-point to B-point relationship is also carried out with consideration of its effects on the relative inclination of the maxillary and mandibular incisors (see Fig 12-35, *N*). The rotation of the maxillary plane can improve the relationship of the maxillary smile arc to make it consonant with the lower lip. With respect to *incisor inclination,* when the maxillary plane is surgically rotated clockwise, the relative incisor inclination decreases. When the maxillary plane is surgically rotated counterclockwise, the incisors become more procumbent. When the mandibular plane is rotated clockwise, the incisors become more proclined. When the mandibular plane is rotated counterclockwise, the incisors become more retroclined. These effects must be considered during the presurgical orthodontic phase and again at the time of detailed preoperative planning.

Assess the Baseline Pyriform Rims, Floor of Nose, and Anterior Nasal Spine Morphology: Anticipate the Location of each after Maxillary Reorientation

The satisfactory surgical positioning of the A-point and the B-point (i.e., pitch orientation) will generally result in more pleasing aesthetics at the pyriform aperture (paranasal) regions[154,178,198,205,212-217] (see Fig. 12-35, *L* and *N*). However,

at the time of Le Fort I osteotomy, the recontouring of the pyriform rims, the floor of the nose, and the anterior nasal spine region to correct baseline skeletal deformities may also be beneficial (see Chapter 15). The advantage of these recontouring procedures to the upper lip and tooth relationship and the aesthetics of the base of the nose should always be considered during preoperative planning.

Riola's textbook of cephalometric measurements confirms that the normal mean vertical height of the anterior nasal spine down to the maxillary incisal edge is 30 mm (±3 mm) for females and 33 mm (±3 mm) for males.[209] At the time of the Le Fort I osteotomy, the patient's specific measurements can be confirmed with a caliper. This is useful information to guide nasal floor reconstruction. For example, with the long face growth pattern, a patient who is undergoing Le Fort I osteotomy with vertical intrusion to reduce a gummy smile will frequently have an average height of between 38 mm and 45 mm. At the time of LeFort I osteotomy, recontouring and lowering the floor of the nose and the anterior nasal spine toward 30 mm is useful to normalize anatomy. In so doing, the nasal airway and the nasal aesthetic unit will be favorably enhanced (see Fig. 16-19).

Assess the Baseline Chin Morphology: Anticipate the Location of the Pogonion after Jaw Reorientation

An individual's lateral cephalometric radiograph when viewed in the NHP is useful to assess the 1) anterior facial heights, 2) any baseline bony chin dysmorphology, 3) the maxillary and mandibular incisor inclination and 4) the A-point to B-point relationships (see Fig. 12-35, *N*).

Riola's table of normative mandibular cephalometric values confirms that the mean vertical height of the chin (i.e., from the mandibular incisal edge to the menton) is approximately 42 mm (±2.7 mm) for females and 49.4 mm (±2.9 mm) for males.[210] These measurements can be a useful benchmark from which to judge the preferred vertical height of the chin.

The effective alteration of the A-point to B-point relationship at the time of orthognathic surgery will in itself generally have a positive effect on the chins appearance.[198,205,212-217] Additional direct surgical alteration of the chin (i.e., *osseous genioplasty*) is also frequently beneficial to achieve enhanced facial aesthetics (i.e., vertical lengthening or shortening, midline shifts, horizontal advancement or in some cases *recontouring of the chin surface anatomy;* see Chapter 37).

These morphologic details should be considered during the immediate preoperative face-to-face patient examination and discussed with the patient and his or her family. A plan for chin management is agreed to and then executed at the time of orthognathic surgery, with the goals of further improving aesthetics, lip function, and possibly breathing (e.g., genioglossal advancement in the individual with obstructive sleep apnea).

Reconfirm the Advantage of and Need for the Segmentation of the Maxilla

The advantage of segmentation of the maxilla should be considered before the initiation of orthodontic treatment. Maxillary segmentation is often a necessary procedure to achieve favorable long-term occlusal stability and periodontal health. It may also be required to achieve an 1) ideal curve of Spee, 2) arch width, 3) incisor inclination and 4) successful closure of dental gaps.

Segmentation is generally performed as either a *"two-piece"* osteotomy, with an interdental osteotomy between the incisors and then parasagittally through the hard palate, or as a *"three-piece" osteotomy,* with interdental osteotomies between the lateral incisor and canine on each side and then down the parasagittal midline on one or both sides (see Chapter 15).

The segmentation of the upper jaw is not an excuse for poor orthodontic preparation. It does provide an opportunity to correct skeletal issues through the basal bone rather than through orthodontic compensation that would otherwise position the teeth outside or on the edge of the alveolar process (see Chapter 17). An orthodontic compensation (i.e., dental camouflage) approach will likely be unstable for the occlusion and the periodontium in the long run (see Chapters 6 and 17).

These decisions (i.e., plans for maxillary segmentation) should be agreed upon before the initiation of orthodontic treatment, reconfirmed at the time of immediate presurgical model planning, demonstrated on the articulated dental models, registered on the prefabricated acrylic splints, and then executed during surgery (see Fig. 12-35, *G*).

Conclusions

From a patient and family perspective, facial aesthetic aspects are of significant importance to the correction of a dentofacial deformity. The most attractive faces will always incorporate a consistent symmetry of proportions in which the golden ratio can be found.

An in-depth visual facial analysis, the use of radiographic and photographic imaging, meticulous analytic model planning, and experienced surgical execution remain the important components of successful orthognathic surgery. There is much to be said for both the scientific and artistic aspects and a methodical approach to accomplish facial aesthetic objectives, the restoration of dental health, and improved head and neck function (i.e., breathing, swallowing, chewing, and speech) through the reconstruction of a dentofacial deformity. The ultimate objective is the creation of Euclidian facial proportions by attempting to reach the *Kanon* of beauty as described by Polyclitus and as visualized by the nautilus found in nature.

For the correction of a dentofacial deformity, the identification and then the reconstruction of key landmarks of the face are the objectives. In the past, simple but practical tools such as the compass and the square were used to

accomplish these goals. These are similar to the instruments that the 15th-century mathematician Luca Pacioli used to create geometric images and that are used for architecture constructions. The orthognathic surgeon now supplements these tools with computer software programs, three-dimensional imaging, and locating techniques.

In current clinical practice, we can take some solace in knowing that even Leonardo da Vinci was not entirely able to articulate the link between theory and practice for the establishment of ideal facial aesthetics. When analyzing the Mona Lisa, academicians still disagree about Leonardo's intent and the meaning of his work vis-à-vis the golden ratio. Nevertheless, with a working knowledge of the golden ratio and of its fascinating impact on the arts and architecture as well as in nature, the clinician's aesthetic sense of harmony and symmetry is enhanced for the benefit of his or her patients.

References

Part I: Facial Proportions and Symmetry

1. Alley TR, Cunningham MR: Average faces are attractive, but very attractive faces are not average. *Psychol Sci* 2:123–125, 1991.
2. Altemus L: Comparative integumental relationships. *Angle Orthod* 33:217–221, 1962.
3. Amoric M: The golden number: application to craniofacial evaluation. *Funct Orthod* 12(1):18–21, 24–25, 1995.
4. Arnett GW, McLaughlin RP: *Facial and dental planning for orthodontists and oral surgeons*, London, 2004, Mosby/Elsevier.
5. Artmann B: *Euclid: the creation of mathematics*, New York, 1999, Springer.
6. Auger TA, Turley PK: The female soft tissue profile as presented in fashion magazines during the 1900s: a photographic analysis. *Int J Adult Orthodon Orthognath Surg* 14:7–18, 1999.
7. Baig MA: Surgical enhancement of facial beauty and its psychological significance. *Ann R Australas Coll Dent Surg* 17:64–67, 2004.
8. Baker BW, Woods MG: The role of the divine proportion in the aesthetic improvement of patients undergoing combined orthodontic/orthognathic surgical treatment. *Int J Adult Orthodon Orthognath Surg* 16(2):108–120, 2001.
9. Ball WWR: *A short account of the history of mathematics*, ed 4, New York, 1960, Dover Publications, pp 50–62. [Reprint. Original publication: London, Macmillan & Co, 1908.]
10. Bashour M: An objective system for measuring facial attractiveness. *Plast Reconstr Surg* 118:757–774, 2006.
11. Bass NM: The aesthetic analysis of the face. *Eur J Orthod* 13:343–350, 1991.
12. Benson P, Perrett D: Face to face with the perfect image. *New Scientist* 133:32, 1992.
13. Boyer CB: *A history of mathematics*, ed 2, New York, 1991, John Wiley & Sons Inc.
14. Carl B, Merzbach U: *A history of mathematics*, ed 2, New York, 1989, John Wiley & Sons Inc.
15. Cook TA: *The curves of life*, New York, 1979, Dover Publications.
16. Cotton WN, Takano WS, Wong WW, Wylie WL: The Downs analysis applied to three other ethnic groups. *Angle Orthod* 21:213–220, 1951.
17. Cox NH, van der Linden FPGM: Facial harmony. *Am J Orthod* 60:175–183, 1971.
18. Coxeter HSM: *Regular polytopes*, ed 3, New York, 1973, Dover Publications.
19. DeBruine LM, Jones BC, Unger L, et al: Dissociating averageness and attractiveness: attractive faces are not always average. *J Exp Psychol Hum Percept Perform* 33:1420–1430, 2007.
20. Decker JD: The divine proportion. *Am J Orthod Dentofacial Orthop* 126(1):19A–20A, 2004.
21. Delaire J: L'analyse architecturale et structurale craniofaciale (de profil): principes theoriques: quelques exemples d'emploi en chirurgie maxillofacile. *Rev Stomatol* 79:1–33, 1978.
22. Delaire J, Schendel S, Tulasne JF: An architectural and structural craniofacial analysis: a new lateral cephalometric analysis. *Oral Surg* 52:226–238, 1981.
23. De Smit A, Dermaut L: Soft tissue profile preferences. *Am J Orthod* 86:67–73, 1984.
24. Donatsky O, Bjorn-Jorgensen J, Hermund NU, et al: Accuracy of combined maxillary and mandibular repositioning and of soft tissue prediction in relation to maxillary antero-superior repositioning combined with mandibular set back: a computerized cephalometric evaluation of the immediate postsurgical outcome using the TIOPS planning system. *J Craniomaxillofac Surg* 37:279, 2009.
25. Dunlap RA: *The golden ratio and Fibonacci numbers*, Singapore and River Edge, NJ, 1997, World Scientific Publishing.
26. Dunlevy HA, White RP, Proffit WR, Turvey TA: Professional and lay judgment of facial esthetic changes following orthognathic surgery. *Int J Adult Orthod Orthognath Surg* 2:151–158, 1974.
27. Eagly AH, Ashmore RD, Makhijani iviG, Longo LC: What is beautiful is good, but … : a meta-analytic review of research on the physical attractiveness stereotype. *Psychol Bull* 110:109–128, 1991.
28. Epker BN, Stella JP, Fish LC: Essentials of treatment planning. In *Dentofacial deformities: integrated orthodontic and surgical correction*, ed 2, St. Louis, 1995, Mosby-Year Book.
29. Farkas LG, Cheung G: Facial asymmetry in healthy North American Caucasians: an anthropometrical study. *Angle Orthod* 51:70–77, 1981.
30. Farkas LG, Kolar JC: Anthropometrics and art in the aesthetics of women's faces. *Clin Plast Surg* 14:599–615, 1987.
31. Ferrario VF, Sforza C, Ciusa V, et al: The effect of sex and age on facial asymmetry in healthy subjects: a cross-sectional study from adolescence to mid-adulthood. *J Oral Maxillofac Surg* 59:382–388, 2001.
32. Fischler R: The early relations of Le Corbusier to the golden numbers. *Environ Plann B Plann Des* 6:95–103, 1979
33. Fitzgerald R, Graivier MH, Kane M, et al: Facial aesthetic analysis. *Aesthet Surg J* 30(Suppl):25S–27S, 2010.
34. Ghyka M: *The geometry of art and life*, New York, 1977, Dover Publications Inc.
35. Gille B: Alberti, Leone Battista. In *Dictionary of Scientific Biography, I*, New York, 1970, Charles Scribner's Sons, pp 96–98.
36. Glassenberg AN, Feinberg DR, Jones BC, et al: Sex-dimorphic face shape preference in heterosexual and homosexual men and women. *Arch Sex Behav* 39:1289–1296, 2010.
37. Gonzalez-Ulloa M: Quantitative principles in cosmetic surgery of the face (profileplasty). *Plast Reconstr Surg* 29:186, 1962.
38. Gonzalez-Ulloa M: A quantum method for the appreciation of the morphology of the face. *Plast Reconstr Surg* 34:241, 1964.
39. Grimm RE: The autobiography of Leonardo Pisano. *Fibonacci Quarterly* 11(1):99–104, 1973.
40. Hambridge J: *Dynamic symmetry: the Greek vase*, New Haven, Conn, 1920, Yale University Press.
41. Harkleroad L: *The math behind the music*, Cambridge, UK, 2006, Cambridge University Press.
42. Heath TL: Euclid and the traditions about him. In Heath TL, editor: *Euclid elements 1:1-6*, 1908, Perseus Digital Library.
43. Heath TL: *The thirteen books of Euclid's elements*, 3 vols, ed 2, New York, 1956, Dover Publications.
44. Heath TL: *A history of Greek mathematics*, 2 vols, New York, 1981, Dover Publications.

45. Hemenway P: *Divine proportion: phi in art, nature, and science*, New York, 2005, Sterling, pp 20–21.
46. Henri R: *The art spirit*, Philadelphia, 1960, J.B. Lippincott.
47. Hermann A: *To think like God: Pythagoras and Parmenides—the origins of philosophy*, Chicago, 2005, Parmenides Publishing.
48. Hogarth W: Analysis of beauty. In Paulson R, editor: *New Haven*, Conn, 1997, Yale University Press, pp 5 and 94.
49. Huntley HE: *The divine proportion: a study in mathematical beauty*, New York, 1970, Dover Publications.
50. Iglesias-Linares A, Yanez-Vico R-M, Moreno-Manteca B, et al: Common standards in facial esthetics: craniofacial analysis of most attractive black and white subjects according to *People* magazine during previous 10 years. *J Oral Maxillofac Surg* 69;e216–e224, 2011.
51. Jacobson A: Orthognathic diagnosis using the proportionate template. *J Oral Surg* 38:820–833, 1980.
52. Jacobson A: Planning for orthognathic surgery—art or science? *Int J Adult Orthod Orthognath Surg* 5(4):217–224, 1990.
53. Kiekens RM, Kuijpers-Jagtman AM, van't Hof MA, et al: Putative golden proportions as predictors of facial aesthetics in adolescents. *Am J Orthod Dentofacial Orthop* 134:480–483, 2008.
54. Kiekens RM, Kuijpers-Jagtman AM, van't Hof MA, et al: Facial aesthetics in adolescents and its relationship to "ideal" ratios and angles. *Am J Orthod Dentofacial Orthop* 133:188, 2008.
55. Kingsley P: *Ancient philosophy, mystery and magic: Empedocles and the Pythagorean tradition*, Oxford, UK, 1995, Oxford University Press.
56. Komori M, Kawamura S, Ishihara S: Averageness or symmetry: which is more important for facial attractiveness? *Acta Psychol (Amst)* 131:136–142, 2009.
57. Langlois JH, Roggman LA: Attractive faces are only average. *Psychol Sci* 1:115–121, 1990.
58. Langlois JH, Roggman LA, Musselman L: What is average and what is not average about attractive faces? *Psychol Sci* 5:214–220, 1994.
59. Le Corbusier: *The modulor*, London, 1951, Faber and Faber Ltd.
60. Lele S, Richtsmeier JT: Euclidean distance matrix analysis: a coordinate-free approach for comparing biological shapes using landmark data. *Am J Phys Anthropol* 86:415–427, 1991.
61. Livio M: *The golden ratio: the story of phi, the world's most astonishing number*, New York, 2002, Broadway Books.
62. Lombardi RE: The principles of visual perception and their clinical application to denture esthetics. *J Prosthet Dent* 29:358–382, 1973.
63. Martin JG: Racial ethnocentrism and judgment of beauty. *J Soc Psychol* 63:59–63, 1964.
64 McNamara JA Jr: Cephalometric analysis of untreated adults with ideal facial and occlusal relationships. *Int J Orthodon Orthognath Surg* 3:221–231, 1988.
65. Merrifield LL: The profile line as an aid in critically evaluating facial aesthetics. *Am J Orthod* 52:804–822, 1966.
66. Midhat Gazale G: *From pharaohs to fractals*, Princeton, NJ, 1999, Princeton University Press.
67. Nguyen DD, Turley PK: Changes in the Caucasian male facial profile as depicted in fashion magazines during the twentieth century. *Am J Orthod Dentofacial Orthop* 114:208–217, 1998.
68. O'Connor JJ, Robertson EF: Euclid of Alexandria. Available at: http://www-history.mcs.st-andrews.ac.uk/Biographies/Euclid.html. Accessed January 31, 2013.
69. O'Meara DJ: *Pythagoras revived*, Oxford, UK, 1989, Oxford University Press.
70. Ore O: *Number theory and its history*, 1948, McGraw-Hill.
71. Pacioli L: *De divina proportione*, Venice, 1509, Paganinus de Paganinis.
72. Panofsky E: *Renaissance and renascenses in Western art*, Stockholm, 1960, Almqvist & Wiksell.
73. Peck H, Peck S: Concept of facial aesthetics. *Angle Orthod* 40:284, 1970.
74. Peck H, Peck S: The aesthetically pleasing face: an orthodontic myth. *Trans Eur Orthod Soc* 47:175, 1971.
75. Peck S, Peck L, Kataja M: Some vertical lineaments of lip position. *Am J Orthod Dentofacial Orthop* 101:519–524, 1992.
76. Peck S, Peck L: Selected aspects of the art and science of facial esthetics. *Semin Orthod* 1:105–126, 1995.
77. Perrett DI, May KA, Yoshikawa S: Facial shape and judgments of female attractiveness. *Nature* 368:239–242, 1994.
78. Powell N, Humphreys B: *Proportions of the aesthetic face*, New York, 1984, Thieme-Stratton.
79. Reyneke JP: *Essentials of orthognathic surgery*, Chicago, 2003, Quintessence Publishing Co Inc.
80. Rhodes G, Harwood K, Yoshikawa S, et al: The attractiveness of average faces: cross-cultural evidence and possible biological basis. In Rhodes G, Zebrowitz L, editors: *Facial attractiveness: evolutionary, cognitive, and social perspectives*, Westport, Conn, 2001, Ablexm.
81. Rhodes G, Tremewan T: Averageness, exaggeration, and facial attractiveness. *Psychol Sci* 7:105–110, 1996.
82. Rhodes G, Yoshikawa S, Clark A, et al: Attractiveness of facial averageness and symmetry in non-Western cultures: in search of biologically based standards of beauty. *Perception* 30:611–625, 2001.
83. Ricketts RM: Planning treatment on the basis of the facial pattern and an estimate of its growth. *Angle Orthod* 27:14–37, 1957.
84. Ricketts RM: Cephalometric synthesis. *Am J Orthod* 46:647–673, 1960.
85. Ricketts RM: Cephalometric analysis and synthesis. *Angle Orthod* 31:141, 1961.
86. Ricketts RM: Esthetics, environment, and the law of lip relation. *Am J Orthodont* 54:272–289, 1968.
87. Ricketts RM: A detailed consideration of the line of occlusion. *Angle Orthod* 48:274–282, 1978.
88. Ricketts RM: Perspectives in the clinical application of cephalometrics, the first 50 years. *Angle Orthod* 51:115, 1981.
89. Ricketts RM: Divine proportion in facial aesthetics. *Clin Plast Surg* 9(4):401–422, 1982.
90. Ricketts RM: The biologic significance of the divine proportion and Fibonacci series. *Am J Orthod* 81(5):351–370, 1982.
91. Ricketts RM, Roth RH, Chaconas SJ, et al: *Orthodontic diagnosis and planning*, Denver, 1982, Rocky Mountain Data Systems.
92. Runion GE: *The golden section and related curiosa*, Glenview, Ill, 1972, Scott, Foresman & Co.
93. Sahlqvist L: *Cardinal alignments and the golden section: principles of ancient cosmography and design*, ed 3, rev, Charleston SC, 2008, BookSurge.
94. Sassouni V: A classification of skeletal facial types. *Am J Orthod Dentofacial Orthop* 55:109, 1969.
95. Sharabi SE, Hatef DA, Hollier LH: Facial attractiveness: is the whole more than the sum of its parts? *Aesthetic Surg J* 30:154–160, 2010.
96. Shell TL, Woods MG: Facial aesthetics and the divine proportion: a comparison of surgical and non-surgical Class II treatment. *Aust Orthod J* 20:51–63, 2004.
97. Sigler LE, transl: *Fibonacci's Liber Abaci*, 2002, Springer-Verlag.
98. Sinclair PM: The divine proportion. *Am J Orthod* 82(2):166–167, 1982.
99. Stakhov AP: *The mathematics of harmony: from Euclid to contemporary mathematics, mathematics and computer science*, Singapore, 2009, World Scientific Publishing.
100. Stewart A: *One hundred Greek sculptors: their careers and extant works, part III of Stewart's Greek sculpture*, New Haven, Conn, Yale University Press, 1990.
101. Struik DJ: *A concise history of mathematics*, New York, 1967, Dover Publications.
102. Weyl H: *Symmetry*, Princeton, NJ, 1952, Princeton University Press.
103. Wolfe SA, Hu L, Berkowitz S: In search of the harmonious face: Apollo revisited, with an examination of the indications for retrograde maxillary displacement. *Plast Reconstr Surg* 99:1261, 1997.
104. Xia J, Shevchenko L, Gateno J, et al: Outcome study of computer-aided surgical simulation in the treatment of patients with craniomaxillofacial deformities. *J Oral Maxillofac Surg* 69:2014–2024, 2011.

105. Zeising A: Neue lehre van den proportionen des meschlischen korpers, 1854, Leipzig.

Part II: Clinical Evaluation of the Patient

106. Ackerman J, Ackerman MB, Brensinger CM, Landis JR: A morphometric analysis of the posed smile. *Clin Orthod Res* 1:2–11, 1998.
107. Ackerman JL, Proffit WR: Soft tissue limitations in orthodontics: treatment planning guidelines. *Angle Orthod* 67:327–336, 1997.
108. Ackerman JL, Proffit WR, Sarver DM: The emerging soft tissue paradigm in orthodontic diagnosis and treatment planning. *Clin Orthod Res* 2:49–52, 1999.
109. Ackerman JL, Proffit WR, Sarver DM, et al: Pitch, roll, and yaw: describing the spatial orientation of dentofacial traits. *Am J Orthod Dentofacial Orthop* 131:305–310, 2007.
110. Ackerman M, Ackerman J: Smile analysis and design in the digital era. *J Clin Orthod* 36:221, 2002.
111. Alexander SA: Diagnosis and treatment planning in orthodontics. *Curr Opin Dent* 2(4):9–13, 1992.
112. Altemus LA: A comparison of cephalofacial relationships. *Angle Orthod* 30:223–240, 1960.
113. Andrews W, Andrews L: Six elements of orofacial harmony. *The Andrews Journal of Orthodontics and Orofacial Harmony* 1:1, 2000.
114. Arnett GW, Bergman RT: Facial keys to orthodontic diagnosis and treatment planning—part I. *Am J Orthod Dentofacial Orthop* 103:299–312, 1993.
115. Arnett GW, Bergman RT: Facial keys to orthodontic diagnosis and treatment planning—part II. *Am J Orthod Dentofacial Orthop* 103:395–411, 1993.
116. Arnett GW, Jelic JS, Kim J, et al: Soft tissue cephalometric analysis: diagnosis and treatment planning of dentofacial deformity. *Am J Orthod Dentofacial Orthop* 116:239–253, 1999.
117. Babister W: *Aircraft dynamic stability and response*, 1980, Elsevier.
118. Bell R, Kiyak HA, Joondeph DR, et al: Perceptions of facial profile and their influence on the decision to undergo orthognathic surgery. *Am J Orthod* 88:323–832, 1985.
119. Bell WH, Jacobs JD, Quejada JG: Simultaneous repositioning of the maxilla, mandible and chin: treatment planning and analysis of soft tissues. *Am J Orthod* 89:28–50, 1986.
120. Beyer JW, Lindauer SJ: Evaluation of dental midline position. *Semin Orthod* 4:146–152, 1998.
121. Bishara SE: Longitudinal soft-tissue profile changes: a study of three analyses. *Am J Orthod* 88:209–223, 1985.
122. Bishara SE, Jakobsen JR, Hession TJ, Treder JE: Soft tissue profile changes from 5 to 45 years of age. *Am J Orthod Dentofacial Orthop* 114:698–706, 1998.
123. Bittner C, Pancherz H: Facial morphology and malocclusions. *Am J Orthod Dentofacial Orthop* 97:308–315, 1990.
124. Bjerin R: A comparison between the Frankfort horizontal and the sella turcica-nasion as reference planes in cephalometric analysis. *Acta Odontol Scand* 15:1–13, 1957.
125. Bjork A: The face in profile: an anthropological x-ray investigation on Swedish children and conscripts. *Svensk Tandl Tidskr* 40(Suppl), 1947.
126. Bjork A: Cranial base development. *Am J Orthodont* 41:198–225, 1955.
127. Broadbent BH Sr, Broadbent GH Jr, Golden WH: *Bolton standards of dentofacial developmental growth*, St. Louis, 1975, C.V. Mosby.
128. Burstone CJ: The integumental profile. *Am J Orthod* 44:1–25, 1958.
129. Burstone CJ: Integumental contour and extension patterns. *Angle Orthod* 29:93–104, 1959.
130. Burstone CJ: Lip posture and its significance in treatment planning. *Am J Orthod* 53:262–284, 1967.
131. Carlotti AE, Aschaffenburg PH, Schendel SA: Facial changes associated with surgical advancement of the lip and maxilla. *J Oral Maxillofac Surg* 44:593, 1986.
132. Chaconas SJ, Bartroff JD: Prediction of normal soft tissue facial changes. *Angle Orthod* 45:12–25, 1975.
133. Cleall J, Alexander WJ, McIntyre HM: Head posture and its relationship to deglutition. *Angle Orthod* 36:335–350, 1966.
134. Cooke MS, Wei SH: A summary five-factor cephalometric analysis based on natural head posture and true horizontal. *Am J Orthod Dentofac Orthop* 93:213–233, 1988.
135. Cooke MS, Wei SH: The reproducibility of natural head posture: a methodological study. *Am J Orthod Dentofac Orthop* 93:280–288, 1988.
136. Cunningham SJ, Crean SJ, Hunt NP, et al: Preparation, perceptions and problems: a long-term follow-up study of orthognathic surgery. *Int J Adult Orthod Orthognath Surg* 11:41–47, 1996.
137. Dann JJ, Fonseca RJ, Bell WH: Soft tissue changes associated with total maxillary advancement: a preliminary study. *J Oral Surg* 34:19, 1976.
138. Downs WB: The role of cephalometrics in orthodontic case analysis and diagnosis. *Am J Orthod* 38:162–182, 1952.
139. Downs WB: Analysis of the dentofacial profile. *Am J Orthodont Oral Surg* 26:192–212, 1956.
140. Ekman P, Davidson RJ, Friesen WV: The Duchenne smile: emotional expression and brain physiology. *J Pers Soc Psychol* 58:3432, 1990.
141. Ellis E III, Johnson DG, Hayward JR: Use of orthognathic surgery simulating instrument in the presurgical evaluation of facial asymmetry. *J Oral Maxillofac Surg* 42:805, 1984.
142. Enlow D, Kuroda T, Lewis A: The morphological and morphogenic basis for craniofacial form and pattern. *Angle Orthod* 41:161–188, 1971.
143. Epker BN, Stella JP, Fish LC: Essentials of treatment planning. In *Dentofacial deformities: integrated orthodontic and surgical correction*, ed 2, St. Louis, 1995, Mosby-Year Book.
144. Farkas LG, Posnick JC: Growth and development of regional units in the head and face based on anthropometric measurements. *Cleft Palate Craniofac J* 29(4):301–302, 1992.
145. Farkas LG, Posnick JC, Hreczko T: Anthropometric growth study of the ear. *Cleft Palate Craniofac J* 29(4):324–329, 1992.
146. Farkas LG, Posnick JC, Hreczko T: Anthropometric growth study of the head. *Cleft Palate Craniofac J* 29(4):303–308, 1992.
147. Farkas LG, Posnick JC, Hreczko T: Growth patterns of the face: a morphometric study. *Cleft Palate Craniofac J* 29(4):308–315, 1992.
148. Farkas LG, Posnick JC, Hreczko T, Pron G: Growth patterns in the orbital region: a morphometric study. *Cleft Palate Craniofac J* 29(4):315–318, 1992.
149. Farkas LG, Posnick JC, Hreczko T, Pron G: Growth patterns of the nasolabial region: a morphometric study. *Cleft Palate Craniofac J* 29(4):318–324, 1992.
150. Fish LC, Epker BN: Surgical-orthodontic cephalometric prediction tracing. *J Clin Orthod* 14:36–52, 1980.
151. Fjellvang H, Solow B: Craniocervical postural relations and craniofacial morphology in 30 blind subjects. *Am J Orthodont Dentofacial Orthop* 90:327–334, 1986.
152. Fogel ML, Stranc MF: Lip function: a study of normal lip parameters. *Br J Plast Surg* 37:542, 1984.
153. Freihofer HPM Jr: The lip profile after correction of retro-maxillism in cleft and non-cleft patients. *J Maxillofac Surg* 4:136–141, 1976.
154. Freihofer HPM Jr: Changes in nasal profile after maxillary advancement in cleft and non-cleft patients. *J Maxillofac Surg* 5:20–27, 1977.
155. Friede H, Kahnberg KE, Adell R, et al: Accuracy of cephalometric prediction in orthognathic surgery. *J Oral Maxillofac Surg* 45:754, 1987.
156. Gateno J, Teichgraeber JF, Xia JJ: Three-dimensional surgical planning for maxillary and midface distraction osteogenesis. *J Craniofac Surg* 14:833–839, 2003.
157. Gateno J, Xia JJ, Teichgraeber JF, et al: Clinical feasibility of computer-aided surgical simulation (CASS) in the treatment of complex cranio-maxillofacial deformities. *J Oral Maxillofac Surg* 65(4):728–734, 2007.

158. Hambleton RS: The soft tissue covering of the skeletal face as related to orthodontic problems. *Am J Orthod* 50:405–420, 1964.
159. Hayes RJ, Sarver DM, Jacobson AJ: Quantification of cervicomental angle changes with mandibular advancement. *Am J Orthod Dentofacial Orthop* 105:383–391, 1994.
160. Hoffman GR, Staples G, Moloney FB: Cephalometric alterations following facial advancement surgery: 1. Statistical evaluation: a review of aesthetic evaluation of the face and an attempt to assess treatment validity. *J Craniomaxillofac Surg* 22:214–219, 1994.
161. Holdaway RA: A soft tissue cephalometric analysis and its use in orthodontic treatment planning: part 1. *Am J Orthod* 84(1):1–28, 1983.
162. Holdaway RA: A soft-tissue cephalometric analysis and its use in orthodontic treatment planning: part II. *Am J Orthod* 85:279–293, 1984.
163. Hulsey CM: An aesthetic evaluation of tooth-lip relationships present in smile. *Am J Orthod* 57:132–144, 1970.
164. Jacobson A: The "Wits" appraisal of jaw disharmony. *Am J Orthod* 67:125–138, 1975.
165. Johnston CD, Burden DJ, Stevenson MR: The influence of dental midline discrepancies on dental attractiveness ratings. *Eur J Orthod* 21:517–522, 1999.
166. Krogman WM, Sassouni V: *A syllabus in roentgenographic cephalometry*, Philadelphia, 1957, Philadelphia Center for Research in Child Growth.
167. Latta GH: The midline and its relation to anatomic landmarks in the edentulous patient. *J Prosthet Dent* 59:681–683, 1988.
168. Lavelle CL: A study of craniofacial form. *Am J Orthod* 49:65–72, 1979.
169. Legan HL, Burstone CI: Soft tissue cephalometric analysis for orthognathic surgery. *J Oral Surg* 38:744–751, 1980.
170. Little JW: Volumetric perceptions in midfacial aging with altered priorities for rejuvenation. *Plast Reconstr Surg* 105:252, 2000.
171. Lundström A, Lundström F, Lebret LM, Moorrees CF: Natural head position and natural head orientation: Basic considerations in cephalometric analysis. *Eur J Orthod* 17:111–120, 1995.
172. Lundström F, Lundström A: Natural head position as a basis for cephalometric analysis. *Am J Orthod Dentofac Orthop* 101:244–247, 1992.
173. Mansour S, Burstone C, Legan H: An evaluation of soft-tissue changes resulting from Le Fort I maxillary surgery. *Am J Orthod* 84:37–47, 1983.
174. Mauchamp O, Sassouni V: Growth and prediction of the skeletal and soft tissue profiles. *Am J Orthod* 64:83–94, 1973.
175. Michiels LYF, Tourne LPM: Nasion true vertical: a proposed method for testing the clinical validity of cephalometric measurements applied to a new cephalometric reference line. *Int J Adult Orthod Orthog Surg* 5:43–52, 1990.
176. Miller EL, Bodden WR, Jamison HC: A study of the relationship of the dental midline to the facial median line. *J Prosthet Dent* 41:657–660, 1979.
177. Mohl N: Head posture and its role in occlusion. *Int J Orthodont* 15:6–14, 1977.
178. Mommaerts MY, Lippens F, Abeloos J, et al: Nasal profile changes after maxillary impaction and advancement surgery. *J Oral Maxillofac Surg* 58:1198, 2000.
179. Moore T, Southard KA, Casko JS, et al: Buccal corridors and smile esthetics. *Am J Orthod Dentofacial Orthop* 127:208, 2005.
180. Moorrees CF: Natural head position: a revival. *Am J Orthod Dentofacial Orthop* 105:512–513, 1994.
181. Moorrees CF, Kean MR, Coenroad FA: Natural head position: a basic consideration for the analysis of cephalometric radiographs. *Am J Phys Anthropol* 16:213–234, 1958.
182. Morley J, Eubank J: Macroesthetic elements of smile design. *J Am Dent Assoc* 132:39–45, 2001.
183. Moses JJ, Lo HH: The use of asymmetric yaw in the correction of lateral facial defects in hemifacial microsomia deformities: a case report. *Int J Adult Orthodon Orthognath Surg* 7(4):229–234, 1992.
184. Mulhave A: *En biostatick undersogelse: mennekts staende stilling teorestisk og statometrisk belyst*, Copenhagen, 1958, Munksgard. Cited in Moorrees CF, Kean MR: Natural head position: a basic consideration in the interpretation of cephalometric radiographs, *Am J Phys Anthropol* 16:213–34, 1958.
185. Murphy KE, Preston CB, Evans WG: The development of instrumentation for the dynamic measurement of changing head posture. *Am J Orthod Dentofacial Orthop* 99:520–526, 1991.
186. Nahoum HI: Anterior open-bite: a cephalometric analysis and suggested treatment procedures. *Am J Orthod* 67:513–521, 1975.
187. Nattestad A, Vedtofte P: Mandibular autorotation in orthognathic surgery: a new method of locating the centre of mandibular rotation and determining consequences in orthognathic surgery. *J Craniomaxillofac Surg* 20:163, 1992.
188. O'Ryan F, Lassetter J: Optimizing facial esthetic in the orthognathic surgery patient. *J Oral Maxillofac Surg* 69:702–715, 2011.
189. O'Ryan F, Schendel S: Nasal anatomy and maxillary surgery: I. Esthetic and anatomic principles. *Int J Adult Orthodon Orthognath Surg* 4:27, 1989.
190. O'Ryan F, Schendel S: Nasal anatomy and maxillary surgery: II. Unfavorable nasolabial esthetics following the Le Fort I osteotomy. *Int J Adult Orthodon Orthognath Surg* 4:75, 1989.
191. O'Ryan F, Schendel S: Nasal anatomy and maxillary surgery: III. Unfavorable nasolabial esthetics following the Le Fort I osteotomy. *Int J Adult Orthodon Orthognath Surg* 4:157, 1989.
192. Padwa BL, Kaiser MO, Kaban LB, et al: Occlusal cant in the frontal plane as a reflection of facial asymmetry. *J Oral Maxillofac Surg* 55:811, 1997.
193. Papadopoulos MA, Lazaridou-Terzoudi T, Oland J, et al: Comparison of soft and hard tissue profiles of orthognathic surgery patients treated recently and 20 years earlier. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 108:e8, 2009.
194. Parekh S, Fields HW, Beck FM, et al: The acceptability of variations in smile arc and buccal corridor space. *Orthod Craniofac Res* 10:15, 2007.
195. Park YC, Burstone CJ: Soft tissue profile—fallacies of hard tissue standards in treatment planning. *Am J Orthod Dentofacial Orthop* 90(I):52–62, 1986.
196. Posnick JC: Discussion: Occlusal plane rotation: aesthetic enhancement in mandibular micrognathia. *Plast Reconstr Surg* 91:1241–1244, 1993.
197. Posnick JC: Quantitative computer tomographic scan analysis: normal values and growth patterns. In Posnick JC, editor: *Craniofacial and maxillofacial surgery in children and young adults*, Philadelphia, 2000, W.B. Saunders Company, pp 36–54.
198. Posnick J, Fantuzzo J, Orchin J: Deliberate operative rotation of the maxillo-mandibular complex to alter the A-point to B-point relationship for enhanced facial esthetics. *J Oral Maxillofac Surg* 64:1687–1695, 2006.
199. Posnick JC, Farkas L: Anthropometric surface measurements in the analysis of craniomaxillofacial deformities: normal values and growth trends. In Posnick JC, editor: *Craniofacial and maxillofacial surgery in children and young adults*, Philadelphia, 2000, W.B. Saunders Company, pp 55–79.
200. Proffit WR, Epker BN: Treatment planning for dentofacial deformities. In Bell WH, Proffit WR, White R, editors: *Surgical correction of dentofacial deformities*, Philadelphia, 1980, W.B. Saunders Company.
201. Proffit WR, Fields HW, Sarver DM: *Contemporary orthodontics*, ed 4, St. Louis, 2007, C.V. Mosby.
202. Proffit WR, White RP: *Surgical-orthodontic treatment*, St. Louis, 1991, C.V. Mosby.
203. Proffit WR, White RP Jr, Sarver DM: *Contemporary treatment of dentofacial deformities*, St. Louis, 2002, C.V. Mosby.
204. Reidel RA: An analysis of dentofacial relationships. *Am J Orthod* 43:103–119, 1957.
205. Reyneke JP, Evans WG: Surgical manipulation of the occlusal plane. *Int J Adult Orthodon Orthognath Surg* 5:99–110, 1990.

206. Richardson A: An investigation into the reproducibility of some points, planes and lines used in cephalometric analysis. *Am J Orthod* 52:631–651, 1966.
207. Rickets RM: The evolution of diagnosis to computerized cephalometrics. *Am J Orthod* 55:795–803, 1969.
208. Riedel RA: An analysis of dentofacial relationships. *Am J Orthod* 43:i3–i19, 1957.
209. Rigsbee OH, Sperry TP, BeGole EA: The influence of facial animation in smile characteristics. *Int J Adult Orthodon Orthognath Surg* 3:233–239, 1988.
210. Riolo ML, Moyers RE, McNamara JA, Hunter WS: *An atlas of craniofacial growth: cephalometric standards from the University School Growth Study. Craniofacial Growth Series Monograph No. 2*, Ann Arbor, 1974, Center for Human Growth and Development, University of Michigan.
211. Rosen H: Aesthetics in facial skeletal surgery. *Perspectives in Plastic Surgery* 6:1, 1993.
212. Rosen HM: Maxillary advancement for mandibular prognathism: indications and rationale. *Plast Reconstr Surg* 87:823, 1991.
213. Rosen HM: Facial skeletal expansion: treatment strategies and rationale. *Plast Reconstr Surg* 89:798, 1992.
214. Rosen HM: *Aesthetic perspectives in jaw surgery*, New York, 1999, Springer.
215. Salzmann JA: The research workshop on cephalometrics. *Am J Orthod* 46:834–847, 1960.
216. Sarver DM: Diagnosis and treatment planning of hypodivergent skeletal pattern with clockwise occlusal plane rotation. *Int J Adult Orthodon Orthognath Surg* 8:113–121, 1993.
217. Sarver DM: *Aesthetic orthodontics and orthognathic surgery*, St. Louis, 1997, Mosby.
218. Sarver DM: The importance of incisor positioning in the esthetic smile: the smile arc. *Am J Orthod Dentofacial Orthop* 120:98–111, 2001.
219. Sarver DM, Ackerman JL: Orthodontics about face: the reemergence of the aesthetic paradigm. *Am J Orthod Dentofacial Orthop* 117:575–576, 2000.
220. Sarver DM, Ackerman MB: Dynamic smile visualization and quantification. Part 2: Smile analysis and design. *Am J Orthod Dentofacial Orthop* 124:116–127, 2003.
221. Sassouni V, Nanda S: Analysis of dentofacial vertical proportions. *Am J Orthod* 50:801, 1964.
222. Savara BS, Singh IJ: Norms of size and annual increments of seven anatomical measures of maxillae in boys from 3 to 16 years of age. *Angle Orthod* 38:104, 1968.
223. Scheideman GB, Bell WH, Legan HL, et al: Cephalometric analysis of dentofacial normals. *Am J Orthod* 78(4):404–420, 1980.
224. Schendel SA, Carlotti AE: Nasal considerations in orthognathic surgery. *Am J Orthod Dentofac Orthop* 100:197–208, 1991.
225. Solow B, Tallgren A: Natural head position in standing subjects. *Acta Odontol Scand* 29:591–607, 1971.
226. Solow B, Tallgren A: Head posture and craniofacial morphology. *Am J Phys Anthropol* 44:417–436, 1976.
227. Spoylar JL: Head position error in cephalometric radiography: an implant study. *Angle Orthod* 57:77–88, 1987.
228. Steiner CC: Cephalometrics in clinical practice. *Angle Orthod* 29:8–29, 1959.
229. Steiner CC: The use of cephalometrics as an aid to planning and assessing orthodontic treatment. *Am J Orthod Dentofacial Orthop* 46:721, 1960.
230. Stella JP, Streater MR, Epker BN, Sinn DP: Predictability of upper lip soft tissue changes with maxillary advancement. *J Oral Maxillofac Surg* 47:697–703, 1989.
231. Stengel RF: *Flight dynamics*, Princeton, NJ, 2004, Princeton University Press.
232. Subtelny JD: A longitudinal study of soft tissue facial structures and their profile characteristics, defined in relation to underlying skeletal structures. *Am J Orthod* 45:481–507, 1959.
233. Talass MF, Baker RC: Soft tissue profile changes resulting from retraction of maxillary incisors. *Am J Orthod Dentofacial Orthop* 91:385–394, 1987.
234. Thompson JR: The rest position of the mandible and its application to analysis and correction of malocclusion. *Angle Orthod* 19:162–187, 1949.
235. Tjan A, Miller G, The G: Some esthetic factors in a smile. *J Prosthet Dent* 51:24–28, 1984.
236. Troulis MJ, Everett P, Seldin EB, et al: Development of a three-dimensional treatment planning system based on computed tomographic data. *Int J Oral Maxillofac Surg* 31(4):349–357, 2002.
237. Turvey TA, et al: Surgical-orthodontic treatment planning for simultaneous mobilization of the maxilla and mandible in the correction of dentofacial deformities. *Oral Surg* 54:491, 1982.
238. Tweed CH: Frankfort mandibular incisor angles in diagnosis, treatment planning and prognosis. *Angle Orthod* 24:121–169, 1954.
239. Usumez S, Orhan M: Reproducibility of natural head position measured with an inclinometer. *Am J Orthod Dentofacial Orthop* 123:451–454, 2003.
240. Vanarsdale R, White R: Three-dimensional analysis for skeletal problems. *Int J Adult Orthodon Orthognath Surg* 9:159, 1994.
241. Viazis AD: A cephalometric analysis based on natural head position. *J Clin Orthod* 25:172–181, 1991.
242. Viazis AD: Comprehensive assessment of anteroposterior jaw relationships. *J Clin Orthod* 26:673–680, 1992.
243. Vig PS, Showfety KJ, Phillips C: Experimental manipulation of head posture. *Am J Orthod* 48:175–186, 1978.
244. Vig R, Brundo G: The kinetics of anterior tooth display. *J Prosthet Dent* 39(5):502–504, 1978.
245. Wagner I, Carlsson G, Ekstrand K, et al: A comparative study of assessment of dental appearance by dentists, dental technicians and laymen using computer-aided image manipulation. *J Esthet Dent* 8:199–205, 1996.
246. West RA, McNeill RW: Maxillary alveolar hyperplasia, diagnosis and treatment planning. *J Maxillofac Surg* 3:239, 1975.
247. Wolfe SA, Berkowitz S: *Plastic surgery of the facial skeleton*, Boston, 1989, Little, Brown and Company.
248. Worms FW, Isaacson RJ, Speidel TM: Surgical orthodontic treatment planning: profile analysis and mandibular surgery. *Angle Orthod* 46(1):1–25, 1976.
249. Wylie GA, Fish LC, Epker BN: Cephalometrics: a comparison of five analyses currently used in the diagnosis of dentofacial deformities. *Int J Adult Orthod Orthog Surg* 2(1):15–36, 1987.
250. Zachrisson BU: Aesthetic factors involved in anterior tooth display and the smile; vertical dimension. *J Clin Orthod* 32:432–445, 1998.

13

Standard Analytic Model Planning for Orthognathic Surgery

JEFFREY C. POSNICK, DMD, MD

- Controversies Surrounding Model Planning for Orthognathic Surgery
- Standard Facial Records for Orthognathic Surgery
- Key Measurements Obtained via Direct Visual Examination to Define Planned Maxillomandibular Surgical Change
- Face-Bow Registration and Transfer to the Semi-Adjustable Articulator
- Use of the Erickson Model Table to Define the Spatial Orientation of an Articulator-Mounted Maxillary Unit
- Use of the Erickson Model Table to Define the Spatial Orientation of an Articulator-Mounted Mandibular Unit
- Separation of the Maxillary Cast from the Articulator Base
- Segmentation of the Maxilla
- Use of the Erickson Model Table Measurements to Reorient the Maxillary Cast
- Intermediate Splint Construction for the Positioning of the Maxilla During Operation
- Reorientation of the Mandibular Cast to Register the Final Occlusion
- Final Splint Construction for the Positioning of the Mandible During Operation
- Finishing of the Prefabricated Acrylic Splints
- Accuracy of the Described Analytic Model Planning Technique: Review of Study
- Conclusions

Unlike most surgical procedures, orthognathic surgery involves not just the thorough medical assessment of the patient but also precise preoperative dental, radiographic, and facial aesthetic planning. The surgeon must carry out a detailed face-to-face examination of the patient to determine variations from normal (Fig. 13-1).

Immediate presurgical records include the following: 1) dental impressions of the maxilla and mandible 2) an accurate bite registration in centric relation (CR) 3) a face-bow recording of maxillary orientation in relationship to the condyles and the upper face and 4) the measurement of specific facial landmarks that includes deviations from normal (Figs. 13-2 and 13-3).

The maxillary model is mounted on a semi-adjustable articulator in accordance with the face-bow recording. The mandibular model is occluded to the mounted maxillary cast with the use of the CR bite[1,2,14,17,18,21,30,52,62,63] (Fig. 13-4). The mounted dental casts are then used to complete model planning[12,50] (Figs. 13-5 through 13-8). Splints made of acrylic are fabricated and used intraoperatively to further ensure the reliable execution of predetermined facial aesthetic and functional objectives (Figs 13-9 through 13-11).

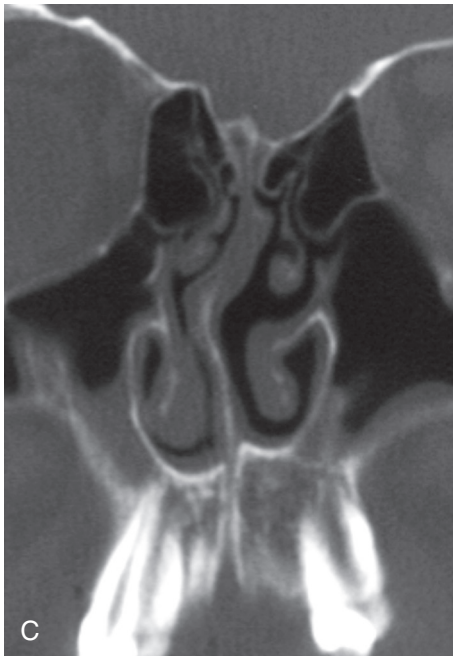
Although clinical decision making regarding the preferred aesthetic reorientation and repositioning of the jaws in the operating room remains both an art and a science (see Chapter 12), the technical aspects of planning should be precise and consistent. Analytic model planning and the use of prefabricated splints continue to represent the standard of care for bimaxillary and segmental maxillary osteotomies.[8,9,15,16,22,26,44,55,77,78,80,85,86]

Despite the benefits of this time-honored approach, there is the potential to introduce error. For example, during bimaxillary surgery, if an inaccurate bite registration is obtained from the patient preoperatively in the clinic setting, this will be transferred to the articulated models; this leads to inaccurate splint construction and then to intraoperative errors when the splints are relied on during surgery. The surgeon's ability to obtain an accurate and reproducible bite registration in CR during the immediate

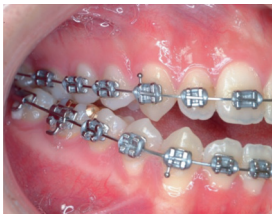
Text continued on p. 405

• **Figure 13-1** A 19-year-old man of Asian descent presented with a maxillary deficient and relative mandibular excess growth pattern. He has an Angle Class III anterior open bite with negative overjet malocclusion as well as a lifelong history of obstructed nasal breathing. The maxillofacial dysmorphology causes difficulties with speech, chewing, swallowing, breathing, and lip closure/posture, and it also negatively affects his facial aesthetics. The upper facial skeleton is symmetric and proportionate; the soft-tissue envelope is distorted but not malformed. There is a good range of motion of the neck and mandible without temporomandibular disorder. After evaluation, a comprehensive orthodontic and surgical approach was discussed and approved. Orthodontic (dental) decompensation required 10 months of active treatment. No extractions were required. Six weeks before surgery, the patient returned for a direct visual examination and the taking of records that included alginate impressions of the maxilla and mandible, centric bite registration, face-bow registration, and the transfer of the data to a semi-adjustable articulator. Decisions were made regarding critical measurements for the surgical repositioning of the jaws. The procedures carried out included the following: a Le Fort I osteotomy in segments (horizontal advancement, cant correction, arch expansion, and clockwise rotation); bilateral sagittal split ramus osteotomies (mandibular adjustment); osseous genioplasty (minimal vertical shortening and horizontal advancement); and septoplasty and inferior turbinate reduction. **A,** Frontal view in repose and occlusal views before treatment. **B,** A Panorex radiograph. This is useful to assess basic condylar morphology and to look for mandibular and dental pathology, including root resorption.

Continued



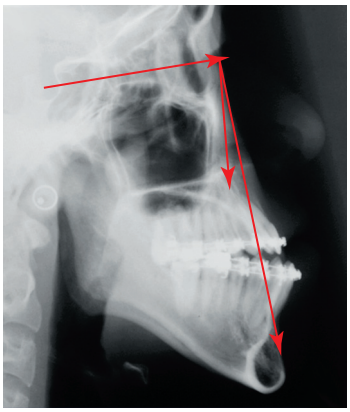
Assess intranasal anatomy
- Septal deviation? "Yes"
- Inferior turbinate hypertrophy? "Yes"
- Tight nasal inlet? "Yes"
- Elevated nasal floor? "No"

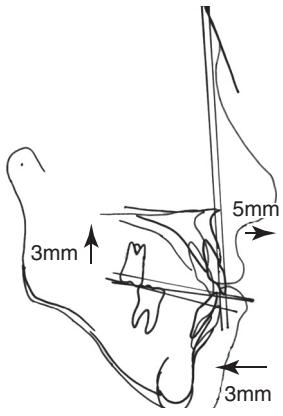


• **Figure 13-1, cont'd C,** The patient's history of lifelong nasal obstruction—in combination with an intranasal speculum and a sinus computed tomography scan examination—confirms a deviated septum (bone and cartilage) and hypertrophic inferior turbinates. Both of these conditions required surgical correction to improve the airway. **D,** Frontal view with smile and occlusal views with orthodontic decompensation in progress.

Maxillary Surgical Plan

Frontal View:	
Maxillary midline	No change needed
Cant correction	2 mm → +1 mm (L), −1 mm (R)
Anterior vertical	No change needed

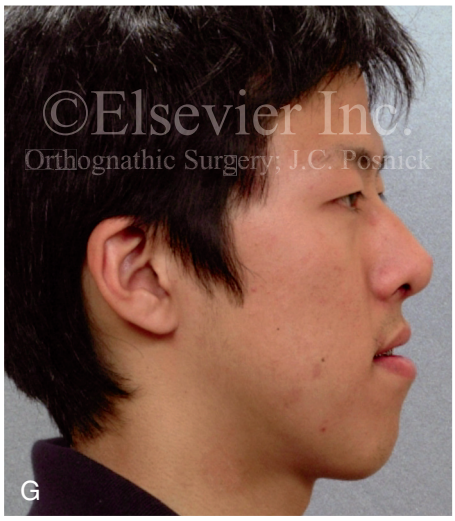




Maxillary clockwise rotation (3 mm) to enhance profile aesthetics

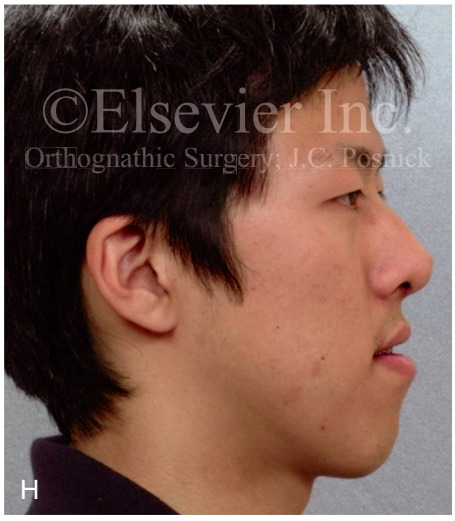
• **Figure 13-1, cont'd** **E,** A frontal facial view confirms that the maxillary midline matches the upper facial midline; that there is a satisfactory lip–tooth relationship in repose and with smile; and that there is canting of the maxilla at the molars (2 mm) as compared with the upper face. **F,** Analysis of the facial profile and the lateral cephalometric radiograph before surgery provides useful information about facial height, incisor inclination, A-point to B-point relationship, maxillary and mandibular projection, and chin morphology.

Continued



Maxillary Surgical Plan	
Profile View:	
Anterior vertical	no change
Posterior vertical	3-mm intrusion
Horizontal	5-mm advance

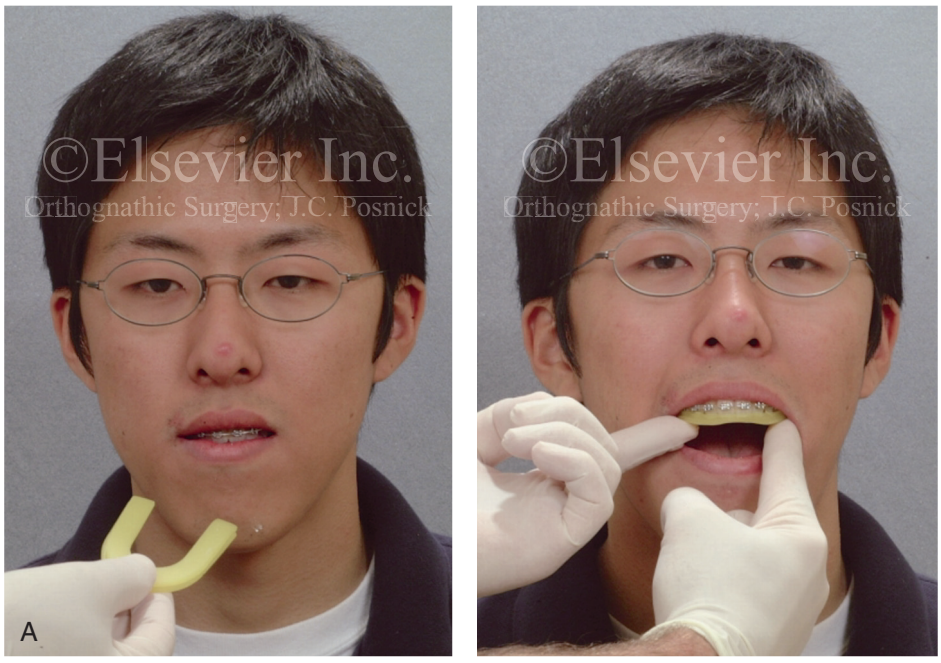
Maxillary clockwise rotation (3 mm) to enhance profile aesthetics



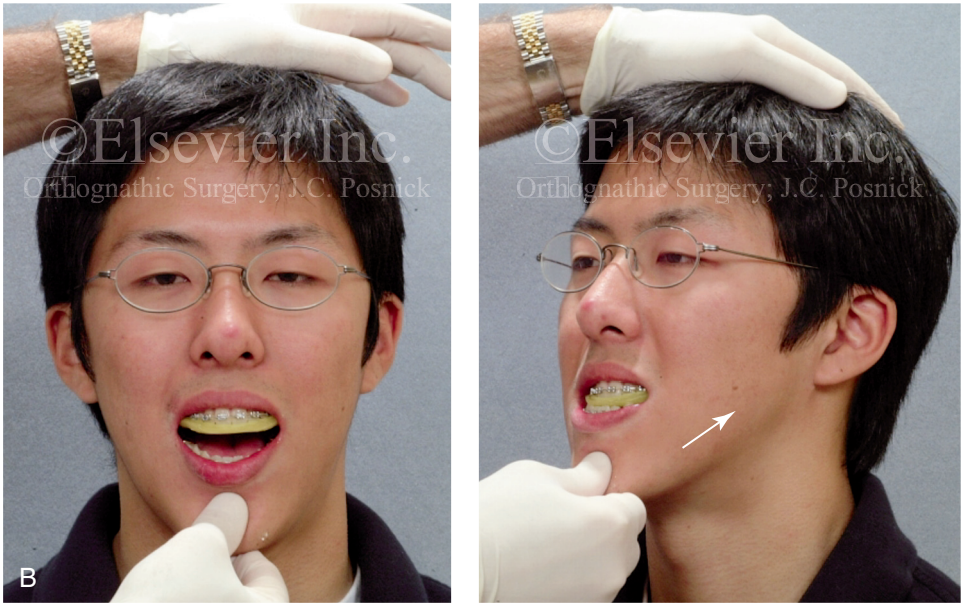
Maxillary Surgical Change	
Horizontal change at incisors	+5
Vertical change at incisors	0
Midline change at incisors	0
Cant correction -*TOTAL*	2
↑−1 on right ↓+1 on left	
Maxillary plane change -*TOTAL* (Clockwise) Counterclockwise	3
Vertical change at T#3 (right) molar (Base + Cant + Plane)	0 −1 −3 = −4
Vertical change at T#14 (left) molar (Base + Cant + Plane)	0 +1 −3 = −2

Measurements are used to spatially reposition the maxilla. The mandible is then set into occlusion with the repositioned maxilla.

• **Figure 13-1, cont'd G,** The direct visual examination of this patient in profile as well as Andrew's analysis confirms the following: an advantage of horizontal advancement at the maxillary incisors (5 mm); no need for vertical change at the maxillary incisors; an advantage of 3-mm clockwise rotation of the maxillary plane (3-mm intrusion at the first molars); and no need for horizontal change at the mandibular incisors. Note that 5-mm horizontal advancement at the incisors will correct the negative overjet and that 3-mm clockwise rotation of the maxillary plane (intrusion at the first molars) will be helpful to restore profile aesthetics and to overcome maxillary incisor procumbancy. **H,** The planned maxillary surgical change is recorded on the orthognathic data sheet. These measurements will be used to spatially reposition and reorient the maxilla first on the articulator for intermediate splint construction and then at the time of Le Fort I osteotomy. During surgery, through sagittal split ramus osteotomies, the mandible will then be set into occlusion with the repositioned maxilla.



"Chin-point guidance" technique for centric relation

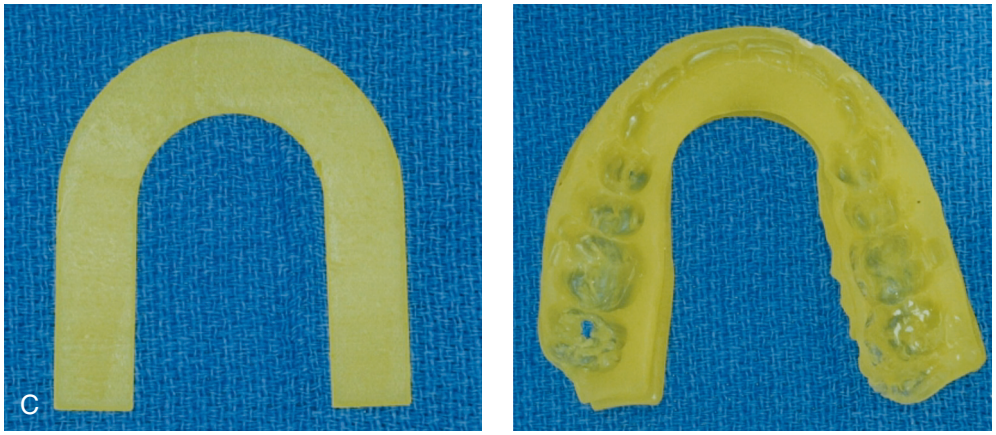


"Chin-point guidance" technique for centric relation

• **Figure 13-2** The chin-point guidance technique is used to capture (register) the centric relation bite. The patient is seated upright and in the natural head position. The chin-point guidance technique is accomplished by using one hand to apply pressure to the subject's chin and the other hand to apply counter pressure to their occiput. This allows the clinician to apply posterior and superior "vectored" force on the anterior aspect of the mandible. By doing so, the condyles will be seated in a superior-anterior location within each glenoid fossa (i.e., centric relation). **A,** The registration of the patient's maxillary occlusion into soft wax. **B,** Applying pressure to the chin with the patient's muscles relaxed. The condyles are seated, and the mandible is rotated up until the teeth first occlude. It is essential to avoid a shift (slide) from the centric relation into a centric occlusion. This may occur in a patient with malocclusion if he or she is allowed to fully clench the teeth together.

Continued

Centric relation (CR) bite (wax)

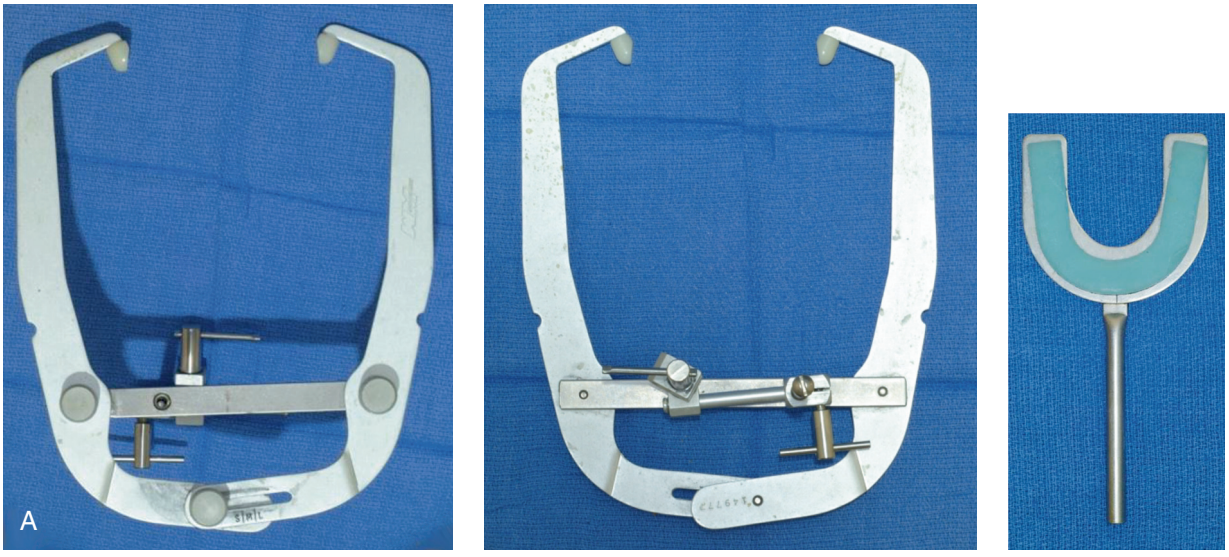


Wax wafer

Bite registration

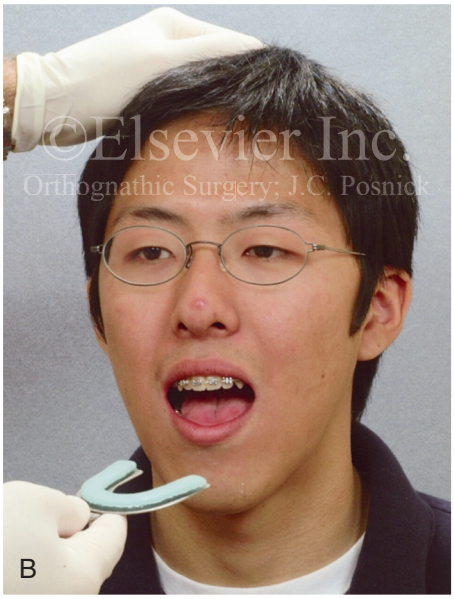
• **Figure 13-2, cont'd C,** Wax wafer before and after bite registration.

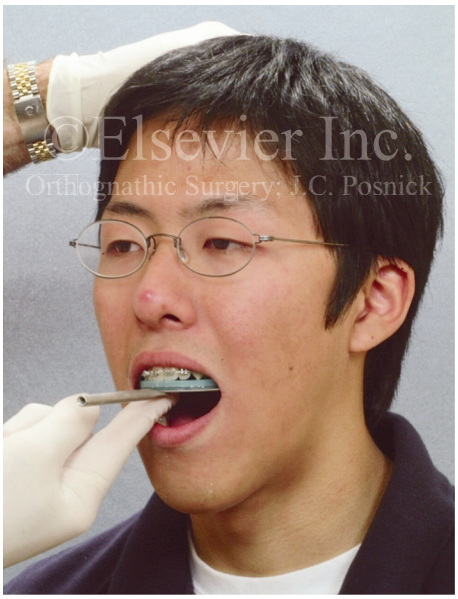
Face-bow registration → For transfer of relationships to articulator



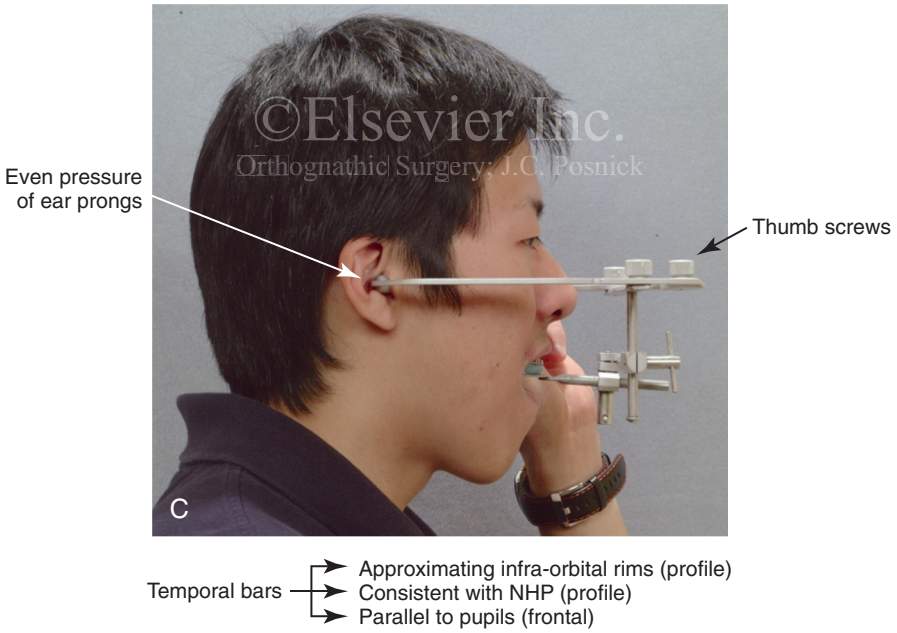
Register spacial orientation between → Condyles/ear canals
Maxilla/maxillary occlusal plane
Skull base/facial plane

• **Figure 13-3 A,** Bite fork with soft wax placed on the upper side is demonstrated. The face-bow apparatus is shown from both the bird's-eye view and the worm's-eye view.



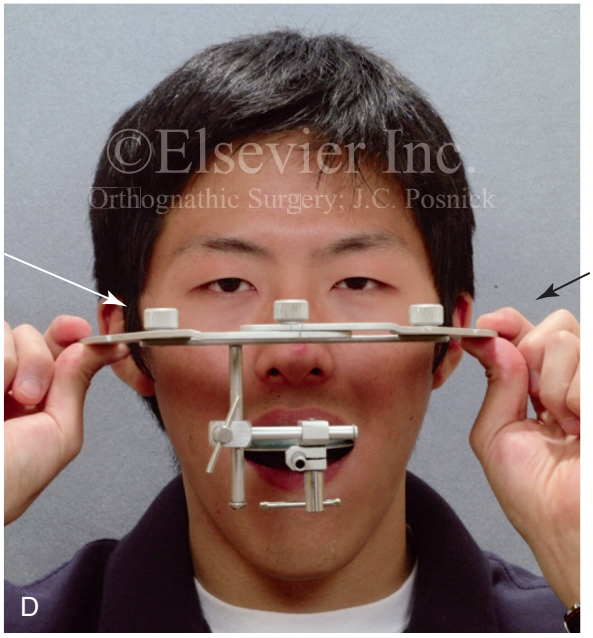


Register patient's maxillary occlusion into soft wax on bite fork

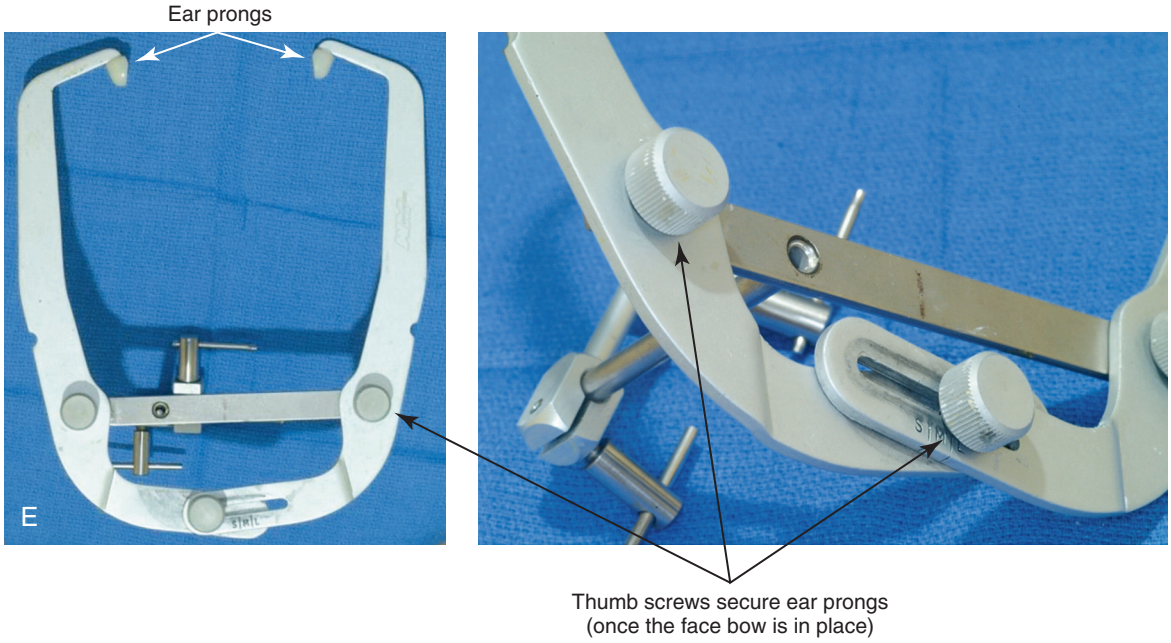


• **Figure 13-3, cont'd** **B,** The patient's maxillary occlusion is registered into soft wax on the bite fork. **C,** The face bow apparatus is held in place through even pressure of the ear prongs in each ear canal. The three thumbscrews are then tightened.

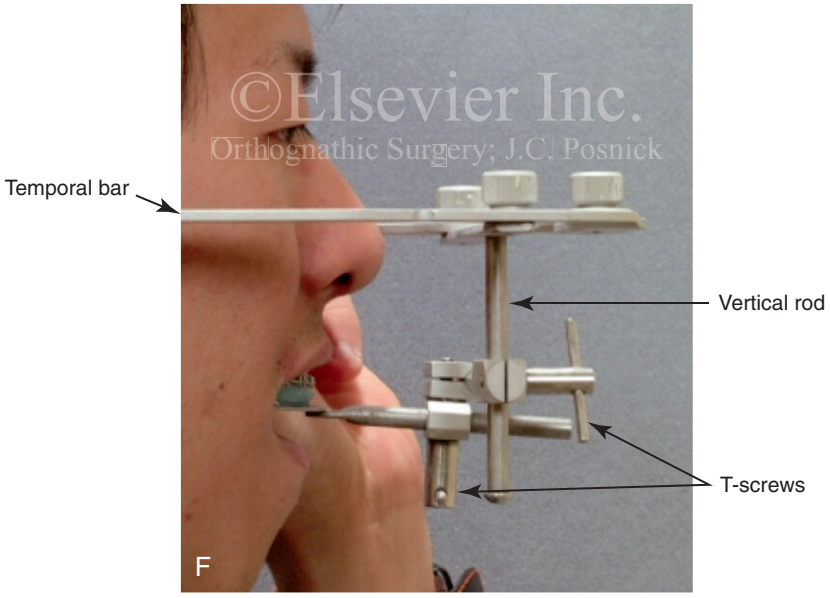
Continued



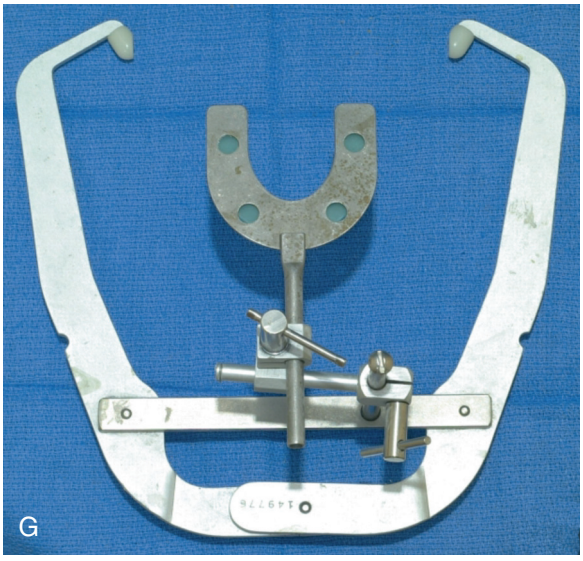
©Elsevier Inc.
Orthognathic Surgery; J.C. Posnick



• **Figure 13-3, cont'd D,** The temporal bar is set parallel with the interpapillary line and consistent with the natural head position. **E,** A close-up view is shown of the thumbscrews that secure the ear prongs in place.



T-screws secure bite fork to vertical rod of face bow



Face-bow apparatus removed from patient
Ready to place on semi-adjustable articulator

• **Figure 13-3, cont'd F,** T-screws secure the bite fork to the vertical rod of the face-bow apparatus. The T-screws are turned clockwise to secure the face bow after confirming that the temporal bar extensions are parallel to the interpupillary line (frontal view) and consistent with the natural head position (profile view). **G,** With the T-screws tight, the thumbscrews are loosened to release the face-bow apparatus from the patient. The face bow is now ready to place on the articulator.

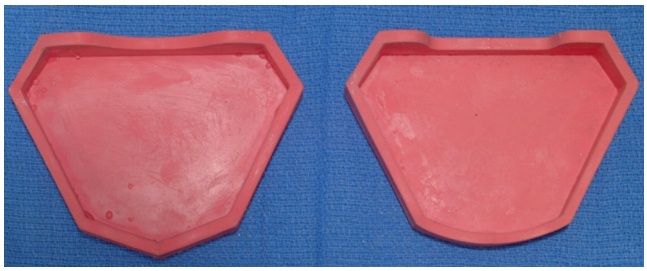
Continued

Alginate impressions of mandible and maxilla

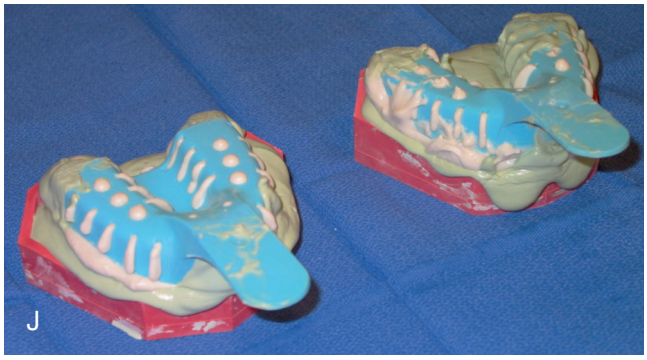
Use vibrator to limit air bubbles

Pour up alginate impressions in green dental (die) stone

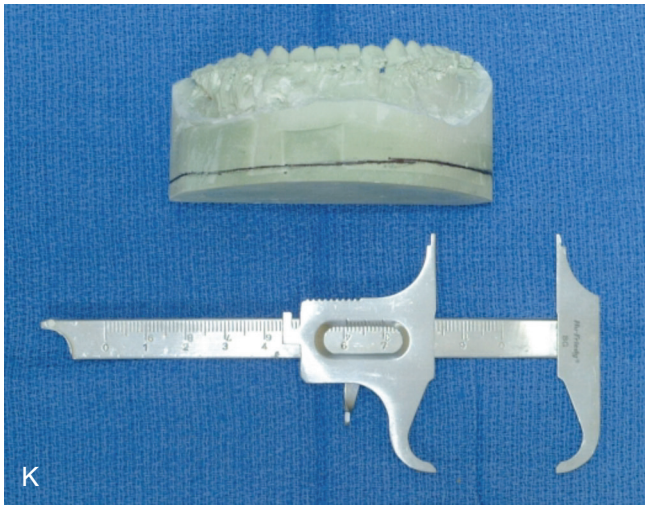
• **Figure 13-3, cont'd H,** Impression trays are selected to fit the patient's arch form. The alginate is mixed, placed in the trays, and then taken to the maxillary and mandibular arches, in turn. Negative impressions of the dentition are recorded in the alginate. **I,** The alginate impressions are "poured up" in green dental (die) stone. The use of a vibrator during this process will limit air bubbles.



Base forms are helpful



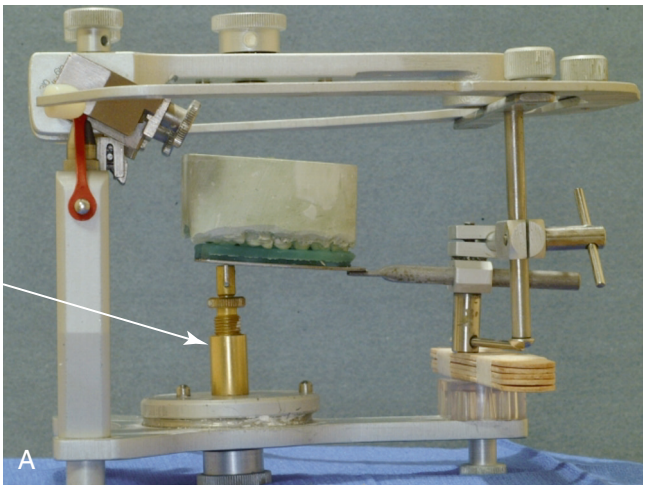
Incorporate at least 30 mm of stone base



Trim each model

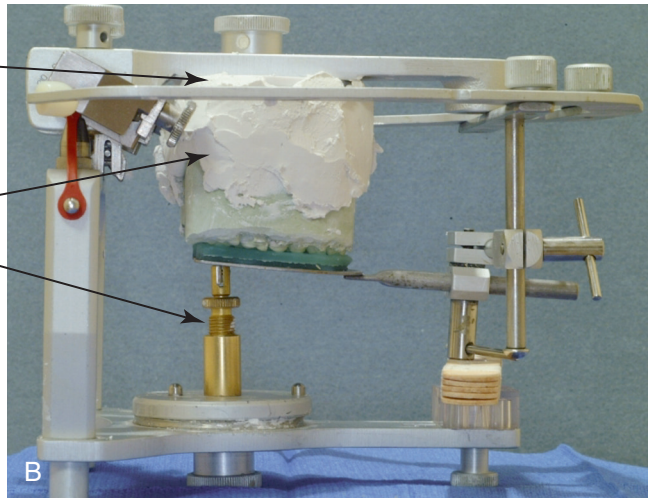
Measure (25-30 mm) down from → First molar cusp tip
→ Canine cusp tip
→ Central incisor cusp tip

• **Figure 13-3, cont'd J,** Base formers are useful when incorporating additional dental stone to achieve at least 30 mm of height for each dental cast. **K,** After the die stone is set (≈30 min), the models can be trimmed to achieve a consistent 25-mm base on the maxillary cast and a 30-mm base on the mandibular cast.

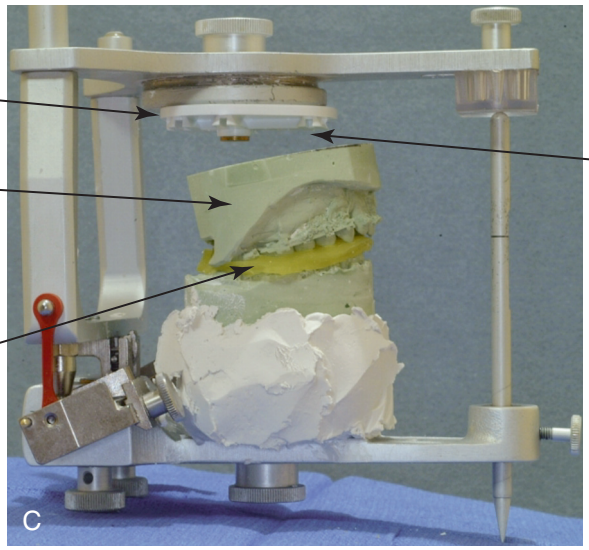


Mounting maxillary cast on articulator using white plaster bite fork support prevents sagging

Base plate

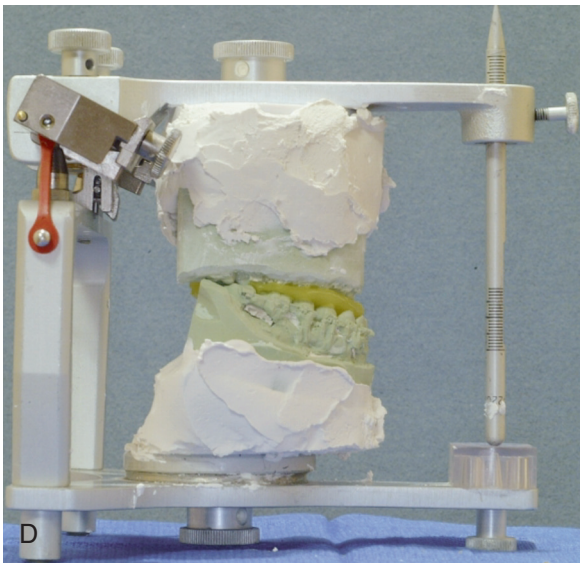


Maxillary cast secured to base plate with plaster



With maxillary unit dry – turn articulator upside down
Use CR wax bite to occlude mandibular cast to maxillary unit

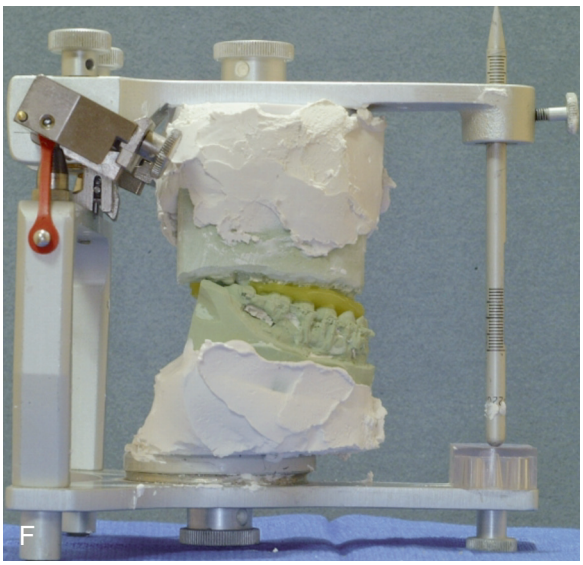
• **Figure 13-4 A,** The trimmed dental casts are then mounted on the semi-adjustable articulator with the use of the face-bow apparatus. The temporal arms of the face bow are maintained parallel to the upper aspect of the articulator and the counter top. Base plates for the maxillary and mandibular units are secured to the articulation. A bite-fork support is useful to prevent the sagging of the face-bow apparatus when mounting the maxillary cast. **B,** The plaster is mixed and added in between the maxillary dental cast and the base plate. **C,** With the maxillary unit initially set (≈15 min), the articulator is turned upside down. The centric relation wax bite is occluded to the maxillary cast. The trimmed mandibular cast is then occluded to the other side of the wax bite.



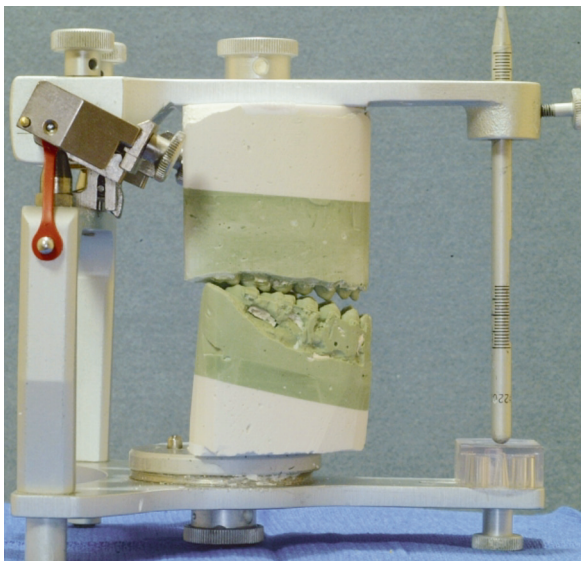
Mandibular and maxillary casts secured to articulator

• **Figure 13-4, cont'd** **D,** With the base plate secured to the articulator, the space that separates the mandibular dental cast from the base plate is filled with white plaster. **E,** The maxillary and mandibular units are removed from the articulator and taken to the grinder for trimming. **F,** The mounted dental casts are shown on the semi-adjustable articulator before and after trimming.

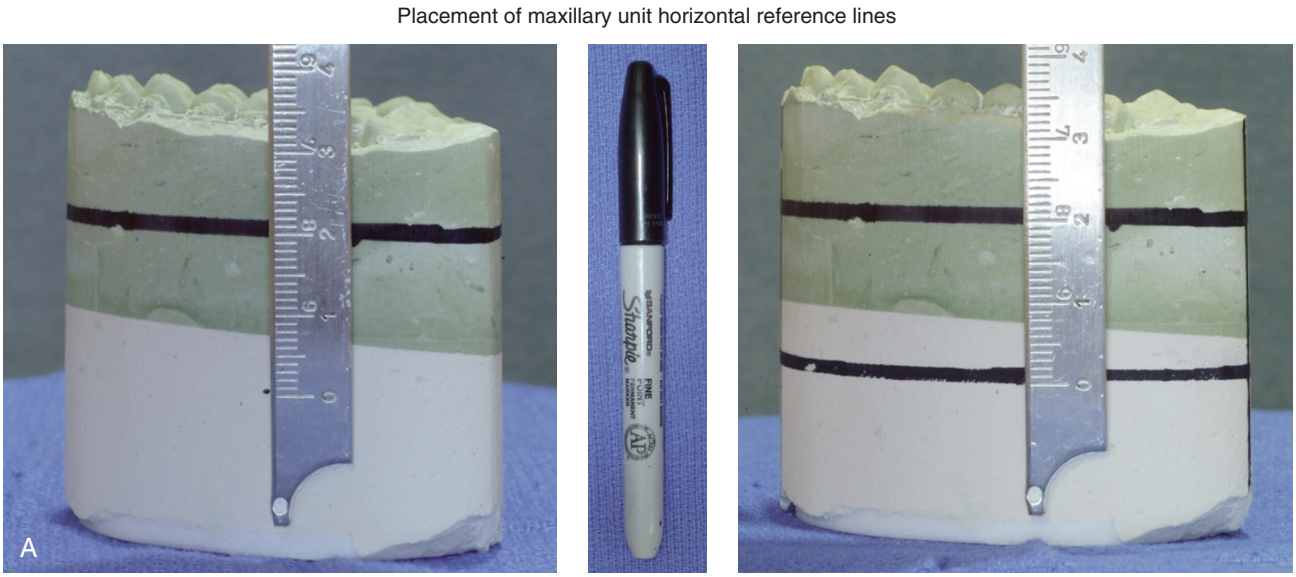
Remove maxillary and mandibular units from articulator, trim each independently on grinder



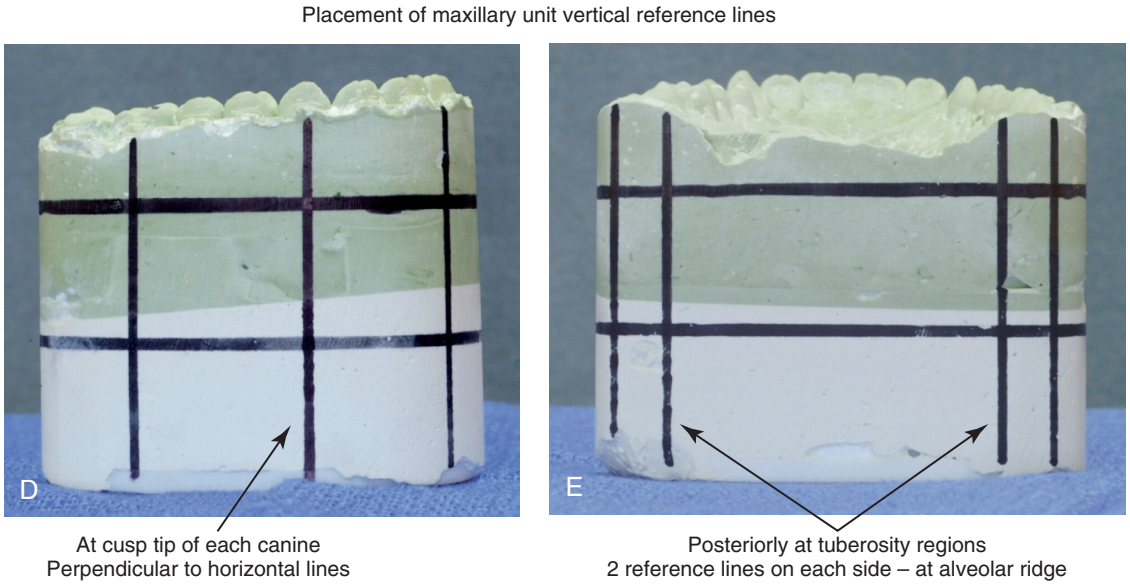
Prior to trimming



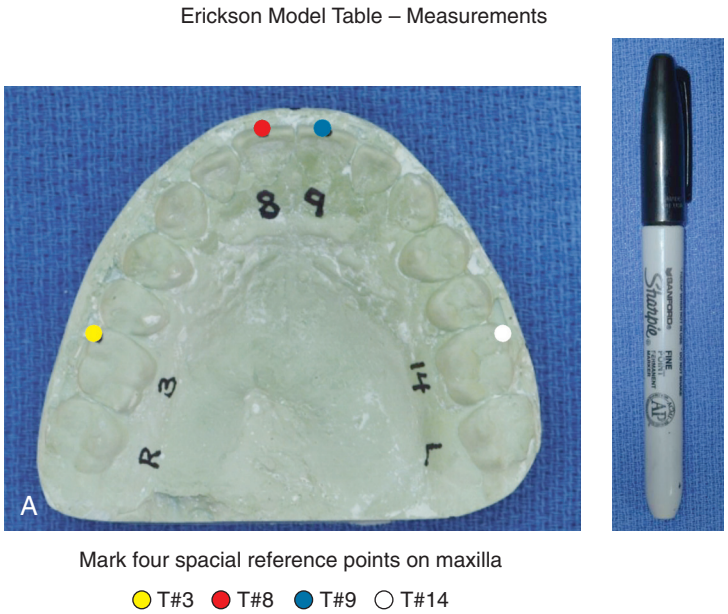
After trimming



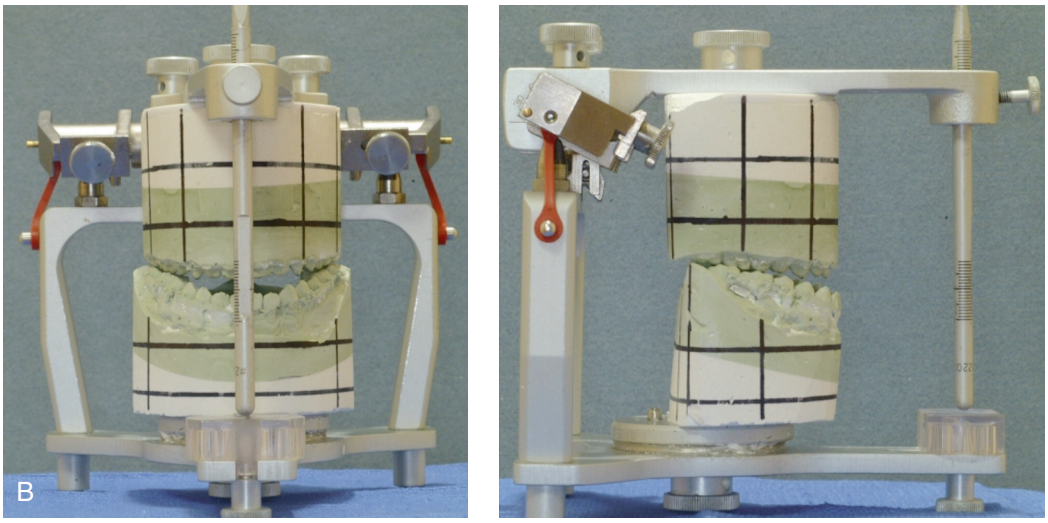
• **Figure 13-5** Marking horizontal and vertical reference lines on the articulated dental casts. **A,** The maxillary unit horizontal reference lines are placed parallel to the base plate. The first horizontal reference line is placed entirely in the green stone. The second is placed entirely in the white plaster, with 15 mm to 20 mm of separation between the lines. **B,** A maxillary unit vertical reference line is placed at the mesiobuccal groove at each first molar, perpendicular to the horizontal lines. **C,** The next maxillary unit vertical reference line is placed between central incisors, perpendicular to the horizontal lines.



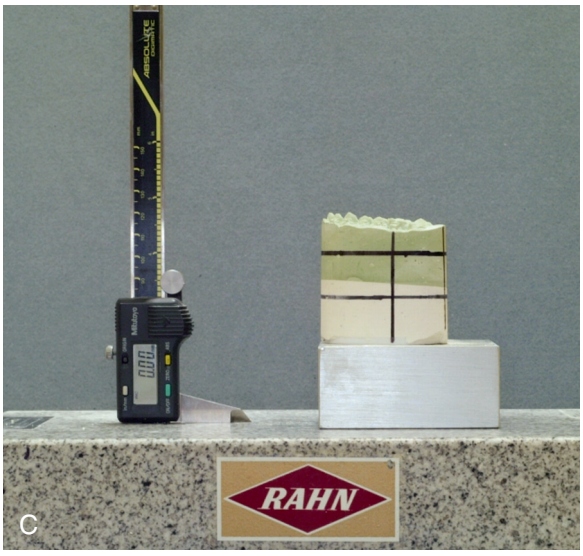
• **Figure 13-5, cont'd** **D,** Additional maxillary unit vertical reference lines are placed at the cusp tip of each canine, perpendicular to the horizontal lines. **E,** Vertical maxillary unit reference lines are also placed posteriorly in the tuberosity regions. Two reference lines are generally placed on each side, adjacent to the alveolar ridges. Similar horizontal and vertical reference lines are placed on the mandibular unit.



• **Figure 13-6** The Erickson Model Table (EMT) is used to register the baseline maxillary orientation in all three planes in space. The baseline measurements are then used to assist with the accurate repositioning and reorienting of the maxilla in accordance with clinical requirements. **A,** With a Sharpie marking pen, a reference dot is placed at the mesiobuccal cusp tip of each maxillary first molar and the midpoint of each maxillary central incisor. *Continued*



Articulated dental casts with reference lines



• **Figure 13-6, cont'd** **B,** The articulated dental casts with horizontal and vertical reference lines already marked are now ready for baseline maxillary reference measurements taken from the EMT. **C,** The maxillary unit is attached to the EMT base mount from which measurements will be made.

ORTHOGNATHIC SURGERY : ANALYTIC MODEL PLANNING

Data entry form

Patient name: _____

Surgical date: _____

Orthodontist: _____

Surgical wire date: _____

Final model date: _____

Dentist: _____

ERIKSON TABLE MEASUREMENTS

MAXILLARY HORIZONTAL

	Baseline (mm)	Change (mm)	Final
Right #8	_____	_____	_____
Left #9	_____	_____	_____

MAXILLARY VERTICAL

	Baseline (mm)	Change (mm)	Final
Right #3	_____	_____	_____
Right #8	_____	_____	_____
Left #9	_____	_____	_____
Left #14	_____	_____	_____

MAX-MIDLINE (Right-side down)

	Baseline (mm)	Change (mm)	Final
#ML	_____	_____	_____

MAX-MIDLINE (Left-side down)

	Baseline (mm)	Change (mm)	Final
#ML	_____	_____	_____

MAX TRANSVERSE (Arch width)

	Baseline (mm)	Change (mm)	Final
#6-11	_____	_____	_____
#3-14	_____	_____	_____

MANDIBULAR HORIZONTAL

	Baseline (mm)	Change (mm)	Final
#ML	_____	_____	_____

MAXILLARY SURGICAL CHANGE

Horizontal change at incisors _____

Vertical change at incisors _____

Midline change at incisors _____

Cant correction-*Total* _____

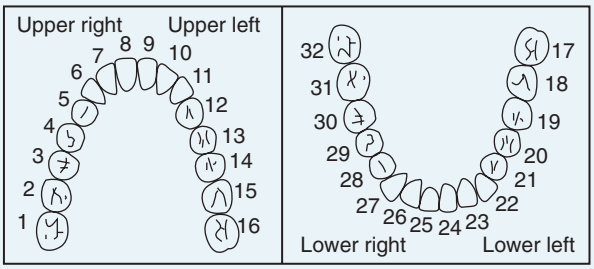
↑↓_____ on right ↑↓_____ on left

Maxillary plane change-*Total* _____
Clockwise - counterclockwise

Vertical change at T#3 (right) molar ____ ____ ____ = _____
(Base+cant+plane)

Vertical change at T#314 (left) molar ____ ____ ____ = _____
(Base+cant+plane)

OCCLUSAL EQUILIBRATION/MISSING TEETH



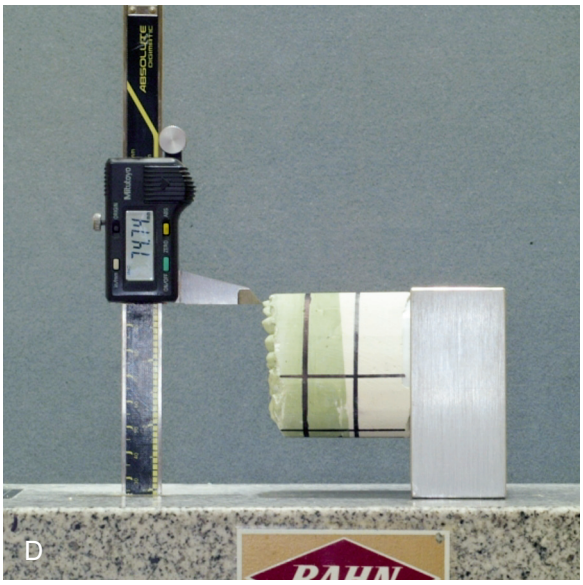
OVERALL SURGICAL PLAN

☐ Le Fort I ☐ 2 seg ☐ 3 seg
☐ Sagittal splits of mandible
☐ Genioplasty
☐ Septoplasty
☐ Reduction of inferior turbs
☐ Wisdom teeth removal
☐ Anterior neck rejuvenation
☐ Graft required
☐ Iliac ☐ Allograft ☐ HA

C

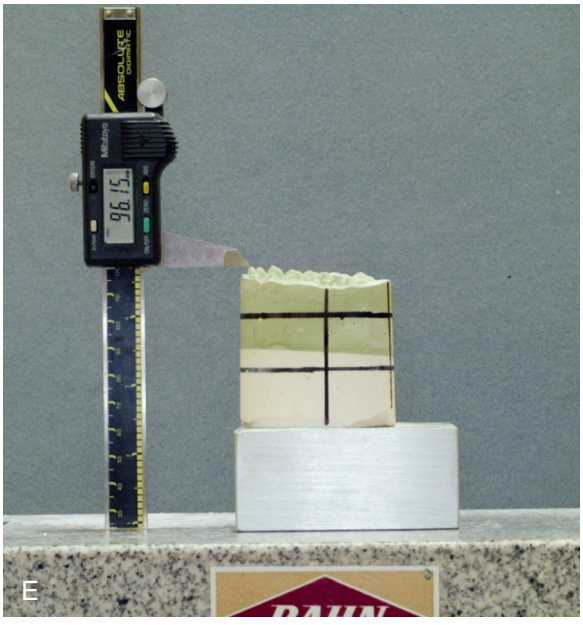
• **Figure 13-6, cont'd** C, The standard orthognathic surgery model-planning data sheet is shown.

Continued



Maxillary Horizontal			
	Baseline (mm)	Change (mm)	Final
Right #8	74.5	+5	79.5
Left #9	74.5	+5	79.5

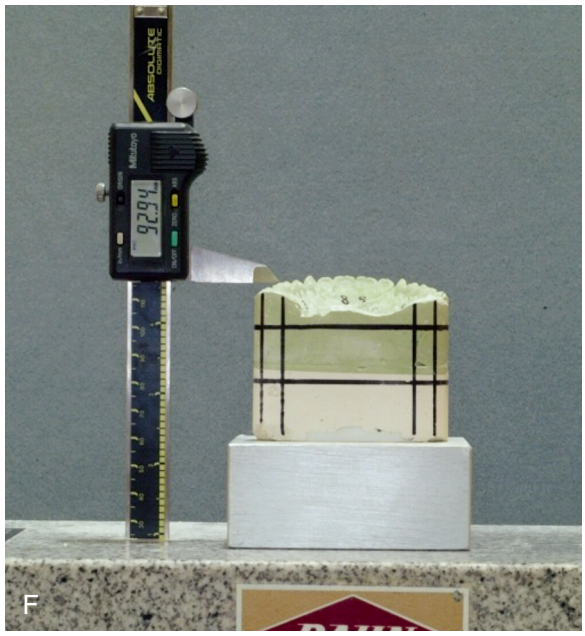
Baseline (horizontal) position of incisors (mm)



Maxillary Anterior Vertical			
	Baseline (mm)	Change (mm)	Final
Right #8	96.2	0	96.2
Left #9	95.8	0	95.8

Baseline (vertical) position of incisors (mm)

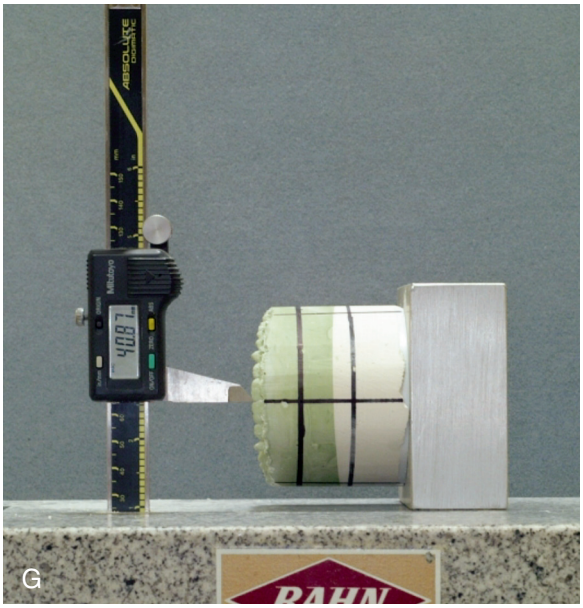
• **Figure 13-6, cont'd D,** The EMT is used to measure the baseline horizontal position of the incisors in millimeters. This is the distance between the base of the EMT and the incisal edge of each maxillary central. The millimeter number is recorded on the data sheet. The desired horizontal surgical change at the maxillary incisors is also written on the data sheet. The final preferred horizontal position of the maxillary incisors, as a result of model planning, is calculated and recorded. **E,** The EMT is used to measure the baseline vertical position of the incisors in millimeters. This is the distance between the base of the EMT and the incisal edge of each maxillary central incisor. The millimeter number is recorded on the data sheet. The desired vertical surgical change at the maxillary incisors is also written on the data sheet. The final preferred vertical position of the maxillary incisors, as a result of model planning, is calculated and recorded.



Maxillary Posterior Vertical			
	Baseline (mm)	Change (mm)	Final
Right #3	93.1	−3/−1	89.1
Left #14	91.7	−3/+1	89.7

(plane/cant)

Baseline (vertical) position of first molars (mm)

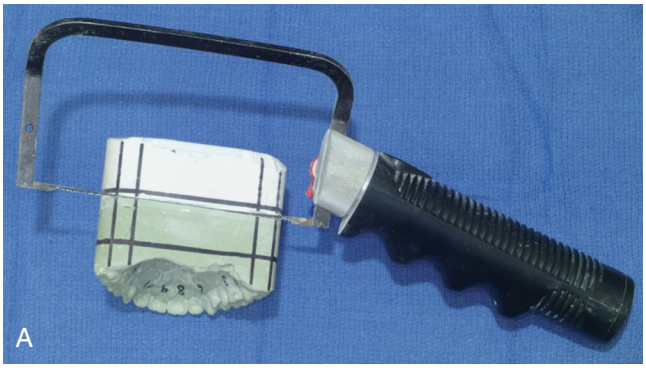


Max-Midline (Right-Side Down)			
	Baseline (mm)	Change (mm)	Final
#ML	43.6	0	43.6

Max-Midline (Left-Side Down)			
	Baseline (mm)	Change (mm)	Final
#ML	41.1	0	41.1

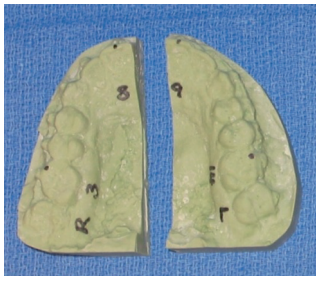
Baseline transverse (midline) position of incisors (mm)

• **Figure 13-6, cont'd F,** The EMT is used to measure the baseline vertical position of each maxillary first molar. This is the distance between the base of the EMT and the mesiobuccal cusp tip of each maxillary first molar. The millimeter number is recorded on the data sheet. The desired vertical surgical change at each maxillary first molar is also written on the data sheet. The final preferred vertical position of the maxillary molars, as a result of model planning, is calculated and recorded. **G,** The EMT is used to measure the baseline transverse midline position of each incisor in millimeters. This is an indication of the maxillary dental midline position from both the right side down and left side down directions. The base measurement is recorded on the data sheet. The desired transverse change at the maxillary incisor is written on the data sheet. The final preferred transverse midline position of the maxillary incisor, as a result of model planning, is calculated and recorded.

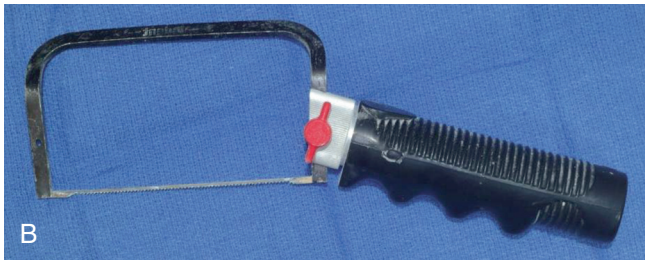


Remove the maxillary unit from the articulator
Then separate the maxillary cast from its base

3–segments?



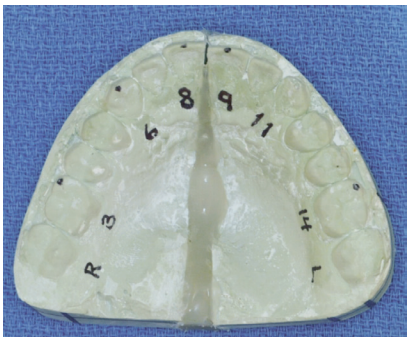
2–segments?



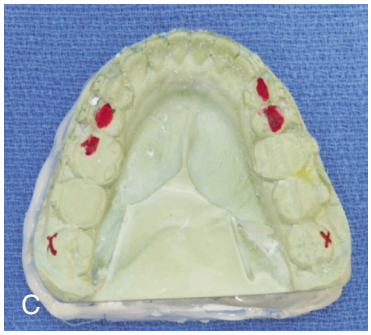
Use die saw to segment maxilla

• **Figure 13-7** The maxillary unit is now removed from the articulator. **A,** The maxillary cast is separated from its base. This is accomplished with the use of a die saw to cut a groove and a knife that is placed into the groove, which is then hit with a hammer. **B,** If indicated, segmentation of the maxillary cast is carried out. This is done in accordance with patient-specific needs to correct the maxillary arch form. Segmentation is generally carried out either as two parts (i.e., between the central incisors and then through the hard palate) or as three parts (i.e., between the lateral incisor and the canine on each side and then through the hard palate). The maxilla is cut by hand with a die saw instrument.

3–segment alignment



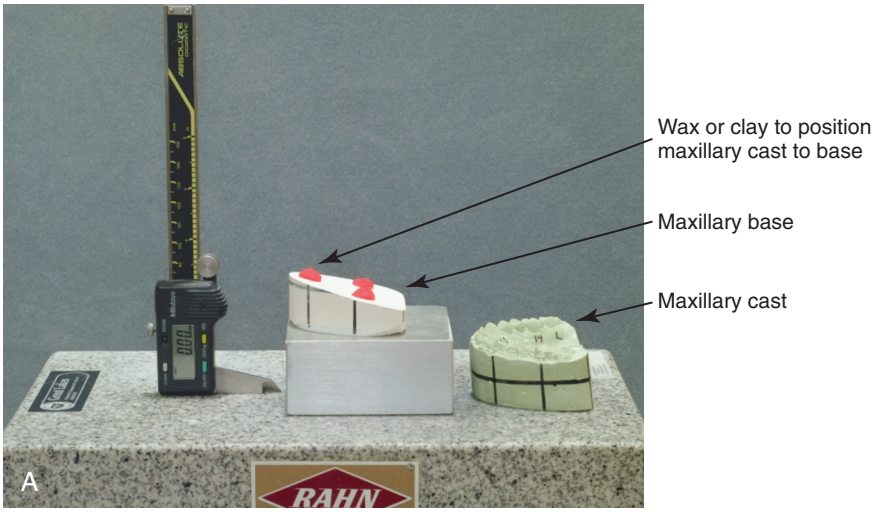
2–segment alignment



Mandibular cast

• **Figure 13-7, cont'd C,** If segmentation of the maxillary cast is required, the cut segments are placed into the corrected arch form by aligning the segments over the mandibular arch. It is important to maintain a physiologic orientation of the interdental papilla, with tight contact between the teeth. Ideally, the adjacent roots are also parallel, with minimal divergence. The maxillary segments are secured together in the corrected arch form with the use of hot glue from a glue gun.

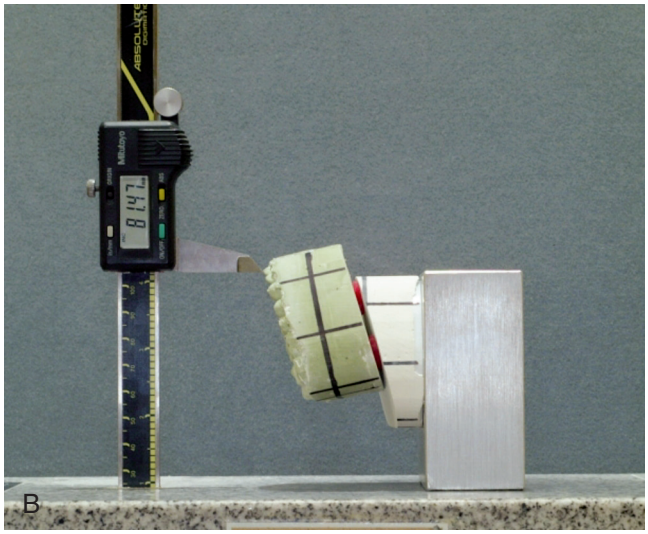
NOTE: If occlusal equilibration is required to achieve the preferred articulation, it is completed on the casts and recorded at this time. The actual occlusal equilibration procedure is completed during surgery.



Maxillary cast is repositioned on maxillary base according to clinical assessment/surgical plan

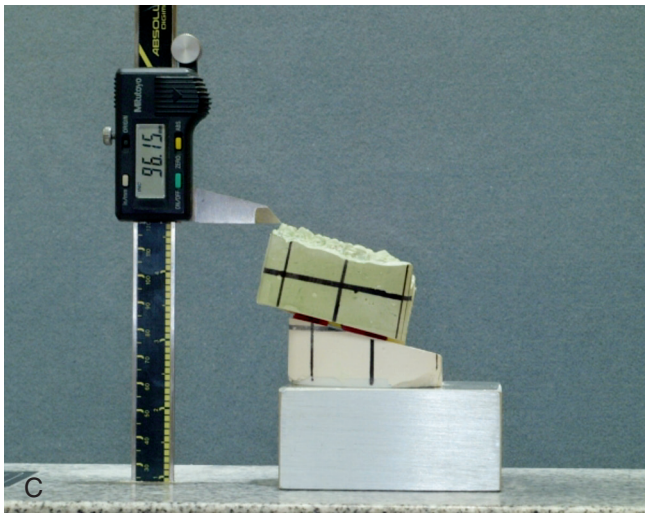
• **Figure 13-8 A,** The maxillary cast is next repositioned and reoriented on the maxillary base in accordance with the clinical assessment and the surgical plan. The Erickson Model Table (EMT) is used to ensure the correct spatial reorientation of the maxillary cast to achieve the clinical objectives.

Continued



Maxillary Horizontal			
	Baseline (mm)	Change (mm)	Final
Right #8	74.5	+5	79.5
Left #9	74.0	+5	79.5

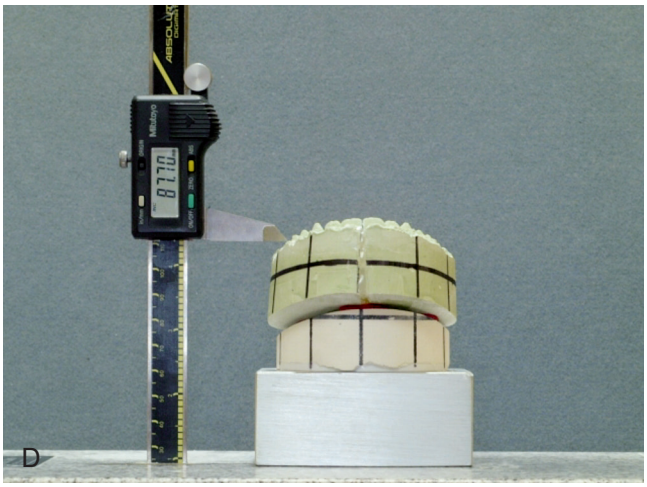
Maxillary horizontal change at incisal edge (5.0-mm advance)



Maxillary Anterior Vertical			
	Baseline (mm)	Change (mm)	Final
Right #8	96.2	0	96.2
Left #9	95.8	0	95.8

Maxillary vertical change at incisal edge (no change)

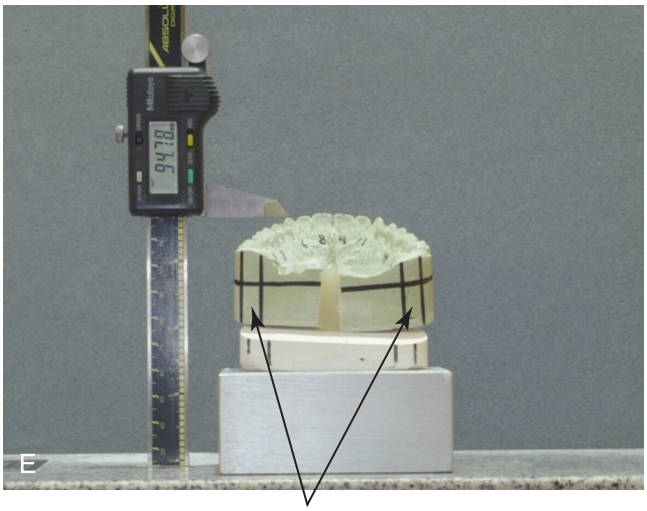
• **Figure 13-8, cont'd B,** The EMT is used to accurately reposition the maxilla. For this patient (see Fig. 13-1), the maxillary horizontal change at the incisal edge is a 5-mm advancement. **C,** In this case, the EMT also confirms that no vertical change will occur at the maxillary incisal edge.



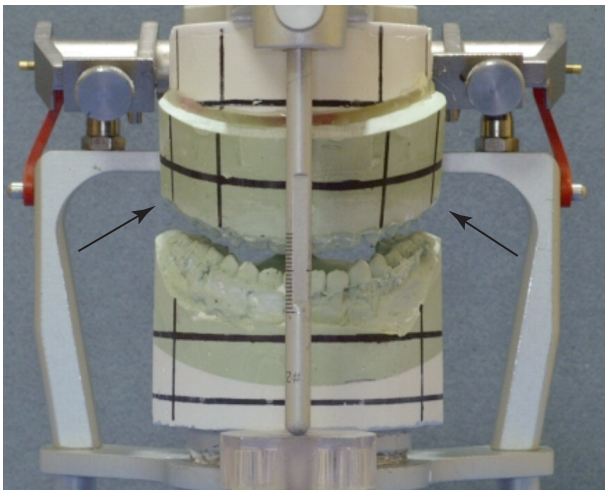
Maxillary Posterior Vertical

	Baseline (mm)	Change (mm)	Final
Right #3	93.1	−3/−1	89.1
Left #14	91.7	−3/+1	89.7

- Maxillary plane change
  - − 3.0 mm vertical (plane) change at right 1st molar
  - − 3.0 mm vertical (plane) change at left 1st molar
- Maxillary cant correction
  - − 1.0 mm vertical (cant) change at right 1st molar
  - + 1.0 mm vertical (cant) change at left 1st molar



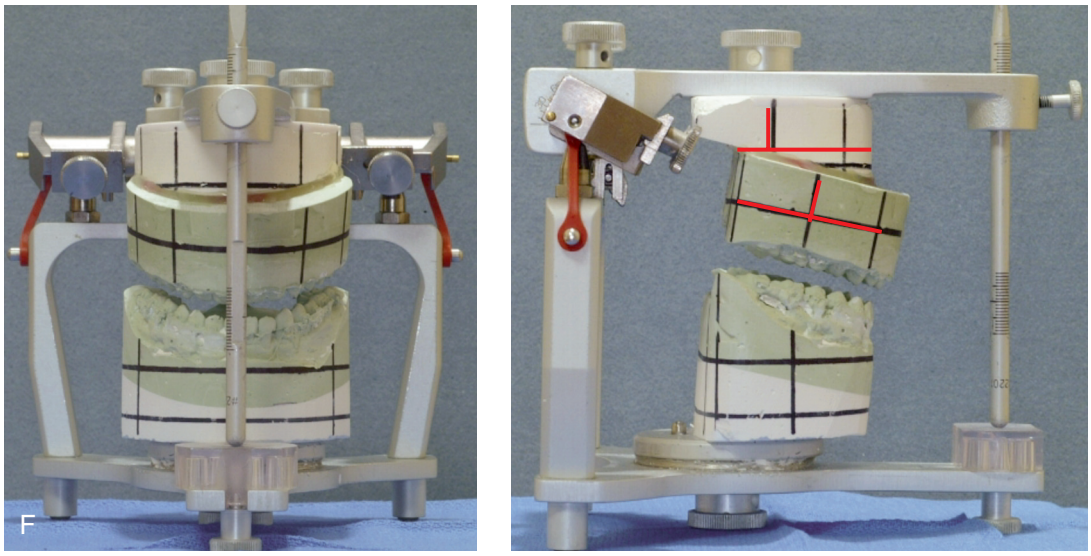
Equal change at both tuberosities
to avoid altering in yaw orientation



Check "yaw" effect
at the occlusal level!!

• **Figure 13-8, cont'd** **D,** The EMT is used to accurately reposition the maxilla at the molars. The confirmation of correct positioning at the mesiobuccal cusp tip of each first molar is essential. The vertical change incorporates the desired three-dimensional roll and pitch changes. **E,** When repositioning the maxilla, the EMT is useful to check for any yaw effect. Establishing equal side-to-side changes at both tuberosity regions is required to avoid a yaw change of the posterior maxilla. The reoriented maxillary unit is placed back onto the articulator for viewing.

Continued



Desired A-pt B-pt alteration to enhance aesthetics (3 mm clockwise rotation of maxilla)

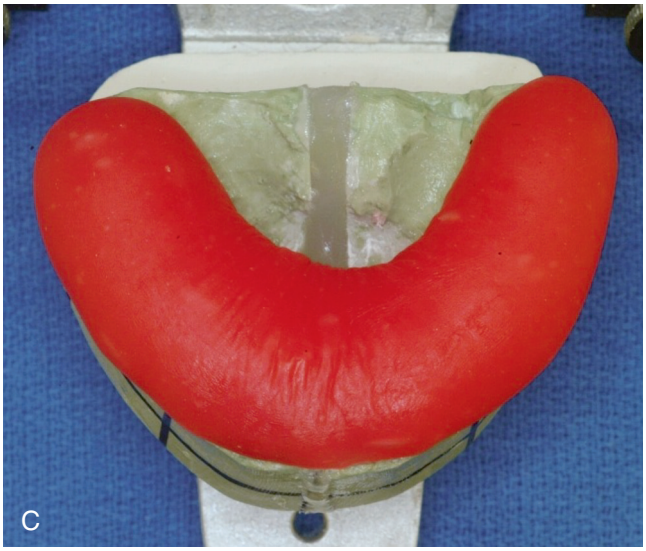
• **Figure 13-8, cont'd F,** The vertical pin on the articulator should remain neutral throughout the process.

NOTE: Before going forward with intermediate splint construction, confirming the yaw orientation of the maxilla relative to the mandible is essential.

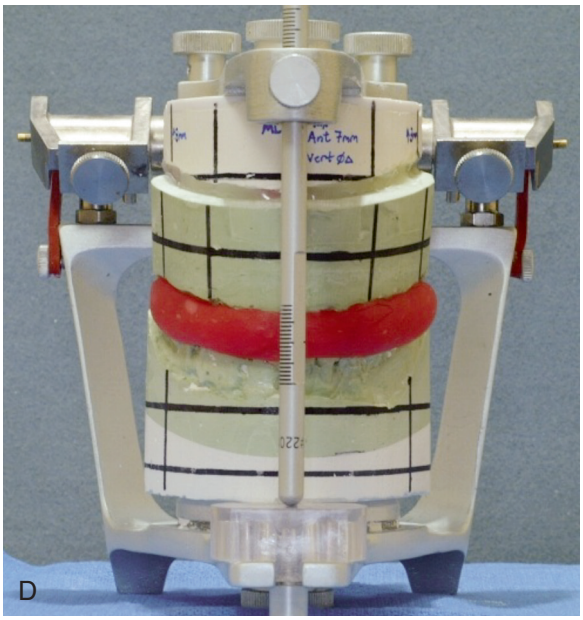
Apply Vaseline to maxillary and mandibular teeth

Use pink acrylic liquid and resin powder

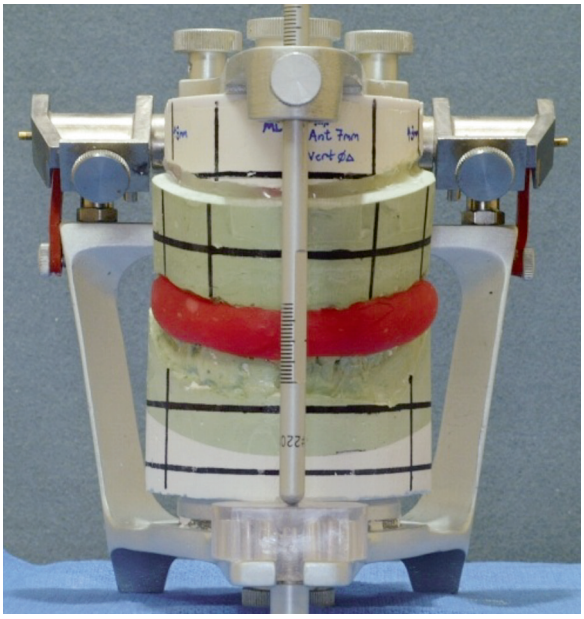
• **Figure 13-9** Construction of the intermediate splint. **A,** Apply a thin coat of Vaseline to the maxillary and mandibular teeth. **B,** Use pink acrylic liquid and orthodontic resin powder, and mix them until the mass does not slump.



Adapt mass to the maxillary occlusal surface



- Close articulator until pin touches
- Mandibular occlusal imprint will result

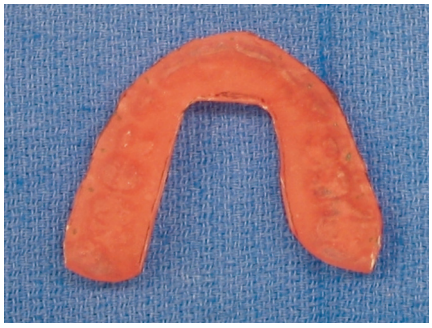


Carefully trim soft acrylic with scissors

• **Figure 13-9, cont'd** **C,** Roll the mixed acrylic mass into a log shape. Adapt the mass to the maxillary occlusal surface. **D,** Close the articulator until the vertical pin touches in a neutral location. A mandibular occlusal surface imprint will result. **E,** Limit "undercuts" from brackets by carefully trimming the soft acrylic with scissors.

Continued

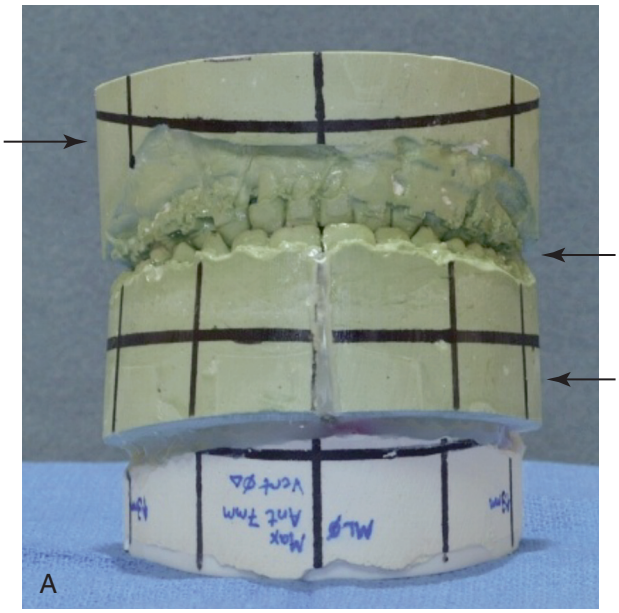
Use lathe to trim excess hardened acrylic



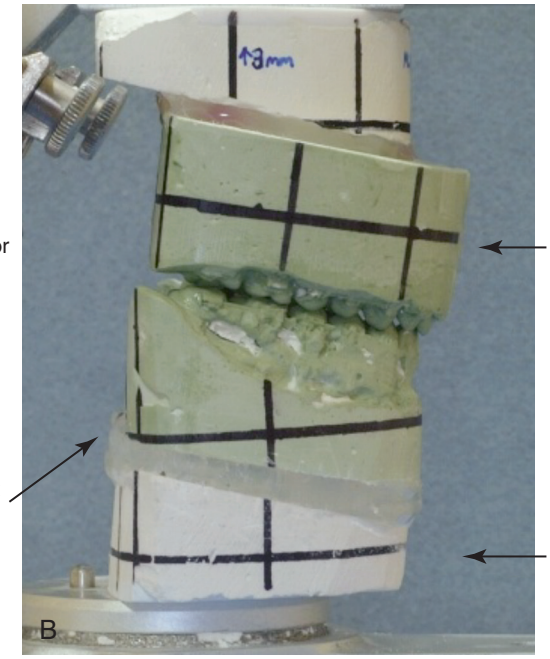
Maintain adequate strength!

Use Dremel drill (acrylic burr) to trim excess hardened acrylic

• **Figure 13-9, cont'd F,** When the acrylic is dry, use a lathe to trim excess hardened acrylic. Maintain adequate strength of the splint to prevent bowing or breakage during surgery. **G,** Use a rotary drill (e.g., a Dremel drill) with an acrylic bur to further trim excess hardened acrylic.



Remove maxillary unit from articulator
Separate mandibular cast from plaster base
Articulate mandibular cast to maxillary unit in "final" occlusion

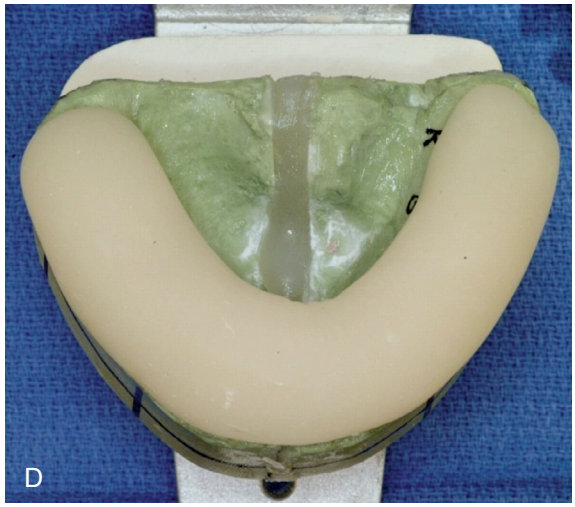


Secure mandibular cast to base with hot glue
Then separate max/mand casts and confirm "final" occlusion

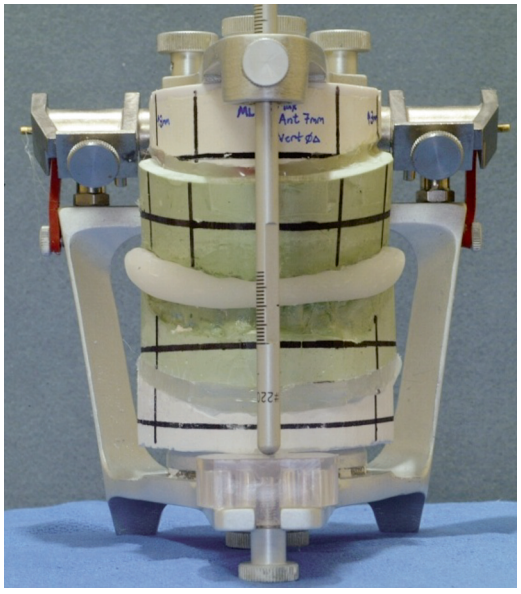
• **Figure 13-10** Construction of the final splint. **A,** Remove the maxillary unit from the articulator. Separate the mandibular cast from the plaster base as previously described for the maxillary unit (see Fig. 13-7, *A*). Next, articulate the mandibular cast with maxillary unit in the final desired occlusion. Secure the final occlusion with hot glue from a glue gun. **B,** Secure the mandibular cast back to the base with the use of either hot glue from a glue gun or white plaster. Maintain the articular pin in a neutral position to achieve this objective. It is generally necessary to remove as least some plaster from the mandibular base during this process; only then is the hot glue or additional plaster used to secure the mandibular cast back to the base. The dried glue that secured the occlusion together is now released. The desired occlusion is confirmed to be accurate before the construction of the final splint.

Continued

Use clear acrylic liquid and resin powder

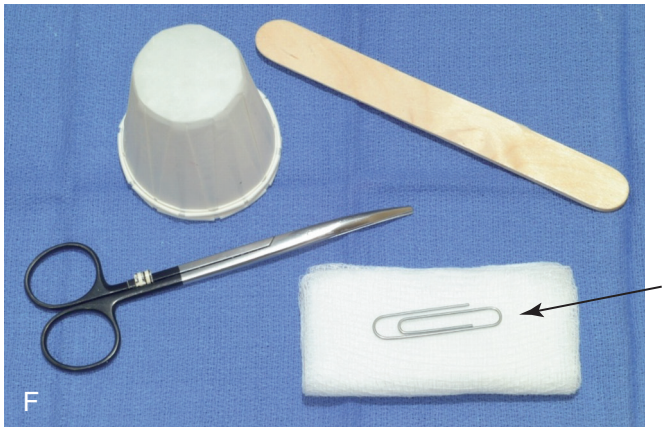


Adapt mass to the maxillary occlusal surface

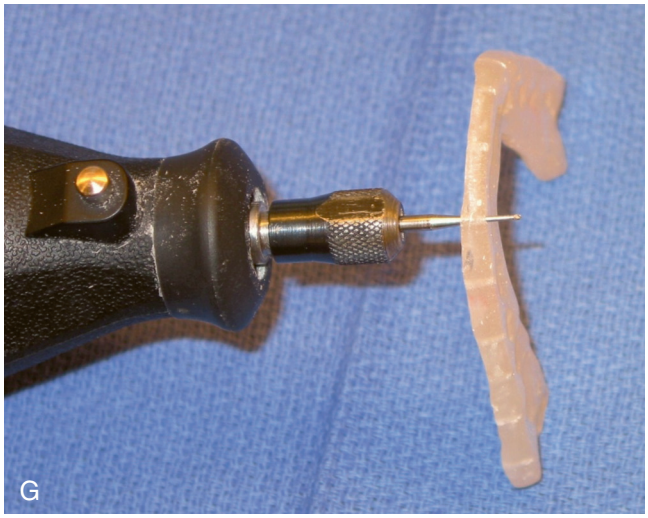


Trim soft acrylic with scissors

• **Figure 13-10, cont'd C,** Use clear acrylic liquid and orthodontic resin powder for the final splint. Mix the two together until the mass does not slump. **D,** Roll the mixed acrylic mass into a log shape. Adapt the mass to the maxillary occlusal surface. **E,** Close the articulator until the vertical pin touches in a neutral position. The mandibular occlusal surface will imprint into the acrylic as a result. Limit "undercuts" from the brackets by carefully trimming the soft acrylic with scissors.



Use additional acrylic to place wire
to reinforce palatal aspect of splint as needed



Drill holes (1.0-mm diameter) in both intermediate and final splints

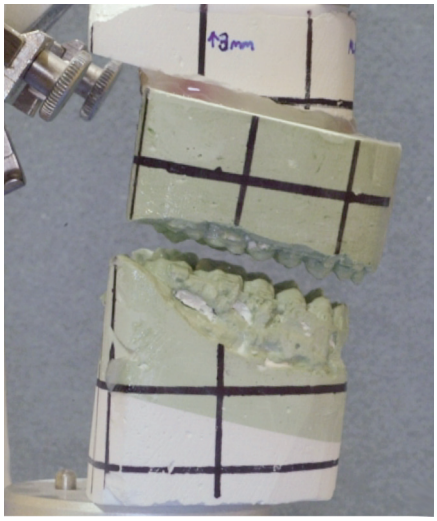
Polish splints on lathe

• **Figure 13-10, cont'd F,** Use additional acrylic to place a metal wire, if needed, to reinforce the palatal aspect of splint. This will prevent bowing or breakage of splint when segmental osteotomies are carried out and when extensive arch expansion is required. **G,** Use a lathe and a rotary drill (e.g., a Dremel drill) to trim excess acrylic from the hardened final splint. Next, place each splint (i.e., intermediate and final) in turn on the maxillary cast, and determine the best location and direction for each interdental drill hole. Complete 1-mm drill holes with the use of a rotary drill (e.g., a Dremel). **H,** Polish the splints on a lathe with the use of a cloth buffing wheel (i.e., pumice and Acrilustre) (Robert Bosch, 1800 W. Central Road, Mt. Prospect, IL).

Intermediate splint

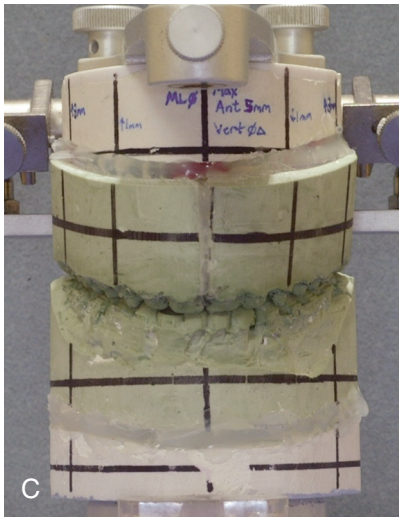
Final splint

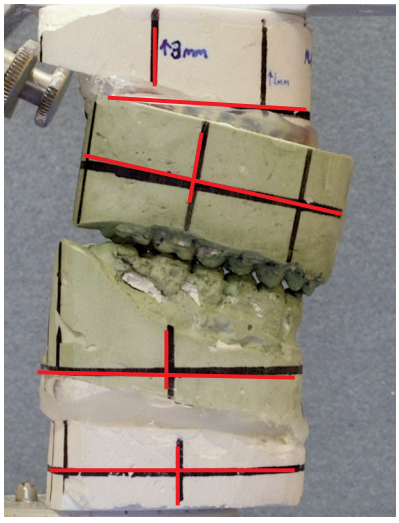
Preoperative articulated casts



Maxilla repositioned

Maxilla and mandible repositioned





Articulated dental casts after analytic model planning

• **Figure 13-11** Dental casts on the semi-adjustable articulator to demonstrate model planning and a view of the finished splints. **A,** Intermediate and final splints finished and ready for use. **B,** Lateral views of articulated dental casts first mounted in a centric relation bite and then after maxillary and final mandibular repositioning. **C,** Frontal and side views of articulated dental casts demonstrating model planning. In this case, note the extent of the maxillary clockwise rotation (3 mm) needed to achieve a preferred A-point to B-point relationship and also to overcome the procumbency of the maxillary incisors.

presurgical clinical examination, is one of the essential steps to avoid unintended errors.[10,28-35,42,43,49,58,60,64,65,71-76,82]

Controversies Surrounding Model Planning for Orthognathic Surgery

Articulator and Face-Bow Device Options

Face bow devices were developed in conjunction with *simple and semi-adjustable hinge articulators* to assist with the registering of the maxillary arch position in relation to the axis of the condylar hinge in three planes of space. The mechanics of the *face-bow device* require a tripod localization. This includes two posterior references to approximate the temporomandibular joints and then an anterior point to relate the maxillary cast three dimensionally to a selected horizontal *facial reference plane*. The posterior reference points that are generally used are the external auditory canals. The anterior point is often aligned with the orbitale (i.e., the infraorbital rims). Selection of the anterior point (e.g., the orbitale) governs the *horizontal facial reference plane*. This will in turn affect the steepness of the recorded maxillary occlusal plane on the articulator; this is discussed later in this chapter.

For orthognathic surgery, analytic model planning typically involves the use of an articulator that provides an approximation of the anatomic relationship between the hinged axis and the incisors. This type of articulator mechanism is designed to approximate the individual's actual mandibular autorotation. This is especially important for bimaxillary surgery, when autorotation of the maxillomandibular complex helps to dictate the position of the maxilla before the mandibular osteotomies are completed. Marko conducted research to demonstrate that the mathematical difference in the horizontal projection achieved when such advancement is carried out in combination with a maxillary impaction of 5 mm is significantly affected by the type of articulator that is used.[46] An average error of 2 mm was found with use of the Galetti articulator *(simple hinge),* whereas only 0.2 mm of error occurred with the Hanau articulator *(semi-adjustable)*.[46]

NOTE: The use of a semi-adjustable articulator in bimaxillary surgery will be more accurate and is therefore preferred to the use of a simple hinge articulator.

O'Malley and Milosevic compared three face-bow semi-adjustable articulator systems to measure any differences in the recorded steepness of the occlusal plane that was produced.[52] The three articulators studied were the Dentatus type ARL; the Denar MkII; and the Whip Mix Quickmount 8800. The recorded measurement of the steepness of the occlusal plane was taken as the angle between the face-bow bite fork and the horizontal arm of the articulator. This measurement was then compared with the angle of the maxillary occlusal plane to the Frankfort plane as measured via lateral cephalometry. Of the three semi-adjustable articulators tested, the Whip Mix Quickmount 8800 came closest to achieving the maxillary occlusal plane (with respect to the Frankfort plane) as measured via lateral cephalometry; it flattened the occlusal plane by only 2 degrees ($P < .05$). The results of the Dentatus and Denar MkII articulators demonstrated flattening of the occlusal plane by 5 degrees and 6.5 degrees, respectively. Ellis also demonstrated how commonly used methods of face-bow registration may erroneously capture the spatial relationship of the maxilla with the Frankfort horizontal plane.[18] This is likely to be problematic if the surgeon intends to use the Frankfort horizontal from the patient's cephalogram as the reference plane from which facial aesthetic objectives will be planned.

A critical step in model planning is the accurate recording of the relationship (i.e., angle and orientation) between a *designated facial plane* and the *maxillary occlusal plane*. As stated, capturing this orientation is accomplished with a face-bow device that is used in the clinic setting. (Note: I use the #8645 Quick Mount Face-Bow, Whip Mix Products, Louisville, KY.) The face-bow registration is then transferred to the semi-adjustable articulator for the mounting of the maxillary model. (Note: I use the #8500 Articulator, Whip Mix Products, Louisville, KY.)

I prefer to judge the desired facial (surgical) change (i.e., horizontal, vertical, roll, pitch, and yaw) and register the face bow from the *natural head position (NHP)* rather than the *Frankfort horizontal plane*.

I prefer to use the NHP orientation as it is the same view from which aesthetics of the face are typically judged by others at conversational distance. Many clinicians prefer to plan angular and linear surgical change for the patient's face from the Frankfort horizontal plane rather than the NHP. If this is done, the surgeon should register the face bow as precisely to the Frankfort line (i.e., the line between the porion and the orbitale) as possible. He or she should then meticulously attempt to mount the cast to match the orientation of the maxilla with that of the Frankfort horizontal plane.

NOTE: The importance of the face-bow semi-adjustable articulator system to the process of accurately capturing the occlusal plane with respect to Frankfort plane orientation is only relevant if the surgeon is planning facial aesthetic changes according to a strict cephalometric analysis (i.e. that also depends on Frankfort plane orientation).

Centric Relation: Definition and Capture

Definition of Centric Relation

In 1987, the definition of CR underwent a change in terminology from being considered a mandibular position in which the condyles are seated in the most "posterior

superior" position to one in which they are placed in the most "superior anterior" position in the glenoid fossa.[72] For analytic model planning in preparation for orthognathic surgery, defining the position of the condylar head during mandibular protrusion and lateral excursions is not critical. However, it is essential to locate a mandibular position that is reproducible with the teeth in occlusion. This must be obtained without the assistance of the patient (i.e., with fully relaxed masticatory muscles), and it needs to be reliably accomplished both preoperatively in the clinical setting and intraoperatively with the patient under anesthesia. This position of the mandible with the teeth in occlusion is usually registered in wax (i.e., a wax bite) and then transferred to the articulator. This is an important step for analytic model planning to be accomplished accurately.

An erroneous inconsistent recording of CR will lead to the malpositioning of the maxilla or mandible during surgery. A survey study conducted by Truitt and colleagues confirmed that 98% of oral and maxillofacial surgeons define CR as the most posterior superior positioning of the mandible.[79] Interestingly, a majority of the orthodontists surveyed agree with the surgeons' definition of CR, but there was less consistency.[79] According to the survey, surgeons and orthodontists do agree that dental casts of the teeth for orthognathic planning should be mounted in CR.

Capturing of Centric Relation

Hellsing and McWilliam used a *subtraction technique* to assess the repeatability of the CR recorded with a *one-handed push-back technique*.[31] They found that the mandibular condyles can be seated in a reproducible position, which is well suited for the registration of CR. Campos and colleagues compared the *swallowing technique* with the *chin-point guidance technique* with the patient in both upright and supine body positions and found that both techniques established a physiologic CR in a reproducible manner.[86] Interestingly, when the chin-point guidance method was used, the work of these authors suggested that reliability was greater with the patient in the upright, seated patient as compared with the supine position.

Simon and Nicholls compared the chin-point guidance, *chin-point guidance with ramus support,* and *bimanual manipulation* techniques.[68] They found that there were no significant differences in the range of mandibular positions that were observed. Interestingly, their study patients did not include individuals with jaw deformities. It is known that patients with specific patterns of jaw deformity (e.g., Class II mandibular retrognathism) are more likely to involuntarily posture the mandible forward during the swallowing technique.[32,33]

The *chin-point guidance technique* is accomplished by using one hand to apply pressure to the subject's chin and the other hand to apply counter pressure to their occiput. This allows the surgeon to apply posterior and superior "vectored" force to the anterior aspect of the mandible. By doing so, the condyles will be positioned in a superior-anterior location within each glenoid fossa (i.e., CR) (see Fig. 13-2).

NOTE: I find the *chin-point guidance technique*—when used both preoperatively in the clinic setting and intraoperatively by the same surgeon—to be both reliable and convenient for the orthognathic surgery patient. This is the method that I have adopted and that I use successfully in clinical practice.

Bite Registration Material

The material used to record CR (e.g., soft wax) is also considered as a potential source for the introduction of error. Adrien and Schouver studied mandibular model-mounting errors; they demonstrated that wax can create error and that the error is directly proportional to the wax's thickness.[2] They suggest that the wax between the approximating teeth be as thin as possible. In addition, the wax should not be punctured, because this may result in sliding from CR to centric occlusion (CO). The wax should also retain its rigidity to avoid the distortion of contact points. The wax bite must be protected after it is removed from the mouth, because it is easily deformed. Ideally, it is used without significant delay to the time that the models are mounted on the articulator (see Fig. 13-2).

Inaccuracies of Osteotomy Site Measurements when used in Isolation to Reorient the Maxilla in Bimaxillary Surgery

The *face-bow device* is used to spatially orient patients' dental models on a *semi-adjustable articulator*. The belief that measurements may then be made at the equivalent of the Le Fort I osteotomy site to accurately reposition or reorient the maxillary cast on the articulator without the use of the *Erickson Model Table* is flawed. The errors of this model surgery and splint-construction approach would then be further compounded if the surgeon relied on actual Le Fort I osteotomy site measurements during operation as a gauge of where to reorient the maxilla without the assistance of *external facial reference landmarks* and measurements.[13,28,36,40,41,53,56,57,59,69] The use of these methods (i.e. osteotomy site measurements) to reposition the maxilla in the dental laboratory before splint fabrication and then during surgery before plate and screw fixation may work for uncomplicated movements (e.g., limited horizontal and vertical repositioning), but it is likely to become grossly flawed when trying to accomplish complex yaw, pitch, and roll orientation changes of the midface.

Accuracy of Current Analytic Model-Planning Techniques to Achieve Desired Results

The *Erickson Model Table* is a device that is used to document the baseline three-dimensional orientation of

the mounted maxillary model on the semi-adjustable articulator. (Note: I use the Erickson Model Block and Platform Model Measuring Kit designed by Great Lakes Orthodontics Products, Tonawanda, NY.) The Erickson Model Table is then used to assist the three-dimensionally reposition and reorient the maxillary cast to the desired facial (surgical) change.[19,20] The reoriented maxillary model on the articulator is then used to construct the intermediate splint. In the operating room, the use of the *prefabricated intermediate splint* in conjunction with precise *measurements of the anterior vertical dimension of the midface* (e.g., direct vertical measurement from the medial canthus to the maxillary incisor on each side of face) has eliminated many of the inaccuracies that were previously experienced during model planning and surgical execution (see Fig. 13-6, *C*).[20,27,36,40,41,51,53,56,57,66,81]

Interestingly, the few studies published in the literature show a wide range of variation between what was planned by the clinician before surgery and what is then actually achieved during operation with reference to the advancement of the maxilla after Le Fort I osteotomy during bimaxillary surgery. An exception is the study of **Gil and colleagues,**[28] who completed a retrospective study to evaluate the predictability of maxillary repositioning (i.e., vertical and horizontal change measured at the maxillary incisors) after Le Fort I osteotomy during bimaxillary surgery. They used analytic model-planning techniques, including the following: 1) face-bow transfer 2) the use of a semi-adjustable articulator 3) the use of Erickson Model Table measurements 4) intermediate splint construction and 5) the use of external facial height reference points during surgery. A consecutive series of patients (n = 32) were analyzed, with measurements taken with consistent lateral cephalometric radiographs before and 1 week after surgery.[69] The results indicate less than a 1-mm difference between the planned and the early postoperative results for both vertical and horizontal repositioning in all patients. **Stanchina and colleagues** also carried out a retrospective study to measure the accuracy of external reference points in conjunction with model planning to reposition the maxilla during bimaxillary surgery.[69] The study subjects (n = 41) showed a surprisingly consistent mean difference between predicted and achieved vertical repositioning of the maxilla (0.36 mm; standard deviation, 0.26 mm). Their success with horizontal repositioning was less accurate. The mean horizontal variance was 1.4 mm (standard deviation, 1.63 mm).

NOTE: The reasons for variation between the planned and actual maxillary advancement in most published studies remain unclear. This is because the descriptions of the key steps that they used are generally not fully described.

Review of Key Steps for Successful Analytic Model Planning and Surgical Execution

1. *The direct visual examination of the patient by the surgeon in the clinic setting.* With the use of facial analysis, key measurements of the individual's maxilla in relationship to the upper face (i.e., the forehead, external ears, and orbits) are recorded. These measurements will confirm variations from normal and define the surgical objectives (see Fig. 13-1, *H*).
2. *The use of a face-bow device to record the spatial orientation of the upper face and condyles with regard to the maxilla.* This is generally done with the use of the external auditory meatus for two of the reference landmarks. The anterior aspect of the face bow (i.e., the frontal view) is adjusted to be parallel with the interpupillary line. With the patient's head and neck in the NHP, the lateral aspects of the face bow are then adjusted to orient the temporal arms to be parallel with the floor (see Fig. 13-3 C and D).
3. *The transfer of the spatial orientation of the maxilla to the semi-adjustable articulator.* This is done with the arms of the face-bow and the upper arm of the articulator parallel to the floor (counter top) (see Fig. 13-4 A).
4. *The taking of an accurate CR bite registration of the patient in the clinic setting.* This is then used to orient the dental models to each other on the semi-adjustable articulator (see Figs. 13-2 and 13-4, *C*).
5. *The use of the Erickson Model Table in the dental laboratory to establish the baseline spatial orientation of the maxillary cast* (see Fig. 13-6, *C*).
6. *The use of the Erickson Model Table to accurately reorient the upper jaw model on the articulator into the desired surgical position* (see Fig. 13-8, *A*).
7. *The fabrication of a splint for use during surgery* to achieve the planned maxillary reorientation (see Fig. 13-9)
8. *The use of external facial measurements* (e.g., the distance from the medial canthus to the maxillary incisor) during operation to establish the baseline vertical position of the maxilla within the upper face and then to accurately reorient the upper jaw to achieve surgical objectives (see Fig 15-18, *A*).
9. *The accurate location of the terminal hinge position of the condyles during operation.* This is done to reproduce the position of the condyles when taking the CR bite registration in the clinic setting, and it is essential before rigid fixation is secured across each osteotomy site (see Fig 15-37, *B*).

COMMENT: The surgeon no longer attempts to correlate millimeter changes at the Le Fort I osteotomy site with the expected facial morphologic outcome. When properly executed, the accuracy of analytic model-planning techniques as described for precise orthognathic surgery has been documented. This is discussed in more detail later in this chapter.

The Role of Computer-Aided Software Design and Model Planning

For a variety of reasons, some patients with complex maxillofacial skeletal deformities undergo treatment that achieves suboptimal results. Despite the surgeon's best efforts, it can be difficult to assess the preferred surgical spatial reorientation of the jaws for the achievement of ideal facial projection and symmetry. Although standard analytic model planning (as described in this chapter) has proven to be effective for most orthognathic surgical problems, three-dimensional deformities of pitch, roll, and yaw continue to confound the ability to achieve predictable results in each and every patient. The current standard treatment planning process is also felt to be time consuming and labor intensive, especially for those clinicians who do not evaluate dentofacial deformities and execute orthognathic surgery as significant components of their practice.

Recently, surgeons have begun looking to computer-aided design and computer-aided modeling software to assist with the planning and implementation of orthognathic procedures.[3-5,7,11,23,24,25,45,47,48,61,67,70,83,84] These technologies allow clinicians to import two-dimensional computed tomography data in a digital imaging format at a computer work station and to then generate a three-dimensional representation of the patient's skeletal and soft-tissue anatomy. The data set can be used to mill a stereolithographic model, or it can be manipulated on the computer screen for purposes of analysis and treatment planning. The ability of virtual model planning (as opposed to analytic model planning, as described in this chapter) to routinely achieve an improved end result for the orthognathic patient has yet to be confirmed. It is also unclear whether the higher costs of virtual model surgery planning can be offset by savings in the clinician's time that would otherwise be required for analytic model planning.

A successful orthognathic outcome is often judged by the achievement of a preferred occlusion, an open upper airway, and enhanced facial aesthetics. Any methods that are proven to better achieve these objectives over the long term with efficiency and reliability are welcomed. Unfortunately, current virtual model surgery planning still requires that alginate impressions of the maxilla and mandible be taken, that plaster models be made of the maxillary and mandibular arches, and that ideal occlusion be hand articulated and marked by the surgeon into the set final occlusion. If segmental osteotomies are required, the surgeon must also cut the plaster models and secure them together as required to achieve the set final position. Furthermore, a computer-generated surgical splint has not yet been proven to be fully reliable for complex, double-jaw surgical cases.[11] Problems that remain to be addressed include the following: the identification of internal reference systems; the making of three-dimensional measurements; the management of interferences by metal (orthodontic) hardware; the orientation of computed tomographic models to the NHP; and the necessity of capturing the computed tomography data while the patient is in CR.

Standard Facial Records for Orthognathic Surgery

- Standardized facial and occlusal view photographs
- Lateral cephalometric radiograph
- Panoramic radiograph
- Alginate impressions of the maxilla and mandible
- CR wax bite registration
- Face-bow registration for transfer to a semi-adjustable articulator
- Facial measurements taken via a direct visual examination (Fig. 13-1H).
- Analytic model planning data entry form (Fig. 13-6C)
- Cone-beam computed tomography scanning (patient specific)
- Full-mouth periapical radiographs (patient specific)

Key Measurements Obtained via Direct Visual Examination to Define Planned Maxillary Surgical Change

- Horizontal change at incisors _____ (mm)
- Vertical change at incisors _____ (mm)
- Midline change at incisors _____ (mm)
- Maxillary plane change (total) _____ (mm)

Clockwise—Counterclockwise

- Maxillary cant correction (total) _____ (mm)

  ↑ ↓ _____on right ↑ ↓ _____on left

- Vertical change at T#3 (right) molar ___ + ___ + ___ = ___ (mm) (Base + Cant + Plane)
- Vertical change at T#14 (left) molar ___ + ___ + ___ = ___ (mm) (Base + Cant + Plane)

Face-Bow Registration and Transfer to the Semi-Adjustable Articulator

An 18-year-old recent high school graduate who presented to this surgeon for the evaluation of an uncorrected dentofacial deformity that was characterized as maxillary deficiency with relative mandibular excess is described. He is shown to demonstrate the technique of analytic model planning as typically carried out (see Figs. 13-1, 13-12, and 13-13).

Face-Bow Registration
(see Figs. 13-2, *A*, through 13-3, *G*)

- Place soft wax on upper side of the face-bow bite fork.
- The midpoint between the central incisors (interproximal) should match the center of the bite fork.
- Register the patient's maxillary occlusion into the soft wax on the bite fork.

- Place an ear prong in each ear canal, and hold the temporal extensions with even pressure medially, superiorly, and forward. Tighten the three thumb screws of the temporal extensions.
- In the frontal view, the face bow is initially adjusted to be parallel with the pupils and in proximity to the infraorbital rims.
  - If the orbits are asymmetric (e.g., in a patient with hemifacial microsomia or the Klippel–Feil anomaly), do not use the interpupillary line as a frontal reference plane. Instead, use the NHP as the facial reference plane.
- In the profile view, the face bow is adjusted to be parallel with the floor while the patient is in the NHP.
  - If the ear canals are asymmetric, do not rely on them for face-bow orientation. For example, for a patient with hemifacial microsomia, if the ear canal on one side is not present or is abnormally located, then place the face-bow ear prong in the appropriate position to approximate the contralateral ear canal. The face bow should be parallel with the floor while the patient is in the NHP (i.e., the NHP is the reference plane).

Text continued on p. 415

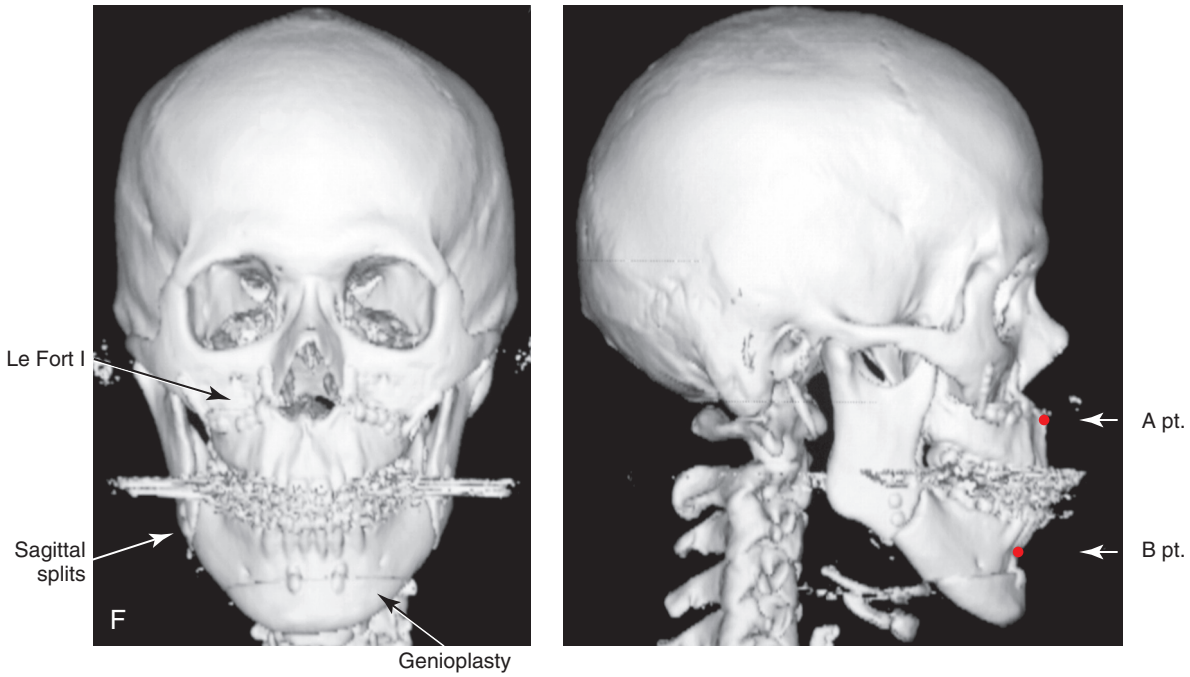
• **Figure 13-12** The patient is shown before and just 5 weeks after surgery. At this time, adequate bone healing has occurred to allow for a return to a more normal diet and physical activity level. The patient is also returned to the care of his orthodontist. **A,** Frontal views in repose before and 5 weeks after surgery. **B,** Frontal views with smile before and 5 weeks after surgery. *Continued*

• **Figure 13-12, cont'd C,** Oblique facial views before and 5 weeks after surgery. **D,** Profile views before and 5 weeks after surgery.

Prior to orthodontics

Pre surgery

5 weeks after surgery



• **Figure 13-12, cont'd E,** Occlusal views before treatment, immediately before surgery, and just 5 weeks after surgery at the time of splint removal. **F,** Computed tomography scan views 5 weeks after surgery. The sites of the maxillary, mandibular, and chin osteotomies and the location of standard fixation can be seen.

• **Figure 13-13** The same patient is shown before and then 1½ years after surgery. **A,** Frontal views in repose before and 1½ years after surgery. **B,** Frontal view with smile before and 1½ years after surgery.

• **Figure 13-13, cont'd C,** Oblique facial views before and 1½ years after surgery. **D,** Profile views before and 1½ years after surgery.

Continued

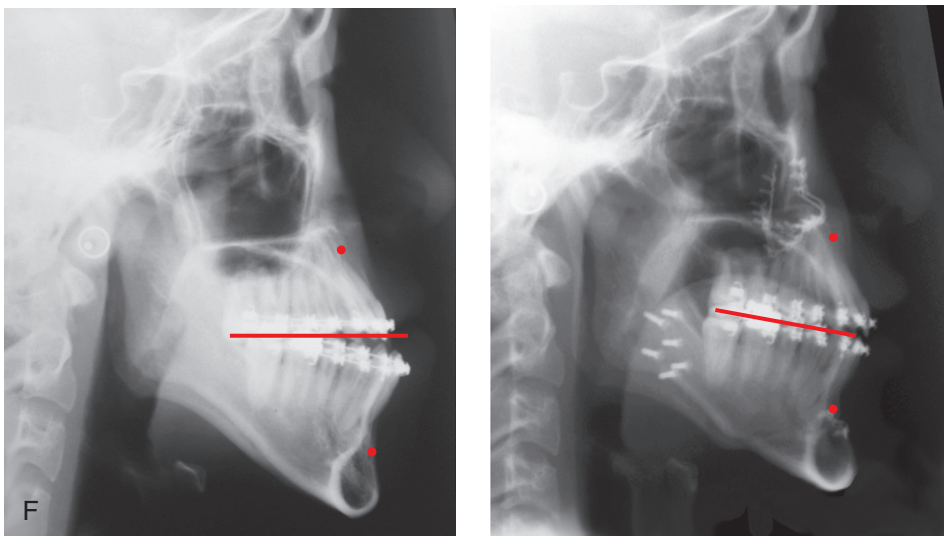
Prior to orthodontics

Pre surgery

5 weeks after surgery

1½ years after surgery

• **Figure 13-13, cont'd E,** Occlusal views before treatment, immediately before surgery, and then 5 weeks and 1½ years after the completion of treatment.



• **Figure 13-13, cont'd F,** Lateral cephalometric views before and after surgery.

- Firmly hold the bite fork to the maxillary occlusal surfaces (through the soft wax). Pressure is applied to the bite fork by the patient with the use of a thumb from each hand. When this is accomplished, do the following:
  - Loosely connect the bite fork, (which is held firmly to the patients maxillary teeth), to the face-bow apparatus, (which is held in place by the clinician), with the use of the T-screws on the vertical rod.
  - In profile and with the patient in the NHP, adjust the face-bow temporal extensions to closely parallel the floor.
  - Tighten the two T-screws to secure the bite fork to the vertical rod of the face-bow apparatus.
- Now that the face-bow registration is complete, loosen the three thumb screws of the temporal extensions to release the ear prong from each ear canal. Remove the face bow from the patient for transfer to the semi-adjustable articulator.

Take Alginate Impressions, Pour the Impressions in Stone, and Trim the Casts
(see Fig. 13-3, *H* through *K*)

- Take accurate alginate impressions of the maxilla and the mandible.
- Pour the alginate impressions in green stone, and incorporate at least 30 mm of base. Base formers are helpful to accomplish adequate base height. To limit air bubbles, use a vibrator when pouring stone into alginate impressions.

NOTE: The use of stone rather than plaster improves strength, with less porosity and less breakage.

- When the casts are dry (≈15 min), remove the alginate and base former from each stone cast. Measure 30 mm of base on the mandibular cast and 25 mm of base on the maxillary cast. Measure up from the following locations:
  - The first molar cusp tip on each side
  - The canine cusp tip on each side
  - The incisor cusp tip on each side
- Use a model grinder to trim at the 25-mm and 30-mm circumferential markings, respectively, for the maxillary and mandibular casts.
- Remove all blebs from the occlusal surface of the dental casts.

Face-Bow Transfer: The Orientation of the Dental Casts (Maxilla and Mandible) on the Semi-Adjustable Articulator
(see Fig. 13-4, *A* through *F*)

- Secure the face-bow device to the semi-adjustable articulator. Adjust both the face-bow device and the upper arm of the articulator to be parallel to the floor and the counter lap.
- Secure the plastic base plate to the dental articulator. Place the maxillary cast on the bite-fork component of the secured face-bow device. Use white plaster to mount the maxillary cast to the base plate.
- Use the bite-fork support device to prevent the sagging of the maxillary cast during this process. Let the plaster initially set (≈15 min), and then remove the face-bow apparatus and the bite-fork support device.
- Turn the articulator upside down, and secure the mandibular cast to the maxillary unit with the use of the CR wax bite registration taken in the clinic setting. Further secure the occlusion in CR with two rubber bands to

compress the maxillary unit and mandibular cast together through the bite registration.
- Place the plastic base plate onto the dental articulator, and then use white plaster to mount the mandibular cast to the base plate. Let the plaster initially set (≈15 min).
- Cut the rubber bands used to secure the occlusion, and remove the wax bite.
- Remove the maxillary unit from the articulator. With the use of a model grinder, trim the maxillary cast, plaster base and the base plate all as one unit.
- Remove the mandibular unit from the articulator. With the use of a model grinder, trim the mandibular cast, the plaster base and the base plate all as one unit.

Placement of Reference Lines on the Maxillary and Mandibular Units (see Fig. 13-5, *A* through *E*)

- With the use of the Erickson Model Table, "score" two horizontal reference lines on the maxillary unit and then on the mandibular unit. Locate the horizontal reference lines on the maxillary and the mandibular units by doing the following:
  - Place the first horizontal line within the green stone.
  - Place the second horizontal line within the white plaster.
  - Ideally, there will be 15 mm to 20 mm of separation between these lines.
- With the use of a thick, black Sharpie pen, mark the "scored" horizontal circumferential reference lines on both the maxillary and mandibular units.
- With the use of a thick, black Sharpie pen, mark the vertical reference lines on both the maxillary and mandibular units. The vertical lines are located perpendicular to the horizontal lines and in the following places:
  - At the cusp tip of each canine
  - At the mesiobuccal groove of each first molar
  - Between the central incisors (i.e., at the dental midline)
  - Posterior at each tuberosity or retromolar area

Use of the Erickson Model Table to Define the Spatial Orientation of the Articulator-Mounted Maxillary Unit* (see Fig. 13-6)

With the use of a thin, black Sharpie pen, locate specific reference points from which measurements will be made. Place a dot at the following reference points:

- The cusp tip of each maxillary canine
- The mesiobuccal cusp tip of each maxillary first molar
- The midpoint of the incisal edge of each maxillary central incisor

Baseline vertical (maxillary) measurements: With the model block on its base, use the electronic caliper to measure the distance (in mm) at the following areas: (see Fig. 13-6 E and F)

- The incisal edge of each maxillary central incisor (#8, #9)
- The mesiobuccal cusp tip of each maxillary first molar (#3, #14)

Baseline horizontal (maxillary) measurements: With the model block on its end, use the electronic caliper to measure the distance (in mm) at the following area: (see Fig. 13-6D).

- The incisal edge of each maxillary central incisor (#8, #9)

Baseline dental midline (maxillary) measurements: With the model block on its side, use the electronic caliper to measure the distance (in mm) at the following areas: (see Fig. 13-6G)

- The dental midline with the left side of the cast down
- The dental midline with the right side of the cast down

Use of the Erickson Model Table to Define the Spatial Orientation of the Articulator-Mounted Mandibular Unit

Baseline horizontal (mandibular) measurement: With the model block on its end, use the electronic caliper to measure the distance (in mm) at the following area:

- The incisal edge of the mandibular central incisors (#24/#25)*

Separation of the Maxillary Cast from the Articulator Base (see Fig. 13-7, *A*)

- Remove the maxillary unit from the articulator. Separate the maxillary cast from the plaster base over a protective surface. You may first use the die saw to cut a groove in between the green stone and the plaster base. Insert a knife into this groove, and tap the knife with a hammer.

*The Erickson Model Table maxillary unit measurements that I find useful are outlined earlier in this chapter. Other measurements can be made in accordance with the clinician's preference.

*This is the only Erickson Model Table mandibular unit measurement that I find useful. Other measurements can be made in accordance with the clinician's preference.

Segmentation of the Maxilla
(see Fig. 13-7, *A*, *B*, and *C*)

Segmentation and Subsequent Reorientation of the Maxilla

- Use a die saw to segment the maxilla, as planned (i.e., two pieces versus three pieces).
- After the maxilla is segmented, align the segments over the mandibular arch into the preferred occlusion. Secure the segments back together with the use of hot glue from a glue gun.
  - Occlusal equilibration may be indicated to achieve the preferred occlusion (e.g., plunging palatal cusps on maxillary molars, bicuspid cusp reduction to level the posterior arch). If so, use a sharp plaster knife to complete adjustments on the occlusal surfaces of the maxillary and mandibular casts. Use a red Sharpie pen to mark the exact locations where enamel adjustments were made. The actual equilibration is carried out in the operating room.
  - When repositioning the maxillary segments into occlusion with the mandibular arch, it is essential to maintain the physiologic positioning of the interdental papilla and the contact points of adjacent teeth at all interdental osteotomy sites. If dental gaps are created or if cervical margins are not reasonably aligned, then periodontal sequelae are more likely.

Measurement of the Maxillary Arch Width Before and After Segmental Osteotomies

Transverse distance between maxillary molars (#3 to #14)
- Initial _____ (mm)
- After segmentation _____ (mm)

Transverse distance between maxillary canines (#6 to #11)
- Initial _____ (mm)
- After segmentation _____ (mm)

Use of the Erickson Model Table Measurements to Reorient the Maxillary Cast
(see Fig. 13-8, *A* through *F*)

- The repositioning of the maxilla on the articulator is meant to accurately match the desired surgical changes. These changes have been determined in accordance with the following: 1) the patient's clinical objectives (i.e., preferred facial aesthetics and improvements in function) 2) by taking into account any biologic skeletal, dental, or soft-tissue limitations and 3) the surgeon's clinical, dental, and radiographic analyses (see Chapter 12).
- After the key desired adjustments to the midface are reproduced on the maxillary unit, they are verified with use of the Erickson Model Table. The key millimeter and angular changes to be confirmed include the following:
  - Maxillary (midface) projection
    - Horizontal change at incisors
  - Maxillary incisor shown in repose and with smile
    - Vertical change at incisors
  - Maxillary plane change (pitch orientation) and cant correction (roll orientation), which are controlled by the following:
    - Vertical change at incisors
    - Vertical change at first molars
  - Maxillary midline position (yaw orientation)
    - Dental midline change (right side down)
    - Dental midline change (left side down)
  - Considerations of lateral tuberosity position (yaw orientation)
    - Analysis of lateral shift (change) at posterior maxilla (tuberosity region)
- Soft wax or clay is used in a tripod fashion to facilitate the initial placement of the maxillary cast back onto the plaster base on the articulator in the preferred position. It is generally necessary to trim the stone or plaster before repositioning. Fine tune the maxilla into the preferred position with the use of the Erickson Model Table (i.e., via horizontal, vertical, and transverse measurements). After these measurements are confirmed, fully secure the preferred maxillary position to the plaster base with hot glue from a glue gun.
- When repositioning the maxilla into the preferred position, take into consideration the yaw orientation. Control the yaw orientation to avoid unwanted asymmetric shifts, which would be visually noticeable in the cheeks of the midface and at the mandibular angles (see Chapter 12).

Intermediate Splint Construction for the Positioning of the Maxilla During Operation (see Fig. 13-9, *A* through *E*)

- With the maxilla secured in the new preferred position but without any changes yet made to the baseline mandibular position, the intermediate (occlusion) splint is constructed.
- Apply a thin coat of Vaseline to the maxillary and mandibular occlusal surfaces with a toothbrush.
- Use pink acrylic liquid and standard acrylic powder. Mix these until the mass does not slump. Apply Vaseline to your hands, and roll the mass into a log shape. Adapt the mass to the maxillary occlusal surface, and close the articulator until the vertical pin touches. This will result in an imprint of the mandible occlusal surfaces on the opposite side.
- Maintain the acrylic just below the level of the base of the maxillary orthodontic brackets and just above the base of the mandibular orthodontic brackets. Use sharp

NOTE: When making the intermediate splint, maintain the vertical pin of the articulator in a neutral position, unless significant inferior positioning (i.e., lengthening) of the maxilla is planned. If so, there are two options: either take the initial CR bite (in the clinic setting) with an increased freeway space (this is equivalent to the clockwise rotation of the mandible while the condyles remain seated in the fossa) or lengthen the vertical pin on the articulator (this is also equivalent to the clockwise rotation of the mandible while the condyles remain seated in the fossa) before making the intermediate splint. Either approach can be used without error as long as the mandibular opening is limited to temporomandibular joint rotational movement. If temporomandibular joint transitional movement occurs, then the splint will no longer reflect the patient's CR bite. If the clinician is uncomfortable with this approach, then a mandible-first approach to analytic model planning should be used (see Chapter 14).

scissors to remove gross excess. This will prevent "undercuts" of acrylic on the brackets.
- Continuously open and close the articulator until the acrylic sets (≈10 min). Maintain the vertical pin in a neutral position and fully closed. This will ensure that no "undercuts" remain.

Trimming of Hardened Acrylic Intermediate Splint (see Fig. 13-9, *F* and *G*)

- Remove excess buccal (labial) acrylic when looking at the model from a bird's-eye view. Use a pencil to mark a line parallel to and just outside of the maxillary orthodontic brackets. Draw a similar line outside of the mandibular brackets. With a lathe and a circular white stone or an acrylic bur, trim excess acrylic, as marked.
- Prevent lingual ramping by removing excess acrylic in the region of the mandibular anterior teeth. This limits interferences that tend to occur as a result of the arc of rotation of the mandible during mouth opening and closing.
- When viewing the splint from the side, mark with a pencil any areas that require further acrylic removal (e.g., just below the base of the maxillary orthodontic brackets, just above the base of the mandibular orthodontic brackets). With a lathe and a circular white stone or an acrylic bur, trim excess acrylic, as marked.

NOTE: If greater strength is needed for the intermediate splint (see Fig. 13-10, *F*), then reinforce with a palatal wire (e.g., use a piece of a heavy paper clip) and additional acrylic.

- If segmental osteotomies are planned, maintain at least 3 mm to 4 mm of lingual acrylic behind the incisors to prevent splint breakage or torque, which may otherwise occur in the operating room.

Reorientation of the Mandibular Cast to Register the Final Occlusion (see Fig. 13-10, *A* and *B*)

- Separate the mandibular cast from the plaster base (as was done for the maxillary cast).
- Articulate the mandibular cast with the maxillary unit in the desired final occlusion, and secure the occlusion with hot glue.
- With the vertical articulator pin remaining in a neutral position, secure the mandibular cast back to plaster base. This may require the removal of interferences in the plaster base or the addition of material (e.g., plaster, hot glue).
- Remove the glue that was used to secure the occlusion, and confirm the accuracy of the final occlusion.

Final Splint Construction for the Positioning of the Mandible During Operation (see Fig. 13-10, *C* through *F*)

- Reapply Vaseline to the maxillary and mandibular occlusal surfaces of the teeth with a toothbrush.
- Mix clear acrylic liquid and standard powder. Mix these until the slumping stops. Vaseline your hands, roll the acrylic into a log shape, and then adapt and readapt it as described for intermediate splint construction.
- Trim the final occlusion splint as described for the intermediate splint. Smooth the lingual edges for patient comfort.
- If segmental osteotomies are planned, maintain at least 3 mm to 4 mm of lingual acrylic behind the incisors to prevent splint breakage or torque, which may otherwise occur in the operating room.

NOTE: If greater strength is needed for the final splint (see Fig. 13-10, *F*), then reinforce with a palatal wire (e.g., use a piece of a heavy paper clip) and additional acrylic.

Finishing of the Prefabricated Acrylic Splints (see Fig. 13-10, *G* and *H*, and Fig. 13-11, *A* and *B*)

- Place each splint in between the maxillary and mandibular units to determine the ideal location and direction of each interdental hole placement. Mark these placements with a pencil.

- Drill holes that are 1 mm in diameter in both the intermediate and final splints at each interdental location for the ease of passage of the wires through the splint. For ease of wire passage, the holes are located below the maxillary orthodontic arch wire.
- Polish the splints on the lathe with a cloth buffing wheel using pumice (i.e., coarse, medium, and then fine) and Acrilustre (Vogel Paint and Wax Co. Inc., 1020 Albany Place, SE; Orange City, Iowa)

Accuracy of the Described Analytic Model Planning Technique: Review of Study

A research study was carried out to assess the accuracy of analytic model planning to achieve the desired maxillary advancement during bimaxillary orthognathic surgery.[54]

Material and Methods

A consecutive series of patients (n = 20) who underwent at a minimum Le Fort I osteotomy with advancement and sagittal ramus osteotomies of the mandible for the correction of dentofacial jaw deformities were included in the study group. The sample consisted of female (n = 9) and male (n = 11) with a mean age of 21 years (range, 14 to 45 years). All study subjects underwent the following: 1) preoperative and 5-week postoperative lateral cephalometric radiographs of acceptable quality with a metric reference frame for analysis; 2) consistent presurgical facial analysis by the surgeon, including millimeter decisions regarding planned horizontal and vertical repositioning and three dimensional reorientation (i.e., pitch, roll, and yaw) of the maxilla; 3) consistent analytic model planning as described in this chapter 4) the construction of intermediate and final splints; 5) the use of the prefabricated intermediate splint to establish the maxillary positioning after Le Fort I osteotomy and before plate and screw fixation, and 6) the use of consistent intraoperative landmarks (i.e., the distance between the medial canthus and the maxillary central incisor measured in millimeters with the use of calipers) to confirm vertical orientation.

The determination of the preferred surgical reorientation of the jaws was based on a combination of the direct visual examination of the patient in the clinic setting, the analysis of the lateral cephalometric radiograph, and the analysis of the profile facial photograph taken in the NHP during the presurgical office visit. The extent of preferred maxillary horizontal advancement in millimeters was ultimately assessed by the surgeon with the patient in the NHP during the direct visual examination, with all gathered data taken into account. The fabrication of an intermediate splint to capture the preferred maxillary horizontal and other vector movements (i.e., pitch, yaw, and roll) with the use of consistent analytic model surgery planning methods was accomplished.

Radiographic Analysis

For each study patient, a presurgical standard cephalometric radiograph was obtained for later analysis. At 5 weeks after surgery, a similar standard cephalometric radiograph was also taken. This was timed to coincide with initial bone healing and the removal of the surgical splint.

Four landmark points (i.e., the sella, the nasion, the porion, and the orbitale) that remained unchanged after Le Fort I advancement were used for this analysis. These points were used to create a consistent reference line from which linear measurements (e.g., a change in the horizontal position of the maxillary incisors) could be made and compared before and after maxillary advancement. To confirm the consistency of the four designated points in the sequential radiographs, a reference angle between SN and FH was verified to remain within 0.5 degrees.

On each cephalometric radiograph, a perpendicular reference line passing through the sella, called the *sella perpendicular (S⊥)*, was then dropped. From S⊥, a direct linear measurement was made parallel to the Frankfort horizontal *(FH)* plane and extended to the maxillary incisal edge *(U1)* (Fig. 13-14).

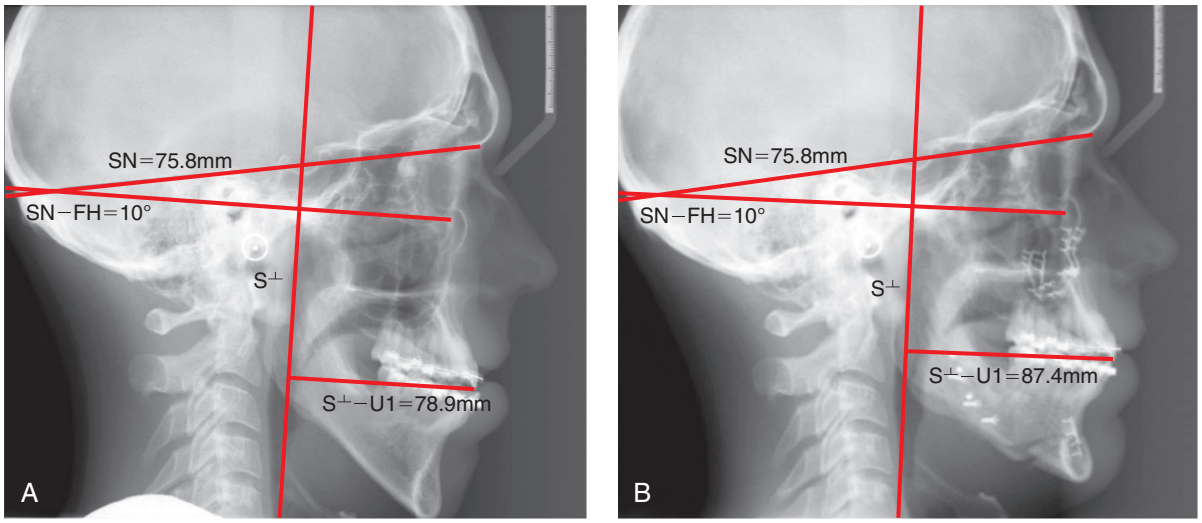
To accurately access the horizontal advancement of the maxillary incisors accomplished during the operation and maintained at 5 weeks, the distance in millimeters from S ⊥ to U1 was measured on both the preoperative and the 5-week postoperative lateral cephalogram. The measurements were then adjusted with the use of a metric reference frame to correct for magnification. Any difference in length of these two data points corresponds with variations in the preoperative and postoperative (i.e., at 5 weeks after surgery) horizontal positions of the maxillary central incisors.

Comparisons between the planned and actually achieved (at 5 weeks after surgery) horizontal positions of the maxillary incisors were made with the paired *t*-test. Statistical significance was set at $P < .05$. Associations between the planned and postsurgical results were investigated with Pearson's correlation.

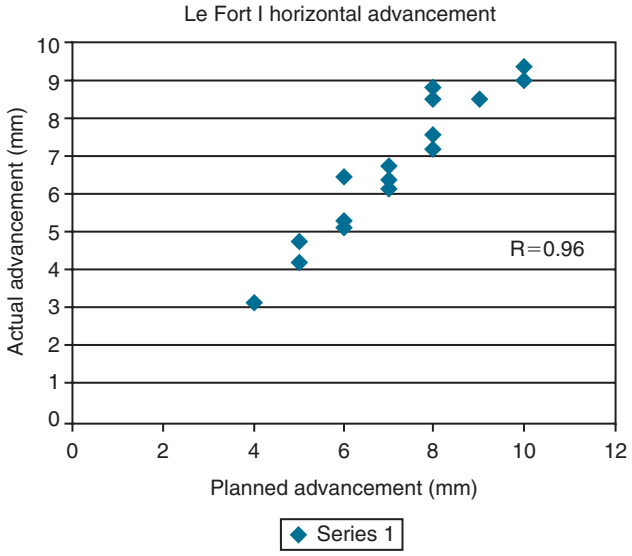
Results

The collected data for all study patients are presented in Table 13-1. In all cases, the surgically achieved and maintained (i.e., at 5 week postoperatively) horizontal advancement measured at the maxillary incisors was within 1.0 mm of what was planned. There was a strong correlation between the two data sets (R = 0.96) (Fig. 13-15). With the use of the absolute measurement values, the mean difference between the planned and actual 5-week postsurgical horizontal advancement of the maxilla was found to be 0.6 mm (range, 0.2 to 1.0 mm). According to the paired *t*-test, this mean difference was not considered to be statistically significant ($P > .05$).

The distribution of the absolute difference in millimeters between the planned, actually achieved, and then



• **Figure 13-14** Example of lateral cephalometric radiographs and measurements taken in study patients showing maxillary advancement in millimeters. Lateral cephalometric radiographs **A,** before and **B,** after Le Fort I advancement. *From Park N, Posnick JC: Accuracy of analytic model planning in bimaxillary surgery,* Int J Oral Maxillofac Surg. *42:807-814, 2013.*



• **Figure 13-15** Data from study patients undergoing Le Fort I advancement. The correlation between the planned and actual advancement at the maxillary incisors is shown (R = correlation coefficient). *From Park N, Posnick JC: Accuracy of analytic model planning in bimaxillary surgery,* Int J Oral Maxillofac Surg. *42:807-814, 2013.*

maintained maxillary advancement is presented in Table 13-2. For the majority of patients, the difference fell within 0.4 to 0.8 mm. In 17 of the 20 patients (85%), there was a small undercorrection; in 3 of the 20 patients (15%) there was a slight overcorrection.

The results of this study confirm that the described method of analytic model planning in orthognathic surgery is reliable in achieving the planned level of maxillary advancement in bimaxillary procedures.

Conclusions

Orthognathic surgery requires a thorough head and neck examination and precise preoperative dental, radiographic, and aesthetic preparation. Although clinical decision making regarding the preferred spatial reorientation of the jaws to achieve improved function and enhanced facial aesthetics remains both an art and a science, the technical aspects of model planning should be precise and consistent. Current methods of analytic model planning and the use of prefabricated splints represent the standard of care for bimaxillary and segmental maxillary osteotomies.

TABLE 13-1 **Maxillary Incisor Vertical Change, Plane Change, and Incisor Horizontal Change: Planned and Actual**

			Horizontal Change		
Patient	Planned Vertical Change	Planned Plane Change	Planned (mm)	Actual (mm)	Difference (mm)
1	–6	0	+8	+8.5	+0.5
2	–3	–3	+4	+3.2	–0.8
3	–2	0	+6	+5.3	–0.7
4	–3	0	+10	+9.0	–1.0
5	0	0	+10	+9.4	–0.6
6	0	+2	+5	+4.8	–0.2
7	–1	+3	+6	+5.2	–0.8
8	–6	0	+7	+6.2	–0.8
9	+2	+2	+5	+4.8	–0.2
10	0	+3	+8	+7.5	–0.5
11	–3	0	+7	+6.4	–0.6
12	–2	–3	+8	+7.6	–0.4
13	+2	+2	+5	+4.2	–0.8
14	+4	+1	+7	+6.8	–0.2
15	+1	+4	+9	+8.5	–0.5
16	–5	–4	+6	+5.2	–0.8
17	–4	0	+6	+6.5	+0.5
18	–5	–2	+8	+7.2	–0.8
19	+3	0	+8	+8.8	+0.8
20	+	0	+10	+9.1	–0.9

Vertical change: +, maxillary lengthening; –, maxillary shortening. Plane change: +, clockwise rotation; –, counterclockwise rotation of maxillary plane.

TABLE 13-2 **Distribution of Difference Between the Planned and Actual Horizontal Advancement at the Maxillary Incisors**

	Difference (mm)				
	0 to 0.2	+0.2 to 0.4	+0.4 to 0.6	+0.6 to 0.8	+0.8 to 1.0
No. of patients (%)	3 (15%)	1 (5%)	6 (30%)	8 (40%)	2 (10%)

From Park N, Posnick JC: Accuracy of analytic model planning in bimaxillary surgery, *Int J Oral Maxillofac Surg*. In press 2013.

References

1. *2000 Series Articulator,* Whip Mix Corporation, 361 Farmington Ave, PO Box 17183, Louisville, KY 40217.
2. Adrien P, Schouver J: Methods for minimizing the errors in mandibular model mounting on an articulator. *J Oral Rehabil* 24:929, 1997.
3. *Amira* software, Houston, Texas, 2010, TGS. Available at www.tgs.com. Accessed January 4, 2010.
4. Assael LA: Editorial. *J Oral Maxillofac Surg* 67:2041, 2009.
5. Bai S, Shang H, Liu Y, et al: Computer-aided design and computer-aided manufacturing locating guides accompanied with prebent titanium plates in orthognathic surgery. *J Oral Maxillofac Surg* 70:2419–2426, 2012.
6. Becker CM, Kaiser DA, Schwalm C: Mandibular centricity: centric relation. *J Prosthet Dent* 83:158, 2000.
7. Bell W, Proffit W, White R: *Surgical correction of dentofacial deformities*, Philadelphia, 1980, W.B. Saunders Company.
8. Bell RB: Computer planning and intraoperative navigation in orthognathic surgery. *J Oral Maxillofac Surg* 69:592–605, 2011.
9. Bryan DC, Hunt NP: Surgical accuracy in orthognathic surgery. *Br J Oral Maxillofac Surg* 31:343–349, 1993.
10. Campos AA, Nathanson D, Rose L: Reproducibility and condylar position of a physiologic maxillomandibular centric relation in upright and supine body position. *J Prosthet Dent* 76:282, 1996.
11. Centenero SAH, Hernandez-Alfaro F: 3D planning in orthognathic surgery: CAD/CAM surgical splints and prediction of the soft and hard tissues results—our experience in 16 cases. *J Craniomaxillofac Surg* 40:162–168, 2012.
12. Chow TW, Clark RKF, Cooke MS: Errors in mounting maxillary casts using face-bow records as a result of an anatomical variation. *J Dent Res* 13:277–282, 1985.
13. Cope MR: Measuring changes in maxillary height during osteotomy surgery. *Br J Oral Maxillofac Surg* 32:257, 1994.
14. Cordray FE: Centric relation treatment and articulator mountings in orthodontics. *Angle Orthod* 66:153–158, 1996.
15. Ellis E, III: Accuracy of model surgery: evaluation of an old technique and introduction of a new one. *J Oral Maxillofac Surg* 48:1161, 1990.
16. Ellis E, III, Tharanon W, Gambrell K: Accuracy of face–bow transfer: effect on surgical prediction and postsurgical result. *J Oral Maxillofac Surg* 30:362, 1992.
17. Ellis E, III: Bimaxillary surgery using an intermediate splint to position the maxilla. *J Oral Maxillofac Surg* 57:53–56, 1999.
18. Ellis E, III: A comparison of 3 methods of face-bow transfer recording: Implications for orthognathic surgery [discussion]. *J Oral Maxillofac Surg* 56:640–641, 2001.
19. Erickson K, Bell WH, Goldsmith DH: Analytical model surgery. In Bell WH, editor: *Modern practice in orthognathic and reconstructive surgery*, ed 1, Philadelphia, 1992, Saunders, pp 155–205.
20. *Erickson Model Table,* Great Lakes Orthodontics, Ltd, 199 Fire Tower Drive, Tonawanda, NY 14151-15111.
21. Gateno J, Forrest KK, Camp B: A comparison of 3 methods of face-bow transfer recording: Implications for orthognathic surgery. *J Oral Maxillofac Surg* 56:635–640, 2001.
22. Gateno J, Xia J, Teichgraeber J, et al: The precision of computer-generated surgical splint. *J Oral Maxillofac Surg* 61:814, 2003.
23. Gateno J, Teichgraeber JF, Xia JJ: Three-dimensional surgical planning for maxillary and midface distraction osteogenesis. *J Craniofac Surg* 14:833–839, 2003.
24. Gateno J, Xia JJ, Teichgraeber JF, et al: Clinical feasibility of computer-aided surgical simulation (CASS) in the treatment of complex cranio-maxillofacial deformities. *J Oral Maxillofac Surg* 65(4):728–734, 2007.
25. Gateno J, Xia JJ, Teichgraeber JF: New 3-dimensional cephalometric analysis for orthognathic surgery. *J Oral Maxillofac Surg* 69:606–622, 2011.
26. Gateno J, Xia JJ, Teichgraeber JF: Effect of facial asymmetry on 2-dimensional and 3-dimensional cephalometric measurements. *J Oral Maxillofac Surg* 69:655–662, 2011.
27. Gil JN, Claus JDP, Manfro R, Lima SM, Jr: Predictability of maxillary repositioning during bimaxillary surgery: accuracy of a new technique. *Int J Oral Maxillofac Surg* 36:296–300, 2007.
28. Gil JN, Campos FE, Claus JD, et al: Medial canthal region as an external reference point in orthognathic surgery. *J Oral Maxillofac Surg* 69:352–355, 2011.
29. Hartzell DH, Maskeroni AJ, Certosimo FC: Techniques in recording centric relation. *Oper Dent* 25:234–236, 2000.
30. Hatzi P, Millstein P, Maya A: Determining the accuracy of articulator interchangeability and hinge axis reproducibility. *J Prosthet Dent* 85:236–245, 2001.
31. Hellsing G, McWilliam JS: Repeatability of the mandibular retruded position. *J Oral Rehabil* 12:1, 1985.
32. Ingervall B: Recording of retruded positions of mandible in children. A comparison between registrations in general anaesthesia and with children awake. *Odontol Revy* 19(4):413–421, 1968.
33. Ingervall B, Helkimo M, Carlsson GE: Recording of the retruded position of the mandible with application of varying external pressure to the lower jaw in man. *Arch Oral Biol* 16:1165–1170, 1971.
34. Jacobson R, Sarver DM: The predictability of maxillary repositioning in Le Fort I orthognathic surgery. *Am J Orthod Dentofacial Orthop* 122:142, 2002.
35. Jasinevicius TR, Yellowitz JA, Vaughan GG, et al: Centric relation definitions taught in 7 dental schools: results of faculty and student surveys. *J Prosthodont* 9:87–94, 2000.
36. Johnson DG: Intraoperative measurement of maxillary repositioning: an ancillary technique. *Oral Surg Oral Med Oral Pathol* 60:266, 1985.
37. Keshvad A, Winstanley RB: An appraisal of the literature on centric relation: part I. *J Oral Rehabil* 27:823, 2000.
38. Keshvad A, Winstanley RB: An appraisal of the literature on centric relation: part II. *J Oral Rehabil* 27:1013, 2000.
39. Keshvad A, Winstanley RB: An appraisal of the literature on centric relation: part III. *J Oral Rehabil* 28:55, 2001.
40. Kretschmer WB, Zoder W, Baciut G, et al: Accuracy of maxillary positioning in bimaxillary surgery. *Br J Oral Maxillofac Surg* 47:446–449, 2009.
41. Kwon TG, Mori Y, Minami K, Lee SH: Reproducibility of maxillary positioning in Le Fort I osteotomy: a 3-dimensional evaluation. *J Oral Maxillofac Surg* 60:287–293, 2002.
42. Landes C, Sterz M: Proximal segment positioning in bilateral sagittal split osteotomy: intraoperative controlled positioning by a positioning splint. *J Oral Maxillofac Surg* 61:1423, 2003.
43. Landes C: Proximal segment positioning in bilateral sagittal split osteotomy: Intraoperative dynamic positioning and monitoring by sonography. *J Oral Maxillofac Surg* 62:22, 2004.
44. Lapp TH: Bimaxillary surgery without the use of an intermediate splint to position the maxilla. *J Oral Maxillofac Surg* 57:57–60, 1999.
45. Lubbers HT, Jacobsen C, Matthews F, et al: Surgical navigation in craniomaxillofacial surgery: expensive toy or useful tool? A classification of different indications. *J Oral Maxillofac Surg* 69:300–308, 2011.
46. Marko JV: Simple hinge and semi-adjustable articulators in orthognathic surgery. *Am J Orthod Dentofacial Orthop* 90:37–44, 1986.
47. *Maxilim* software, Mechelen, Belgium, 2010, Medicim Medical Image Computing. Available at www.medicim.be. Accessed January 4, 2010.
48. McCormick SU, Drew SJ: Virtual model surgery for efficient planning and surgical performance. *J Oral Maxillofac Surg* 69:638–644, 2011.
49. Moses JJ, Lo HH: The use of asymmetric yaw in the correction of lateral facial defects in hemifacial microsomia deformities: a case report. *Int J Adult Orthodon Orthognath Surg* 7(4):229–234, 1992.
50. Nattestad A, Vedtofte P, Mosekilde E: The significance of an erroneous recording of the

center of mandibular rotation in orthognathic surgery. *J Craniomaxillofac Surg* 19:254, 1991.
51. Nishioka GJ, Van Sickels JE: Modified external reference measurement technique for vertical positioning of the maxilla. *Oral Surg Oral Med Oral Pathol* 64:22, 1987.
52. O'Malley AM, Milosevic A: Comparison of three face bow/semiadjustable articulator systems for planning orthognathic surgery. *Br J Oral Maxillofac Surg* 38:185–190, 2000.
53. Ong TK, Banks RJ, Hildreth AJ: Surgical accuracy in Le Fort I maxillary osteotomies. *Br J Oral Maxillofac Surg* 39:96–102, 2001.
54. Park N, Posnick JC: Accuracy of analytic model planning in bimaxillary surgery. *Int J Oral Maxillofac Surg* 42:807–814, 2013.
55. Perez D, Ellis E, III: Sequencing bimaxillary surgery: mandible first. *J Oral Maxillofac Surg* 69:2217–2224, 2011.
56. Polido WD, Ellis E, III, Sinn DP: An assessment of the predictability of the maxillary surgery. *J Oral Maxillofac Surg* 48:697–701, 1990.
57. Polido WD, Ellis E, III, Sinn DP: An assessment of the predictability of the maxillary repositioning. *Int J Oral Maxillofac Surg* 20:349–352, 1991.
58. Politi M, Toro C, Costa F, et al: Intraoperative awakening of the patient during orthognathic surgery: a method to prevent the condylar sag. *J Oral Maxillofac Surg* 65:109, 2007.
59. Posnick JC, Fantuzzo JJ, Orchin J: Deliberate operative rotation of the maxillo-mandibular complex to alter the A-point to B-point relationship for enhanced facial esthetics. *J Oral Maxillofac Surg* 64:1687–1695, 2006.
60. Posnick JC, Nakano P, Taylor M: A modified occlusal splint to avoid tracheotomy for total midface osteotomies. *Ann Plast Surg* 29(3):223–230, 1992.
61. Quevedo LA, Ruiz JV, Quevedo CA: Using a clinical protocol for orthognathic surgery and assessing a 3-dimensional virtual approach: current therapy. *J Oral Maxillofac Surg* 69:623–637, 2011.
62. *Quick Mount Face-Bow,* Whip Mix Corporation, 361 Farmington Ave, PO Box 17183, Louisville, KY 40217.
63. Rinchuse DJ, Kandasamy S: Articulators in orthodontics: an evidence-based perspective. *Am J Orthod Dentofacial Orthop* 129:299–308, 2006.
64. Rinchuse DJ, Kandasamy S: Centric relation: a historical and contemporary orthodontic perspective. *J Am Dent Assoc* 137:494–501, 2006.
65. Roth RH: Gnathological considerations for orthodontic therapy. In McNeill C, editor: *Science and practice of occlusion*, Chicago, 1997, Quintessence, pp 502–514.
66. Scarbrough FE, Ghali GE, Smith BR: Anatomic guidelines for the placement of external references for maxillary repositioning. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 84:465, 1997.
67. Schendel S, Jacobsen R: Three-dimensional imaging and computer simulation for office-based surgery. *J Oral Maxillofac Surg* 67:2107, 2009.
68. Simon RL, Nicholls JI: Variability of passively recorded centric relation. *J Prosthet Dent* 44:21, 1980.
69. Stanchina R, Ellis E, III, Gallo WJ, Fonseca RJ: A comparison of two measures for repositioning the maxilla during orthognathic surgery. *Int J Adult Orthodon Orthognath Surg* 3:149–154, 1998.
70. Swennen RJ, Mollemans W, Schutyser F: Three-dimensional treatment planning of orthognathic surgery in the era of virtual imaging. *J Oral Maxillofac Surg* 67:2080, 2009.
71. Tarantola GJ, Becker IM, Gremillion H: The reproducibility of centric relation: a clinical approach. *J Am Dent Assoc* 128:1245, 1997.
72. The glossary of prosthodontic terms. *J Prosthet Dent* 58:713, 1987.
73. The glossary of prosthodontic terms. *J Prosthet Dent* 71:40, 1994.
74. The glossary of prosthodontic terms. *J Prosthet Dent* 81:39, 1999.
75. The glossary of prosthodontic terms. *J Prosthet Dent* 94:10, 2005.
76. Thompson JR: The rest position of the mandible and its application to analysis and correction of malocclusion. *Angle Orthod* 19:162–187, 1949.
77. Troulis MJ, Everett P, Seldin EB, et al: Development of a three-dimensional treatment planning system based on computed tomographic data. *Int J Oral Maxillofac Surg* 31(4):349–357, 2002.
78. Turvey T: Sequencing of two-jaw surgery: the case for operating on the maxilla first. *J Oral Maxillofac Surg* 69:2225, 2011.
79. Truitt J, Strauss RA, Best A: Centric relation: a survey study to determine whether a consensus exists between oral and maxillofacial surgeons and orthodontists. *J Oral Maxillofac Surg* 67:1058–1061, 2009.
80. Uechi J, Okayama M, Shibata T, et al: A novel method for the 3-dimensional simulation of orthognathic surgery by using a multimodal image-fusion technique. *Am J Orthod Dentofacial Orthop* 130:786–798, 2006.
81. Van Sickels JK, Larsen AJ, Triplett RG: Predictability of maxillary surgery: a comparison of internal and external reference marks. *Oral Surg* 61:542–545, 1986.
82. Wood DP, Elliot RW: Reproducibility of the centric relation bite registration technique. *Angle Orthod* 64:211–220, 1994.
83. Xia J, Gateno J, Teichgraeber J: New clinical protocol to evaluate craniomaxillofacial deformity and plan surgical correction. *J Oral Maxillofac Surg* 67:2093, 2009.
84. Xia JJ, McGrory JK, Gateno J, et al: A new method to orient 3-dimensional computed tomography models to the natural head position: a clinical feasibility study. *J Oral Maxillofac Surg* 69:584–591, 2011.
85. Xia JJ, Shevchenko L, Gateno J, et al: Outcome study of computer-aided surgical simulation in the treatment of patients with craniomaxillofacial deformities. *J Oral Maxillofac Surg* 69:2014–2024, 2011.
86. Zizelmann C, Hammer B, Gellrich NC, et al: An evaluation of face bow transfer for the planning of orthognathic surgery. *J Oral Maxillofac Surg* 70:1944–1950, 2012.

14

Mandible-First Analytic Model Planning for Orthognathic Surgery

JEFFREY C. POSNICK, DMD, MD

- Controversies Surrounding Centric Relation
- Mandible-First Analytic Model Planning Technique
- Technical Aspects of Executing the Mandible-First Approach During Orthognathic Surgery
- Mandible-First Analytic Model Planning: Step-by-Step Approach
- Case Presentations
- Conclusions

Unlike most surgical procedures, orthognathic surgery involves not just thorough medical assessment but also precise preoperative dental, radiographic, and facial aesthetic planning. The surgeon also carries out a detailed face-to-face examination of the patient to determine variations from normal.

Immediate presurgical records include current dental impressions of the maxillary and mandibular arches; accurate bite registration in centric relation (CR); a face-bow registration; and the recording of specific facial measurements, including deviations from normal. The dental models are placed on a semi-adjustable articulator for further analysis, and they are then used to complete analytic model planning.[1,2,10,13,15,39,40] Acrylic splints are fabricated and used intraoperatively to further ensure the reliable execution of the predetermined reorientation of the maxilla and mandible to achieve facial aesthetic and functional objectives.

Although the making of clinical decisions about the preferred aesthetic repositioning of the jaws remains both an art and a science (see Chapter 12), the technical aspects of model planning should be precise and consistent. Analytic model planning and the use of prefabricated splints continue to represent the standard of care for bimaxillary and segmental maxillary osteotomies.[6,14,17,52]

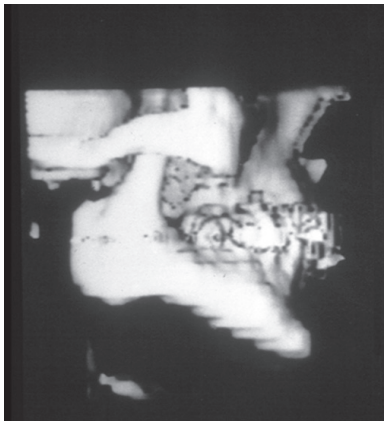
Despite the benefits of this time-honored model planning approach, there is the potential for the introduction of error. For example, if an inaccurate CR bite registration is obtained from the patient preoperatively. The surgeon's ability to obtain an accurate and reproducible bite registration in CR during the immediate presurgical clinical examination, during the occlusion of the dental models on the articulator in the laboratory, and when using the splints in the operating room is essential to avoid errors when *standard analytic model planning* is employed.[5,12,13,17-28,32,33,35,36,53]

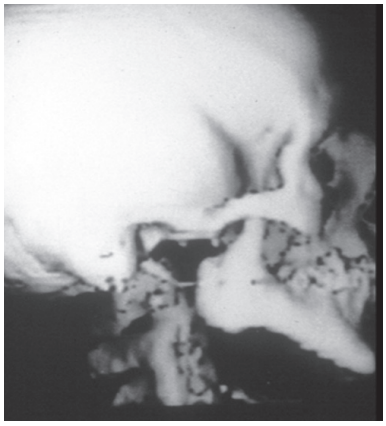
There are specific clinical settings in which the obtaining of an accurate and reliable CR can be problematic.[8,11,34,37,39,52] Examples include the following individuals: 1) those who are missing a condyle after tumor resection 2) those with severe atrophy of the condyle after trauma and 3) those with specific syndromes in which the condyle-ascending ramus is aplastic (e.g., Treacher Collins syndrome, or hemifacial microsomia with Kaban type IIB or III malformation; see Chapters 27 and 28) (Fig. 14-1) For those patients with an unreliable CR, a *"mandible-first" approach to analytic model planning* is required.

Controversies Surrounding Centric Relation

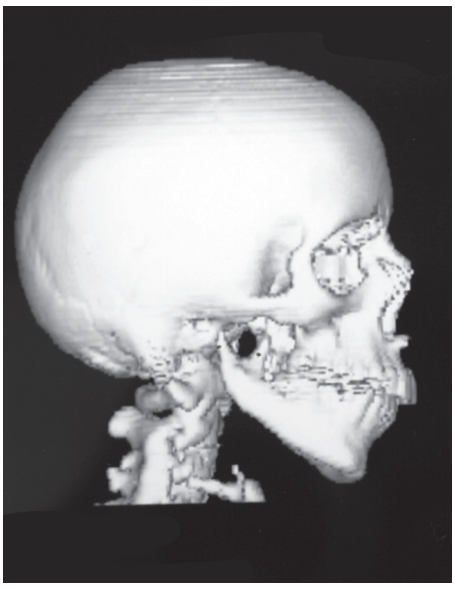
Definition of Centric Relation

The definition of CR remains somewhat controversial, and it is frequently misstated.[46-49] In 1987, the definition of CR underwent a change in terminology from being considered a mandibular position in which the condyles are seated in the most "posterior superior" position to one in which the condyles are placed in the most "superior anterior" position in the glenoid fossa.[45,46] For analytic model planning in





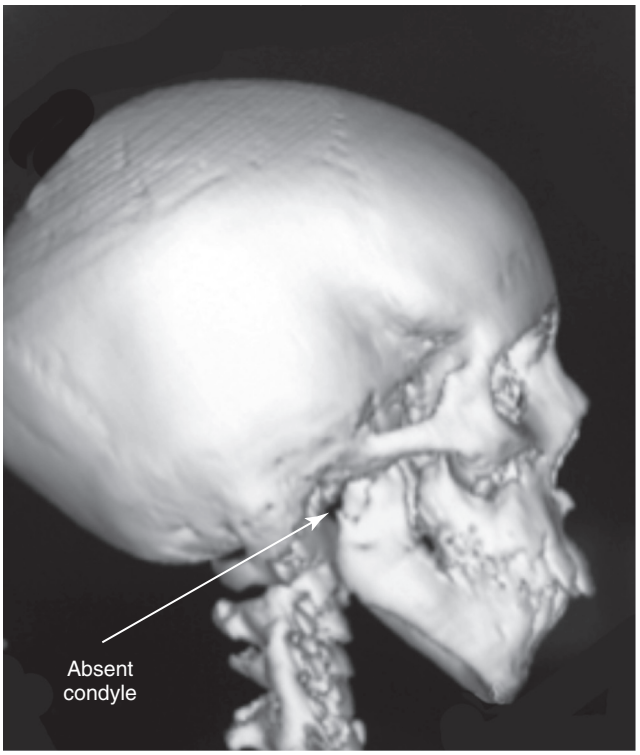
Kaban type II B



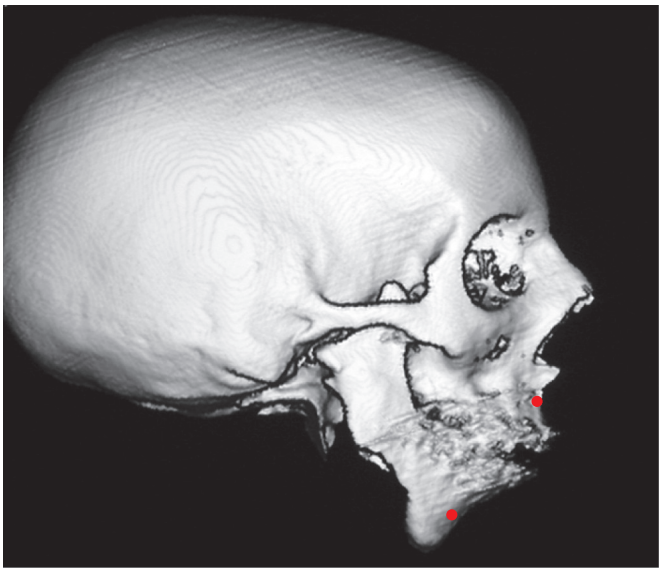
Kaban type II B

• **Figure 14-1** There are specific clinical settings in which obtaining an accurate and reliable centric relation (CR) can be problematic. Examples include individuals with atrophy of a condylar head (e.g., after a displaced condyle fracture) or those with specific syndromes in which aplasia of the glenoid fossa and condyle of the ascending ramus is extensive (e.g., Treacher Collins syndrome, hemifacial microsomia [HFM] with Kaban type IIB or III mandibular malformations). In addition, there are unique circumstances in which patients undergo bimaxillary procedures that require posterior maxillary lengthening to the extent that clockwise rotation of the mandible would result in a translational movement of the condyles in the glenoid fossa. In these cases, a mandible-first approach to analytic model planning is indicated. Several clinical examples are shown. **A,** A 16-year-old boy who had sustained a fracture of the right condyle of the mandible earlier during his childhood presented as a teenager with facial asymmetry. Complete atrophy of the right condyle had occurred, with a loss of posterior facial height. For this reason, a stable CR bite registration was not possible. Facial and computed tomography (CT) scan views are shown to confirm these facts. **B,** A 14-year-old girl with right-sided HFM and a Kaban type IIB condyle and ascending ramus malformation is shown. Because of these conditions, obtaining a consistent CR bite registration was not possible. Facial and CT scan views confirm these findings.

Continued



Kaban type II B



Kaban type II A

• **Figure 14-1, cont'd C,** An 11 year-old-boy who was born with right-sided HFM and a Kaban type IIB condyle and ascending ramus malformation is shown. Because of these conditions, obtaining a consistent CR bite registration was not possible. Facial and CT scan views confirm these findings. **D,** A teenage girl with a bilateral form of HFM and a Kaban type IIA condyle and ascending ramus malformation is shown. Although a stable CR bite registration could be obtained, the correction of the bimaxillary deformity requires significant counterclockwise rotation of the maxilla, including the lengthening of the posterior facial height of the maxilla. The use of standard maxilla-first analytic model planning would require clockwise rotation of the mandible to place the intermediate splint to the extent that translational movement of the condyles in the glenoid fossa would occur. For this reason, a mandible-first approach is required. Facial and CT scan views confirm these findings.

preparation for orthognathic surgery, defining the exact position of each condylar head during mandibular protrusion and lateral excursions is not critical. However, it is essential to locate a mandibular position that is reproducible with the teeth in occlusion. This must be obtained without the assistance of the patient (i.e., with fully relaxed masticatory muscles), and it needs to be reliably accomplished both preoperatively and intraoperatively. This position must also be transferable to the articulator in the laboratory for model planning to be accomplished accurately. An erroneous and inconsistent recording of CR will lead to the malpositioning of the maxilla or mandible during surgery.

A survey study conducted by Truitt and colleagues confirmed that 98% of oral and maxillofacial surgeons define CR as the most posterior superior seating of the condyle in the fossa.[51] Interestingly, a majority of the orthodontists surveyed agree with the surgeons' definition of CR, but there was less consistency.[22,41] According to the survey, surgeons and orthodontists do agree that, in preparation for orthognathic surgery, analytic model planning requires that casts of the teeth be mounted in CR on a semi-adjustable articulator.

Capturing Centric Relation

Hellsing and McWilliam used a subtraction technique to assess the repeatability of CR that is recorded with a *one-handed push-back technique*.[21] They found that, with the use of this technique, the mandibular condyles can be seated in a reproducible position and thus be well suited for the registration of CR. Campos and colleagues compared the *swallowing technique* with the *chin-point guidance technique* when the patient was in both upright and supine body positions.[3,5,9,15,21] Both techniques were found to establish physiologic CR in a reproducible manner. Interestingly, when the chin-point guidance method was used, the work of these authors suggested that reliability was greater with the patient in the upright, seated position as compared with the supine position.

Simon and Nicholls compared the *chin-point guidance, chin-point guidance with ramus support,* and *bimanual manipulation* techniques.[43] They found that there were no significant differences in the range of mandibular positions that were observed. Interestingly, their study patients did not include individuals with jaw deformities. It is known that patients with specific patterns of jaw deformity (e.g., Class II retrognathism) are more likely to involuntarily posture the mandible forward during the swallowing technique.

When a reliable CR can be obtained, we find the *chin-point guidance technique*—when used both preoperatively and intraoperatively by the same clinician—to be both reliable and convenient for the orthognathic surgery patient. This is the method that has been adopted by this author, and it has been used successfully for orthognathic patients that have a reliable CR. By using one hand to apply pressure to the subject's chin and the other hand to apply opposing pressure to the occiput, the clinician is able to apply posterior and superior "vectored" force to the anterior mandible. This will seat the condyles in a superior-anterior location within each glenoid fossa (i.e., CR).

Unstable (Unreliable) Centric Relation

Alternative methods to the standard analytic model planning approach to bimaxillary orthognathic surgery have been described in an attempt to improve the accuracy and predictability of outcomes.[8,11,34,37,50,52] Recently, surgeons have also begun looking to computer-aided design and computer-aided modeling software to assist with the planning and implementation of orthognathic procedures (see Chapter 13).[3,4,7,14,16,18,19,20,29,30,31,38,42,44,54,55,56]

The subset of patients in which obtaining an accurate CR is not feasible are well suited for a *"mandible-first" approach*. Common clinical examples of these patients include the following: 1) those with specific congenital anomalies (e.g., hemifacial microsomia with Kaban Type IIB or III malformations) (Fig. 14-2 and 14-3) 2) those with specific types of trauma (e.g., after condylar fracture with resorption or after ankylosis release with condylar resection) 3) those with specific pathology (e.g., after condylar resection for tumor) and 4) those with the inability to cooperate in the office setting (e.g., those with neuropsychiatric disorders that involve uncontrolled masticatory muscle spasticity).

A *mandible-first approach to analytic model planning* is also indicated in unique clinical circumstances in which significant posterior maxillary lengthening is planned to the extent that the clockwise rotation of the mandible will require the translational movement of the condyles in the glenoid fossa during the use of the intermediate splint (Fig. 14-4). Fortunately, these cases are not common; very few patients require the actual lengthening of the posterior maxillary height. Furthermore, those who do generally either require the closure of baseline posterior open bites (i.e., cases that involve the failure of the posterior dentition to erupt) or are undergoing simultaneous advancement. For example, the advantage of the counterclockwise rotation of the maxillary and mandibular planes (i.e., pitch orientation) is frequently seen in the clinical setting of a long face growth pattern. This counter clockwise rotation is generally accomplished via the differential intrusion of the maxilla (i.e., anterior more so than posterior). This does not require the lengthening of the posterior maxillary height or the overall posterior facial height (see Chapter 21).

Mandible-First Analytic Model Planning Technique

We prefer to use the *mandible-first analytic model planning technique,* in only specific clinical situations (see Fig. 14-1 through 14-4).[8,11,34,37,39,52] However, whichever method of model planning is used, all orthognathic patients require a thorough clinical history and physical examination. A

maxillofacial and focused dental assessment is also completed with particular emphasis on the accurate documentation of the following: 1) any maxillary occlusal cant (i.e., role orientation) 2) the maxillary and mandibular dental midline positions relative to the facial midline (i.e., yaw orientation) 3) any disproportions in vertical facial height 4) any disproportions in the horizontal projection of each jaw and 5) any advantage to the alteration of the maxillary or mandibular planes (i.e., pitch orientation); (see Chapter 12).

The basic technical aspects of *mandible-first analytic model planning* include the following:

1. To register the bite in a patient with a unilateral "free-floating" condyle–glenoid fossa relationship (e.g., Kaban type IIB or III hemifacial microsomia, the absence of the condyle after tumor resection or trauma), the mandible is manipulated with the "normal" condyle placed reliably in the terminal hinge position and with the "abnormal" (i.e., contralateral) condyle seated in its most posterior-superior position. This will demonstrate the maximum mandibular deformity. A wax bite registration is taken in this position.
2. A face-bow registration is obtained. Care is taken to accurately reproduce the patient's existing maxillary plane (pitch orientation), cant (role orientation), and midline (yaw orientation) in relationship to the upper face. If the cranial base and the external auditory canals have essentially normal morphology, this is accomplished in a traditional fashion with the use of the standard face-bow technique, which involves placing an earpiece in each ear canal. If the patient's ear canals are abnormally formed or positioned (e.g., in a patient with hemifacial microsomia with external auditory canal atresia), adjustments are made by using references to the parallelism of the floor and the morphology of the orbits. When doing so, the earpieces maybe placed against the patient's cheek skin (rather than in the dysmorphic or absent external auditory canal). The patient's head and neck are positioned by the operator in the natural head position for the face-bow registration (see Chapters 12 and 13).
3. Two maxillary and one mandibular alginate impressions are taken. The three dental impressions are poured in green die stone. The first maxillary cast is mounted onto the articulator in the standard fashion with white plaster and the use of the face-bow registration for orientation. The wax bite registration is then used to articulate the maxillary and mandibular casts to each other. The mandibular cast is then mounted on the articulator in the standard fashion, also with the use of white plaster (see Chapter 13).
4. The first maxillary mounting is removed, and the second maxillary cast is mounted. This is accomplished with the use of the original bite registration against the already mounted mandibular cast. Vertical and horizontal reference lines are created on each maxillary and mandibular unit (see Chapter 13). The Erickson Model Table is used to make baseline millimeter reference measurements on both maxillary units and the mandibular unit (see Chapter 13).
5. The original maxillary cast is then separated from its plaster base and three-dimensionally repositioned and reoriented according to the treatment plan. The Erickson Model Table is used to confirm the correct reorientation. If segmentation is required, it is completed before the reorientation of the maxilla to the plaster base (see Chapter 13). The reorientation of the maxilla takes into account the preferred horizontal, vertical, transverse (segmental) midline (yaw), cant (roll), and plane (pitch), needs (see Chapter 12). The maxillary cast is then secured to the plaster base with hot glue in the planned treatment position on the articulator (see Chapter 13).
6. The mandibular cast is then separated from its base and repositioned into the preferred final occlusion to the reoriented maxillary unit with the use of hot glue. The mandibular cast is remounted to its plaster base in the new position on the articulator with hot glue. The occlusion is released (i.e., the glue that secured the occlusion is removed), and a "maxillary-positioning" splint is fabricated with acrylic (see Chapter 13).
7. Next, the repositioned (reoriented) maxillary unit is removed from the articulator and replaced with the "uncut" second maxillary unit. This indicates the new mandibular location and the "unoperated" maxillary position. The "mandibular-positioning" splint is then fabricated with acrylic.

Technical Aspects of Executing the Mandible-First Approach During Orthognathic Surgery

Consider as an example an individual with hemifacial microsomia and a Kaban type IIB mandibular malformation ((Fig. 14-2 and 14-3). When using the mandible-first approach, the "normal" side of the ramus of the mandible is split sagittally in the usual way (i.e. sagittal ramus osteotomy of mandible). The distal mandible is then placed into its treatment-planned final position with the use of the prefabricated "mandibular-positioning" splint, and intermaxillary fixation (IMF) is established. To fully mobilize the distal mandible and secure it into the new occlusion, it may be necessary to 1) complete an ipsilateral coronoidectomy, 2) excise portions of the rudimentary (ipsilateral) mandible, or 3) both.

After the distal mandible is in the splint and secured with IMF, the proximal segment on the "normal" side is seated into the terminal hinge position. Rigid internal fixation (e.g., bicortical screws) is then established across the ramus osteotomy site (see Chapter 15).

The deficient portion of the ipsilateral mandible is constructed with the use of a bone graft (e.g., a costochondral graft). The neocondyle is seated in the glenoid fossa. Once in place, fixation of the graft is achieved to the distal

mandible with an extended titanium plate and screws. The IMF is released, and the occlusion is confirmed to be accurate and reproducible into the splint.

The "mandibular-positioning" splint is removed, and the maxillary (Le Fort I) osteotomy is performed. After downfracture and disimpaction (including segmentation, if indicated), the "maxillary-positioning" splint is used to achieve the treatment-planned maxillary reorientation. Plate and screw fixation is secured across the maxillary osteotomy sites in the usual manor (see Chapter 15). The IMF is released, and an accurate and reproducible occlusion is confirmed. The splint is either removed or left in place, depending on the patient's clinical healing needs.

Mandible-First Analytic Model Planning: Step-by-Step Approach

1. Obtain a wax bite registration with the normal (unaffected) condyle seated in the terminal hinge position and with the abnormal (hypoplastic) condyle positioned as posterior and superiorly against the skull base as possible.
2. Use the face-bow device to register the relationships of the maxilla, glenoid fossa, and the skull base while the patient is in the natural head position (NHP). Use a modified technique if ear canals and the orbital symmetry are abnormal.
3. Take alginate impressions (two maxillary, one mandibular).
4. Pour alginate impressions in green die stone with a 25- to 30-mm base.
5. Mount the first maxillary cast onto the articulator base plate with the use of white plaster and the face-bow recording.
6. Turn the articulator upside down and place the mandibular cast onto the already mounted maxillary unit with the use of the wax bite registration. Mount the mandible cast onto the articulator base plate with white plaster.
7. Remove the mounted maxillary unit, and mount the second maxillary cast onto the articulator base plate with white plaster and the use of the same CR bite registration.
8. Mark vertical and horizontal reference lines on all three units, and take baseline measurements on the first maxillary unit with the use of the Erickson Model Table.
9. Perform model surgery on the first maxillary cast (including segmentation, if indicated). Place the maxilla in its treatment-planned location. Use the Erickson Model Table to confirm correct positioning. Secure the maxillary cast to the articulator (in its new location) with hot glue.
10. Separate the mandibular cast from the base. Secure the mandibular cast with hot glue into the optimal final occlusion against the repositioned maxillary cast. Secure the mandibular cast with hot glue or plaster back onto the base plate of the articulator.
11. Release the occlusion (i.e., remove the glue), and fabricate the "maxillary-positioning" splint in the standard fashion with the use of acrylic.
12. Remove the first maxillary (cut) unit, which is currently on the articulator, and place the second (uncut) maxillary unit.
13. Fabricate the "mandibular-positioning" splint in the standard fashion with the use of acrylic. This will be with the final (cut) mandibular unit and initial (uncut) maxillary unit.

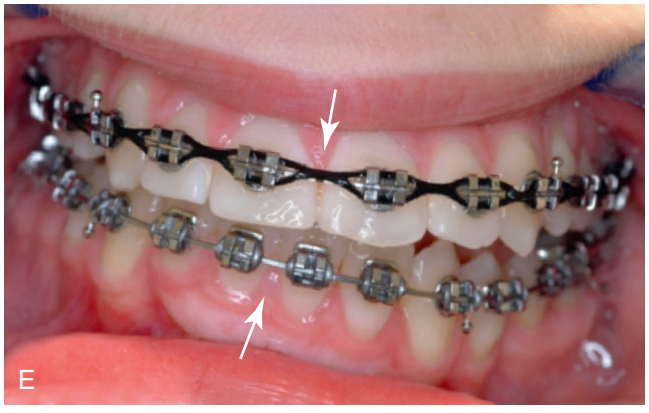
Case Presentations

• CASE 14-1

Hemifacial Microsomia

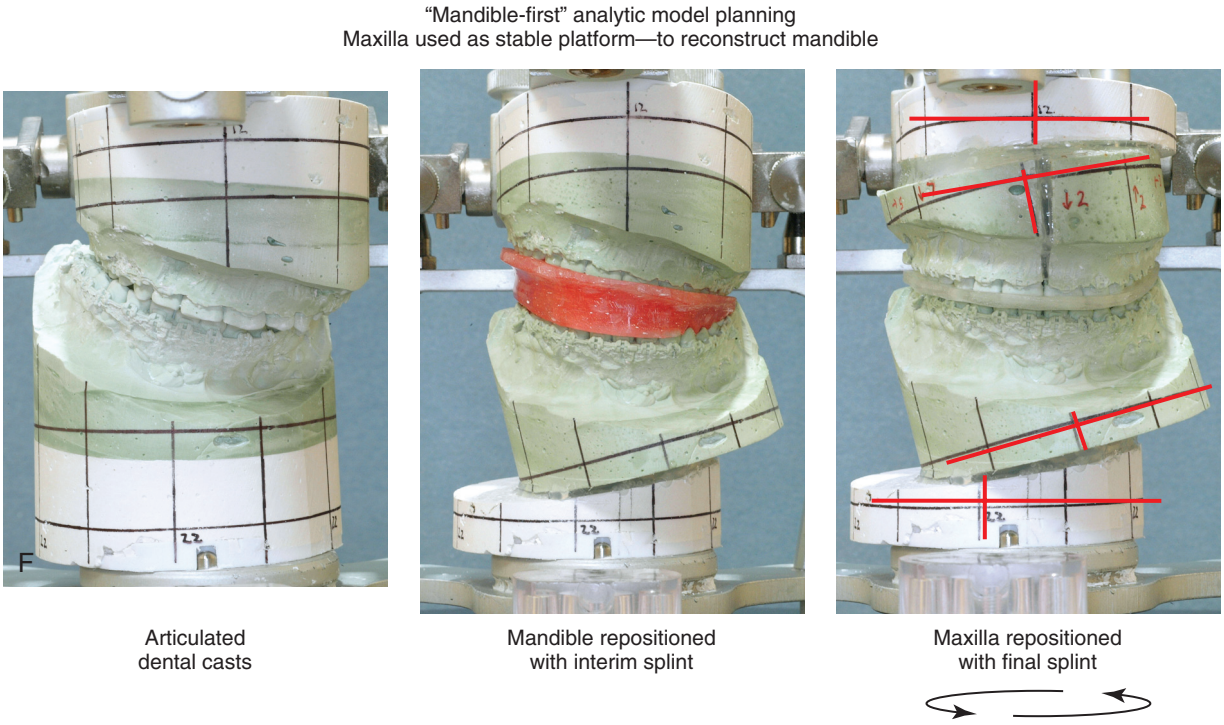
A child was born with right-sided hemifacial microsomia that was manifested by mild hypoplasia of the affected soft tissues, paresis of the temporal branch of the facial nerve, a degree of zygoma and orbital hypoplasia, and a Kaban type IIB mandibular malformation (Fig. 14-2). The right external ear was displaced but had acceptable formation. She underwent orthodontic alignment during early adolescence, and she was referred to this surgeon when she was 14 years old. At age 16, after the completion of her orthodontic preparation, she underwent a maxillary Le Fort I osteotomy with three-dimensional repositioning (cant correction, occlusal plane adjustment, midline correction, and vertical and horizontal adjustments); sagittal split osteotomy of the left ramus; reconstruction of the right condyle and ascending ramus with an autogenous costochondral graft; osseous genioplasty (horizontal advancement and asymmetry correction); and intranasal procedures (septoplasty, and inferior turbinate reduction).

• **Figure 14-2** A 14-year-old girl who was born with right-sided hemifacial microsomia and a Kaban type IIB condyle and ascending ramus malformation was followed longitudinally without treatment intervention until skeletal maturity. Facial asymmetry demonstrated no progression during this time frame. During the adult dentition and with orthodontic dental decompensation complete, she underwent single-stage reconstruction that included a maxillary Le Fort I osteotomy a left-sided sagittal split ramus osteotomy; right mandible reconstruction with costochondral graft septoplasty and inferior turbinate reduction. Improvements in the inferior border of mandible symmetry after surgery have been documented. However, significant soft-tissue asymmetry in the pre-auricular cheek region remains. Fat grafting is an option to consider. **A,** Frontal views in repose before and after reconstruction. **B,** Frontal views with smile before and after reconstruction.



• **Figure 14-2, cont'd** **C,** Right oblique facial views before and after reconstruction. **D,** Left oblique facial views before and after reconstruction. **E,** Occlusal views before and after reconstruction.

Continued



• **Figure 14-2, cont'd F,** Articulated dental casts that indicate "mandible-first" analytic model planning. **G,** Lateral cephalometric radiographs before and after reconstruction.

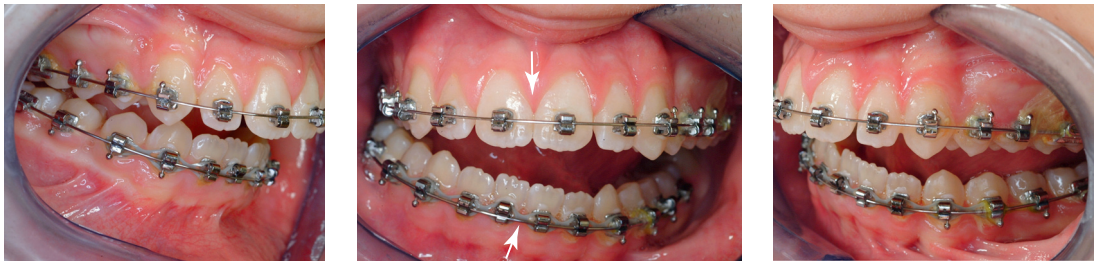
• CASE 14-2

Hemifacial Microsomia

A child was born with right-sided hemifacial microsomia, which was manifested by moderate hypoplasia of the right-sided soft tissues, displacement and deformity of the external ear, anomalous external auditory canals, and a Kaban type IIB mandibular malformation (Fig. 14-3). Left-sided macrosomia repair was carried out during childhood. During the late mixed dentition, the patient was referred to this surgeon for evaluation (see Fig. 28-15). After orthodontic preparation, he underwent a maxillary Le Fort I osteotomy in two segments with three-dimensional repositioning (horizontal advancement, vertical shortening, transverse expansion, dental midline shift, occlusal plane change, and cant correction); sagittal split osteotomy of the left ramus; reconstruction of the right condyle and ascending ramus of the mandible with an autogenous costochondral graft; osseous genioplasty (advancement and asymmetry correction); an intranasal procedure (septoplasty and inferior turbinate reduction); and bilateral otoplasty (set-back).

• **Figure 14-3** A child who was born with hemifacial microsomia of the right side of face and a type IIB condyle and ascending ramus malformation was followed longitudinally without treatment intervention until skeletal maturity (see Fig. 28-15). Facial asymmetry demonstrated no progression during this time frame. During the adult dentition and with orthodontic dental decompensation complete, the patient underwent single-stage reconstruction that included a maxillary Le Fort I osteotomy a left-sided sagittal split ramus osteotomy; right mandibular reconstruction with costochondral graft osseous genioplasty septoplasty, inferior turbinate reduction, and bilateral otoplasty (ear set-back). **A,** Facial views in repose before and after reconstruction. **B,** Facial views with smile before and after reconstruction.

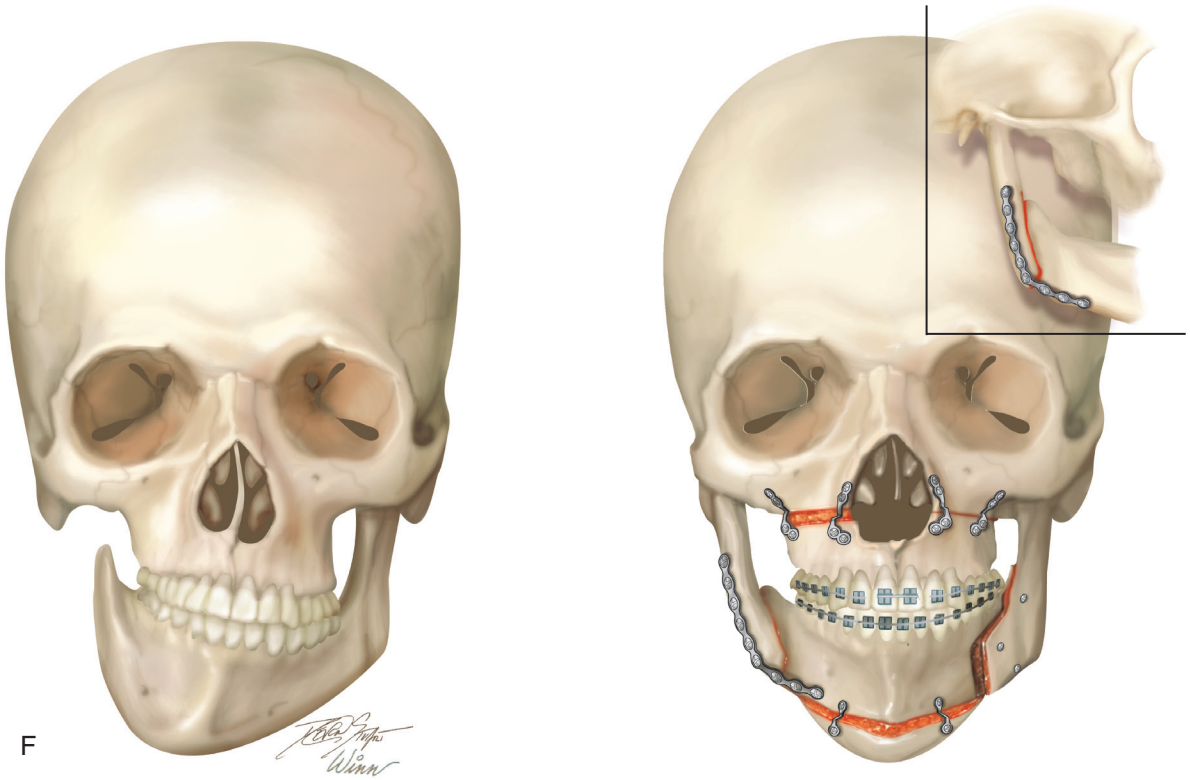
Continued



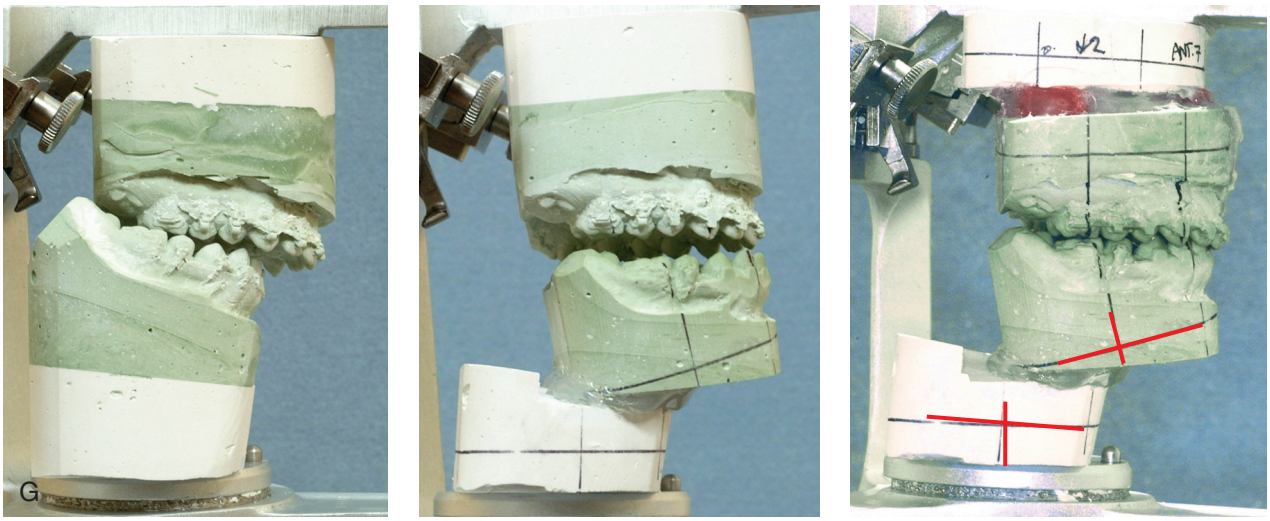
Pre surgery

After treatment

• **Figure 14-3, cont'd C,** Left facial oblique views before and after reconstruction. **D,** Right oblique facial views before and after reconstruction. **E,** Occlusal views before surgery and after the completion of treatment.



"Mandible-first" analytic model planning.
The maxilla is used as stable platform—to reconstruct mandible



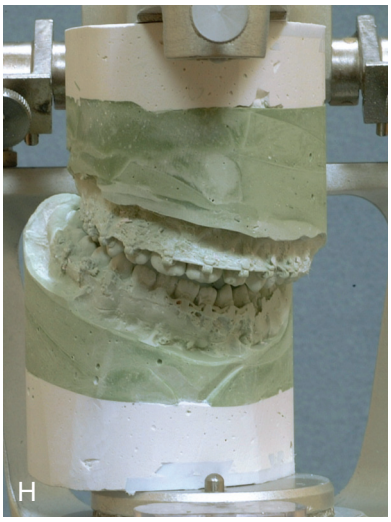
Articulated dental casts

Mandible repositioned

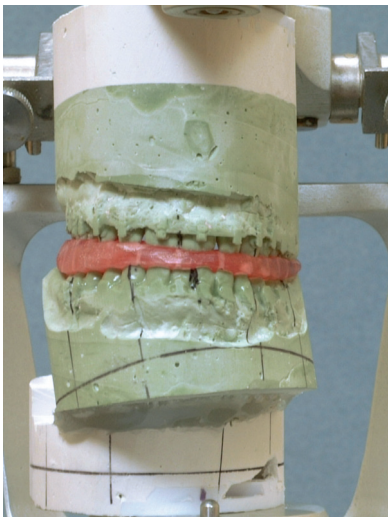
Maxilla repositioned

• **Figure 14-3, cont'd** **F,** Illustrations of the presenting anatomy and the reconstructive procedures that were carried out. **G** & **H,** Articulated dental casts that indicate "mandible-first" analytic model planning. *Part F modified by an original illustration by Bill Winn.* *Continued*

"Mandible-first" analytic model planning
Maxilla used as stable platform—to reconstruct mandible

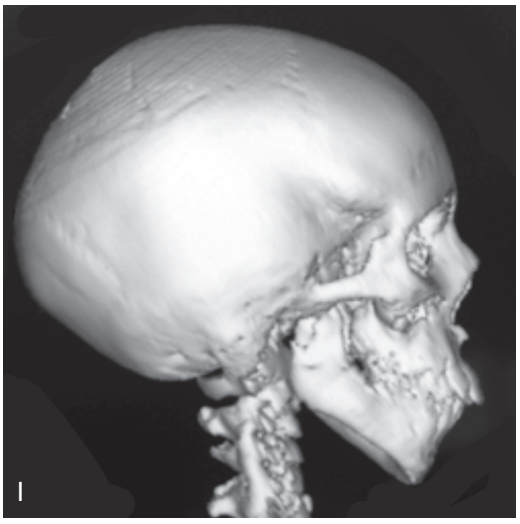


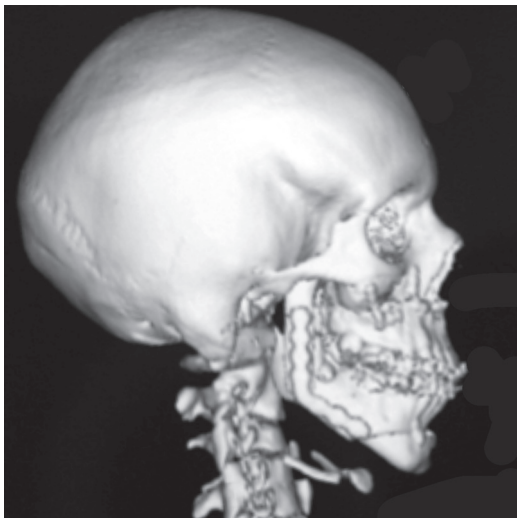
Articulated dental casts



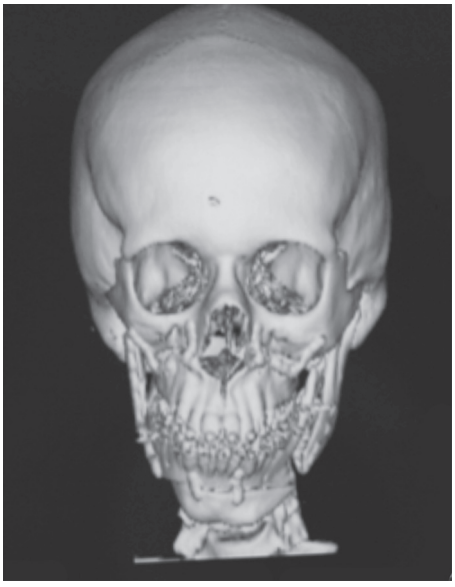
Mandible repositioned

Maxilla repositioned





©Elsevier Inc.
Orthognathic Surgery: J.C. Posnick



• **Figure 14-3, cont'd I,** Lateral computed tomography scan views before and after reconstruction. **J,** Frontal facial and computed tomography scan views after reconstruction.

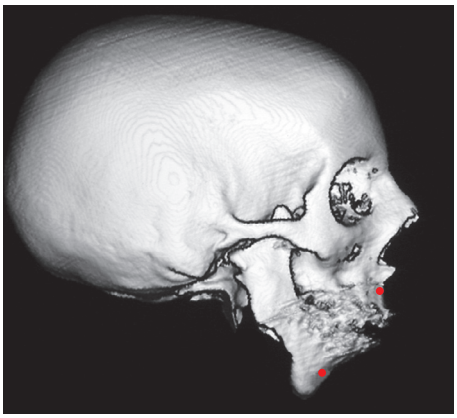
• CASE 14-3

Hemifacial Microsomia

A child was born with an asymmetric bilateral form of hemifacial microsomia that was manifested by moderate hypoplasia of the affected soft tissues (including the muscles of mastication), bilateral microtia, atresia of the external auditory canals, and mild bilateral hypoplasia of the zygomas and the orbits. Bilateral asymmetric Kaban type IIA mandible malformations were present (Fig. 14-4). The patient's previous surgeries included multiple external ear reconstructive procedures by another surgeon, including the placement of autogenous costochondral grafts. She also underwent an attempt to open the left external auditory canal by another surgeon, but this was aborted after facial nerve concerns. Mandibular reconstruction was attempted twice (when the patient was 7 and 11 years old) by another surgeon with the use of distraction osteogenesis techniques, but this had limited results. The previous procedures created intraoral and facial scarring, and a 15-mm anterior open-bite malocclusion remained.

At 14 years old, the patient was referred to this surgeon for evaluation (see Fig. 28-16). After additional orthodontic preparation, she underwent maxillary Le Fort I osteotomy; bilateral coronoidectomies; bilateral sagittal ramus osteotomies (with autogenous iliac corticocancellous interpositional bone grafts); osseous genioplasty (advancement and shortening); and intranasal procedures (septoplasty and inferior turbinate reduction). The described mandible-first analytic model planning technique was used.

Unfavorable A-pt. to B-pt. relationship



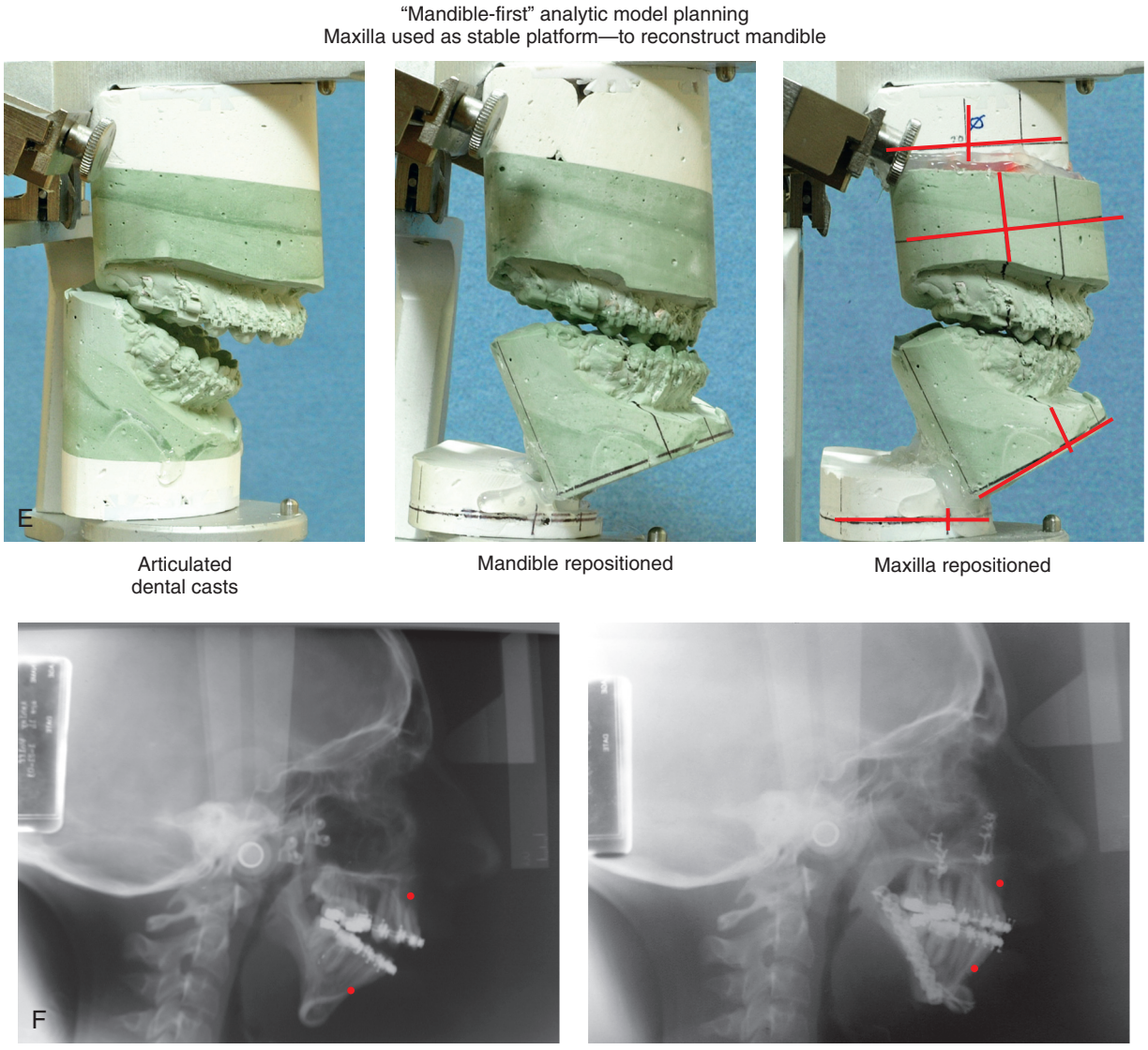
Distraction osteogenesis effects on mandibular ramus/angle

• **Figure 14-4** A girl was born with a bilateral form of hemifacial microsomia and Kaban type IIA condyle and ascending ramus malformations (see Fig. 28-16). When she was 7 and 11 years old (and at another institution), she underwent bilateral distraction osteogenisis of the mandible. The procedures were unsuccessful. She also underwent bilateral external ear reconstruction with a suboptimal result. The patient wore external hearing aids. Earlier orthodontic treatment was carried out (at another institution) including four bicuspid extractions. After presenting to this surgeon when she was 15 years old, the patient underwent reconstruction that included a Le Fort I osteotomy bilateral ramus osteotomies with interpositional grafting; osseous genioplasty; septoplasty, inferior turbinate reduction, and nasal floor recontouring. **A,** Profile facial and computed tomography scan views at the time of referral to this surgeon. **B,** Frontal views with smile before and after reconstruction. *Continued*

Pre surgery with 4 bicuspid extractions

After treatment

• **Figure 14-4, cont'd C,** Oblique facial views before and after reconstruction. **D,** Occlusal views after orthodontic decompensation (i.e., four bicuspid extractions) and then 1 year after the completion of treatment.



• **Figure 14-4, cont'd** **E,** Articulated dental casts that indicate "mandible-first" analytic model planning. **F,** Lateral cephalometric radiographs before and after reconstruction.

Conclusions

During model planning in preparation for orthognathic surgery, the introduction of error can occur when obtaining the bite registration in the clinical setting. There are specific clinical settings in which the obtaining of an accurate and reliable bite registration can be problematic. For these patients, a *mandible-first approach to analytic model planning* for the achievement of accurate and reliable results is required.

References

1. *2000 Series Articulator,* Whip Mix Corporation, 1 Farmington Ave, PO Box 17183, Louisville, KY 40217.
2. Adrien P, Schouver J: Methods for minimizing the errors in mandibular model mounting on an articulator. *J Oral Rehabil* 24:929, 1997.
3. *Amira* software, Houston, Texas, 2010, TGS. Available at http://www.tgs.com. Accessed January 4, 2010.
4. Assael LA: Editorial. *J Oral Maxillofac Surg* 67:2041, 2009
5. Becker CM, Kaiser DA, Schwalm C: Mandibular centricity: centric relation. *J Prosthet Dent* 83:158, 2000.
6. Bell W, Proffit W, White R: *Surgical correction of dentofacial deformities,* ed 6, Philadelphia, 1980, W.B. Saunders Company, pp 155–199.
7. Bell RB: Computer planning and intraoperative navigation in orthognathic surgery. *J Oral Maxillofac Surg* 69:592–605, 2011.
8. Buckley MJ, Tucker MR, Fredette SA: An alternative approach for staging simultaneous maxillary and mandibular osteotomies. *Int J Adult Orthodon Orthognath Surg* 2:75, 1987.

9. Campos AA, Nathanson D, Rose L: Reproducibility and condylar position of a physiologic maxillomandibular centric relation in upright and supine body position. *J Prosthet Dent* 76:282, 1996.
10. Chow TW, Clark RKF, Cooke MS: Errors in mounting maxillary casts using face-bow records as a result of an anatomical variation. *J Dent Res* 13:277–282, 1985.
11. Cottrell DA, Wolford LM: Altered orthognathic surgical sequencing and a modified approach to model surgery. *J Oral Maxillofac Surg* 52:1010, 1994.
12. Ellis E, III: Accuracy of model surgery: evaluation of an old technique and introduction of a new one. *J Oral Maxillofac Surg* 48:1161, 1990.
13. Ellis E, III, Tharanon W, Gambrell K: Accuracy of face-bow transfer: effect on surgical prediction and postsurgical result. *J Oral Maxillofac Surg* 30:362, 1992.
14. Ellis E, III: Bimaxillary surgery using an intermediate splint to position the maxilla. *J Oral Maxillofac Surg* 57:53–56, 1999.
15. *Erickson Model Table,* Great Lakes Orthodontics, Ltd, 199 Fire Tower Drive, Tonawanda, NY 14151–15111.
16. Gateno J, Xia J, Teichgraeber J, et al: The precision of computer-generated surgical splint. *J Oral Maxillofac Surg* 61:814, 2003.
17. Gateno J, Teichgraeber JF, Xia JJ: Three-dimensional surgical planning for maxillary and midface distraction osteogenesis. *J Craniofac Surg* 14:833–839, 2003.
18. Gateno J, Xia JJ, Teichgraeber JF, et al: Clinical feasibility of computer-aided surgical simulation (CASS) in the treatment of complex cranio-maxillofacial deformities. *J Oral Maxillofac Surg* 65(4):728–734, 2007.
19. Gateno J, Xia JJ, Teichgraeber JF: New 3-dimensional cephalometric analysis for orthognathic surgery. *J Oral Maxillofac Surg* 69:606–622, 2011.
20. Gateno J, Xia JJ, Teichgraeber JF: Effect of facial asymmetry on 2-dimensional and 3-dimensional cephalometric measurements. *J Oral Maxillofac Surg* 69:655–662, 2011.
21. Hellsing G, McWilliam JS: Repeatability of the mandibular retruded position. *J Oral Rehabil* 12:1, 1985.
22. Jasinevicius TR, Yellowitz JA, Vaughan GG, et al: Centric relation definitions taught in 7 dental schools: results of faculty and student surveys. *J Prosthodont* 9:87, 2000.
23. Keshvad A, Winstanley RB: An appraisal of the literature on centric relation: part I. *J Oral Rehabil* 27:823, 2000.
24. Keshvad A, Winstanley RB: An appraisal of the literature on centric relation: part II. *J Oral Rehabil* 27:1013, 2000.
25. Keshvad A, Winstanley RB: An appraisal of the literature on centric relation: part III. *J Oral Rehabil* 28:55, 2001.
26. Landes C, Sterz M: Proximal segment positioning in bilateral sagittal split osteotomy: Intraoperative controlled positioning by a positioning splint. *J Oral Maxillofac Surg* 61:1423, 2003.
27. Landes C: Proximal segment positioning in bilateral sagittal split osteotomy: intraoperative dynamic positioning and monitoring by sonography. *J Oral Maxillofac Surg* 62:22, 2004.
28. Lapp TH: Bimaxillary surgery without the use of an intermediate splint to position the maxilla. *J Oral Maxillofac Surg* 57:57–60, 1999.
29. Lubbers HT, Jacobsen C, Matthews F, et al: Surgical navigation in craniomaxillofacial surgery: expensive toy or useful tool? A classification of different indications. *J Oral Maxillofac Surg* 69:300–308, 2011.
30. *Maxilim* software, Mechelen, Belgium, 2010, Medicim Medical Image Computing. Available at http://www.medicim.be. Accessed January 4, 2010.
31. McCormick SU, Drew SJ: Virtual model surgery for efficient planning and surgical performance. *J Oral Maxillofac Surg* 69:638–644, 2011.
32. Nattestad A, Vedtofte P, Mosekilde E: The significance of an erroneous recording of the center of mandibular rotation in orthognathic surgery. *J Craniomaxillofac Surg* 19:254, 1991.
33. O'Malley AM, Milosevic A: Comparison of three face bow/semi-adjustable articulator systems for planning orthognathic surgery. *Br J Oral Maxillofac Surg* 38:185, 2000.
34. Perez D, Ellis E, III: Sequencing bimaxillary surgery: mandible first. *J Oral Maxillofac Surg* 69:2217–2224, 2011.
35. Politi M, Toro C, Costa F, et al: Intraoperative awakening of the patient during orthognathic surgery: a method to prevent the condylar sag. *J Oral Maxillofac Surg* 65:109, 2007.
36. Posnick JC, Fantuzzo JJ, Orchin J: Deliberate operative rotation of the maxillo-mandibular complex to alter the A-point to B-point relationship for enhanced facial esthetics. *J Oral Maxillofac Surg* 64:1687–1695, 2006.
37. Posnick JC, Ricalde P, Pong NG: A modified approach to "model planning" in orthognathic surgery for patients without a reliable centric relation. *J Oral Maxillofac Surg* 64:347–356, 2006.
38. Quevedo LA, Ruiz JV, Quevedo CA: Using a clinical protocol for orthognathic surgery and assessing a 3-dimensional virtual approach: current therapy. *J Oral Maxillofac Surg* 69:623–637, 2011
39. *Quick Mount Face-Bow,* Whip Mix Corporation, 361 Farmington Ave, PO Box 17183, Louisville, KY 40217.
40. Rinchuse DJ, Kandasamy S: Articulators in orthodontics: an evidence-based perspective. *Am J Orthod Dentofacial Orthop* 129:299–308, 2006.
41. Rinchuse DJ, Kandasamy S: Centric relation: a historical and contemporary orthodontic perspective. *J Am Dent Assoc* 137:494–501, 2006.
42. Schendel S, Jacobsen R: Three-dimensional imaging and computer simulation for office-based surgery. *J Oral Maxillofac Surg* 67:2107, 2009.
43. Simon RL, Nicholls JI: Variability of passively recorded centric relation. *J Prosthet Dent* 44:21, 1980.
44. Swennen RJ, Mollemans W, Schutyser F: Three-dimensional treatment planning of orthognathic surgery in the era of virtual imaging. *J Oral Maxillofac Surg* 67:2080, 2009.
45. Tarantola GJ, Becker IM, Gremillion H: The reproducibility of centric relation: a clinical approach. *J Am Dent Assoc* 128:1245, 1997.
46. The glossary of prosthodontic terms. *J Prosthet Dent* 58:713, 1987.
47. The glossary of prosthodontic terms. *J Prosthet Dent* 71:40, 1994.
48. The glossary of prosthodontic terms. *J Prosthet Dent* 81:39, 1999.
49. The glossary of prosthodontic terms. *J Prosthet Dent* 94:10, 2005.
50. Thomas PM: Altered orthognathic surgical sequencing and a modified approach to model surgery [discussion]. *J Oral Maxillofac Surg* 52:1020–1021, 1994.
51. Truitt J, Strauss RA, Best A: Centric relation: a survey study to determine whether a consensus exists between oral and maxillofacial surgeons and orthodontists. *J Oral Maxillofac Surg* 67:1058–1061, 2009.
52. Turvey T: Sequencing of two-jaw surgery: the case for operating on the maxilla first. *J Oral Maxillofac Surg* 69:2225, 2011.
53. Van Sickels JE, Larsen AJ, Triplett RG: Predictability of maxillary surgery: a comparison of internal and external reference marks. *Oral Surg Oral Med Oral Pathol* 61:542, 1986.
54. Xia J, Gateno J, Teichgraeber J: New clinical protocol to evaluate craniomaxillofacial deformity and plan surgical correction. *J Oral Maxillofac Surg* 67:2093, 2009.
55. Xia JJ, McGrory JK, Gateno J, et al: A new method to orient 3-dimensional computed tomography models to the natural head position: a clinical feasibility study. *J Oral Maxillofac Surg* 69:584–591, 2011.
56. Xia JJ, Shevchenko L, Gateno J, et al: Outcome study of computer-aided surgical simulation in the treatment of patients with craniomaxillofacial deformities. *J Oral Maxillofac Surg* 69:2014–2024, 2011.

15

Sequencing of Orthognathic Procedures: Step-by-Step Approach

JEFFREY C. POSNICK, DMD, MD

- Description and Sequencing of Orthognathic and Other Frequent Simultaneous Procedures: Step-by-Step Approach
- Conclusions

Maxillofacial surgeons and orthodontists are the primary clinicians who care for individuals who present with jaw deformities, malocclusions, and the associated facial dysmorphologies. When an orthognathic approach is indicated, there are a limited number of basic surgical techniques that are used to facilitate the three-dimensional repositioning of the skeletal units (i.e., the maxilla, the mandible, and the chin region) to achieve desired improvements in head and neck function and to enhance facial aesthetics. By and large, these options include the Le Fort I (Type) osteotomy, with or without segmentation of the down-fractured maxilla; sagittal split ramus osteotomies of the mandible; and an oblique osteotomy of the chin. Each of these three basic osteotomies and its associated fixation techniques has evolved gradually to its current usage (see Chapter 2).[1-6]

This chapter provides the surgeon with a step-by-step technical description for the safe execution of Le Fort I osteotomy of the maxilla, sagittal split ramus osteotomies of the mandible, osseous genioplasty, and other frequent simultaneously performed procedures (e.g., septoplasty, reduction of inferior turbinates, recontouring of the nasal floor, removal of wisdom teeth, neck region soft-tissue rejuvenation) as I routinely carry them out (Video 6 and 7). The approach is described as it is typically executed for the patient who is undergoing all of these procedures simultaneously. An individual's unique anatomy and clinical presentation may require variations in surgical approaches and sequencing of treatment.

Description and Sequencing of Orthognathic and Other Frequent Simultaneous Procedures: Step-by-Step Approach

Preparation and Draping

Step 1. The patient is placed supine on the operating room table with the head in a Mayfield (horseshoe) headrest in the neutral neck position.

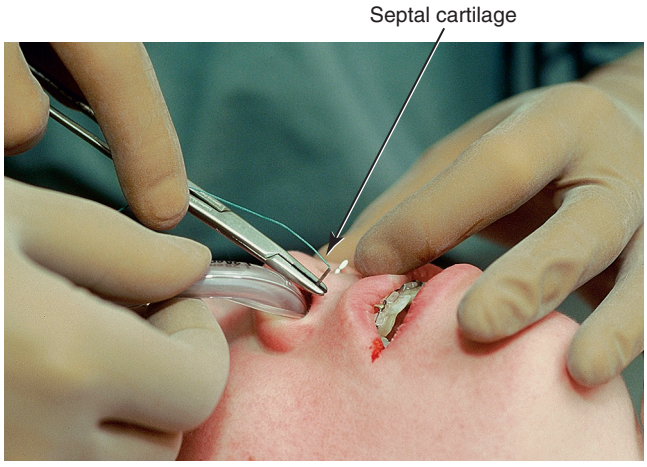
Step 2. A nasotracheal cuffed (Ring-Adair-Elwyn [RAE]) tube is inserted by the anesthesiologist and then secured by the surgeon to the cartilaginous septum with suture (#0 Ethibond). An additional suture (#0 Ethibond) is placed in the midline anterior hair-bearing scalp and then around the endotracheal tube extension over the top of the head to further secure it and to limit tension on the nose (Fig. 15-1).

Step 3. Ophthalmic antibiotic ointment and protective corneal shields are placed beneath the eyelids (or temporary tarsorrhaphies are performed, if preferred) to prevent corneal injury during the operation (Fig. 15-2, *A* through *C*).

Step 4. The anesthesiologist places monitoring equipment for the safe delivery of deliberate hypotensive anesthesia (i.e., arterial line, Foley catheter, large-bore venous lines, pulse oximeter, carbon dioxide monitor, and temperature probe). The bed is then positioned at approximately 15 degrees reverse Trendelenburg (see Chapter 11).

Step 5. Antibiotics (e.g., Cefazolin (Ancef); 15 to 20 mg/kg and up to 1 g) and steroids (e.g., Dexamethasone 0.5 mg/kg/dose, max: 8 mg) are administered intravenously by the anesthesiologist.

Step 6. The head and neck are prepped with povidone–iodine (Betadine) solution. Avoid using soap or alcohol solutions that could irritate the cornea and the mucous membranes. Draping should provide for adequate exposure of the forehead, the eyes,



• **Figure 15-1** Step 2.

the external ears, the face, and the neck. Staples are used to secure the drapes (Fig. 15-3).

Step 7. The mouth and pharynx are suctioned, and a moistened gauze with a radiopaque marker is inserted as a throat pack. The teeth and dorsum of the tongue are cleansed with a sterile toothbrush (1.5% hydrogen peroxide or 0.12% chlorhexidine). The mouth is irrigated with normal saline solution and suctioned.

Step 8. Steroid cream (e.g., betamethasone 0.5%) is applied to the lips to limit edema that could otherwise result from contusion during surgery.

Step 9. Lidocaine solution (Xylocaine 1% with 1 : 100,000 epinephrine) is infiltrated into the lateral vestibules of the mandible, the facial vestibule of the chin, and the labial vestibules of the maxilla.

Cortical Cuts for Sagittal Split Ramus Osteotomies (Video 6:2 and 7:2)

Step 10. A medium rubber mouth prop is placed between the maxillary and mandibular molars on one side to maintain mouth opening. Attention is turned to the lateral vestibule of the contralateral mandible.

Step 11. A. *Incision Option One:* No erupted wisdom tooth for extraction. If there is no erupted wisdom tooth to extract (i.e., if the wisdom tooth is fully impacted, absent, or erupted and is to remain in place), an incision is made with a Bovie electrocautery device or a knife (no. 15 blade) in the depth of the vestibule adjacent to the second molar (Fig. 15-4, *A*). The incision extends both anteriorly and posteriorly for a total length of 4 cm. The incision is carried down to bone. A sufficient cuff of mucosa is maintained adjacent to the gingiva of the molar teeth for relaxed wound closure.

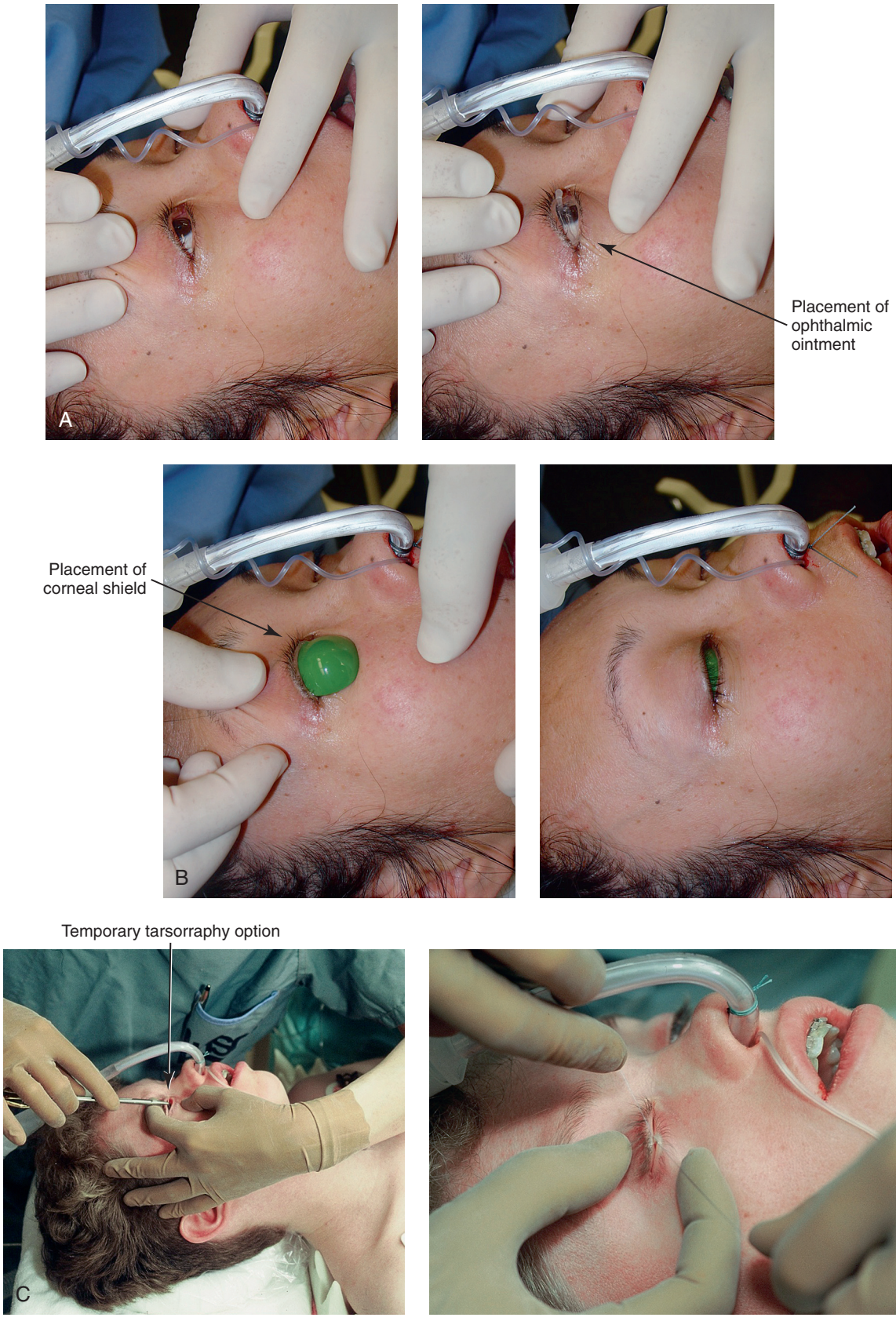
B and C. *Incision Option Two:* Erupted or partially erupted wisdom tooth for extraction. If there is a partially erupted (Fig. 15-4, *B*) or fully erupted (Fig. 15-4, *C*) wisdom tooth to be removed, the incision starts 1 cm distal and lateral to the wisdom tooth and extends to the distobuccal line angle of the tooth. The incision continues anteriorly along the cervical gingival margin of the tooth within the sulcus and then anterior and lateral into the depth of the vestibule. The incision is carried down to bone. A sufficient cuff of mucosa, including part of the papilla, is maintained adjacent to the gingiva of the second molar for relaxed wound closure.

Step 12. The subperiosteal dissection exposes the anterior aspect of the lateral ramus and the posterior body of the mandible down to the inferior border (Fig. 15-5, *A*). The extent of the dissection anterior to the gonial angle depends on the planned buccal extension of the proximal segment. The dissection exposes the anterior superior aspect of the ramus up toward the coronoid process, with stripping of the temporalis muscle tendon (Fig. 15-5, *B C*). The dissection exposes the medial aspect of the ramus superior toward the sigmoid notch and inferior toward the mandibular canal (Fig. 15-6, *A*). (I do not dissect below the inferior alveolar neurovascular bundle or circumferentially around it, because I believe this may cause additional contusion and paresthesia.)

Step 13. Retraction of the soft tissues is performed with a curved Kocher clamp on the coronoid process (superior exposure); a channel retractor superior to the neurovascular bundle (medial ramus exposure); an inferior border retractor placed anterior to the gonial angle (anterior exposure); and a tongue retractor (lingual exposure) (Fig. 15-6).

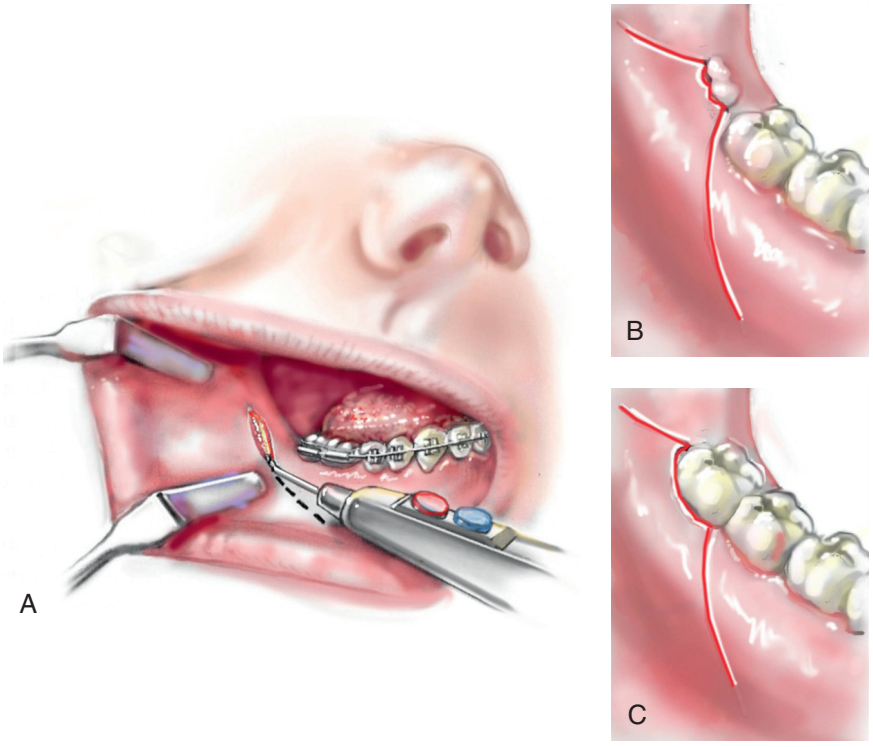
Step 14. With a sterile pencil, the osteotomy lines are marked out for the sagittal splitting of the ramus of the mandible. A reciprocating saw (i.e., with a short, straight blade) completes the cortical osteotomies.

A. The first cut is made horizontally through the medial cortex of the ramus. This is judged to be just superior to the occlusal plane of the mandibular molars and well inferior to the region of the anterior aspect of the ramus, where the medial and lateral cortices join and no longer maintain a medullary cavity. The medial osteotomy need not extend more than 2 cm posterior (Hunsuck modification) (see insert of Fig. 15-6, *A*). By keeping the medial cut "low" (i.e., close to the mandibular molar occlusal surface) and "short" (i.e., only 2 cm back), the incidence of a "bad" split (i.e., with

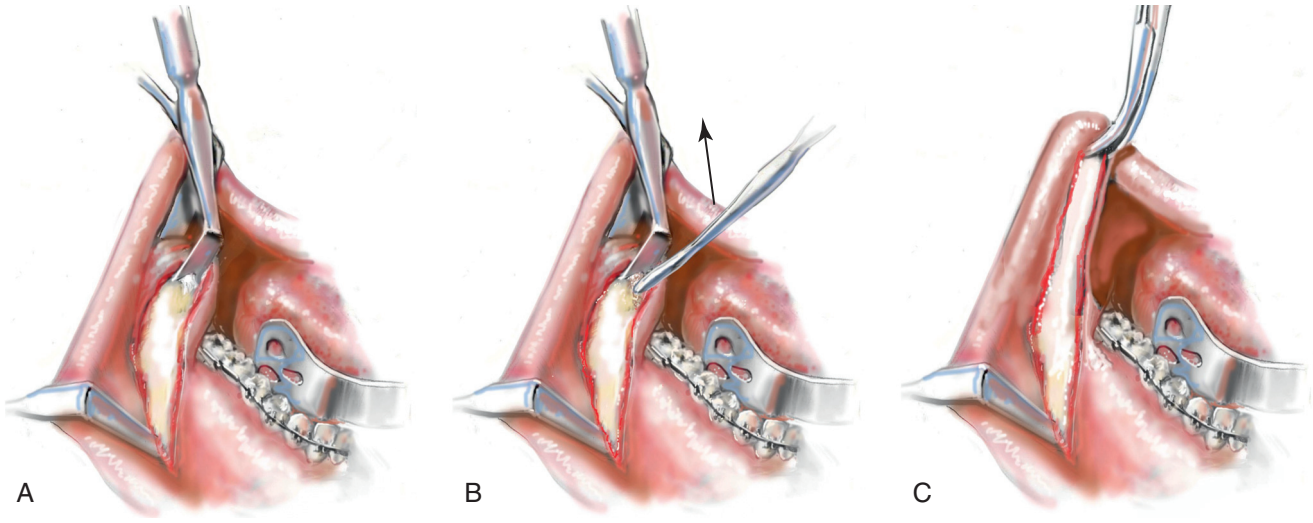


• **Figure 15-2** Step 3.

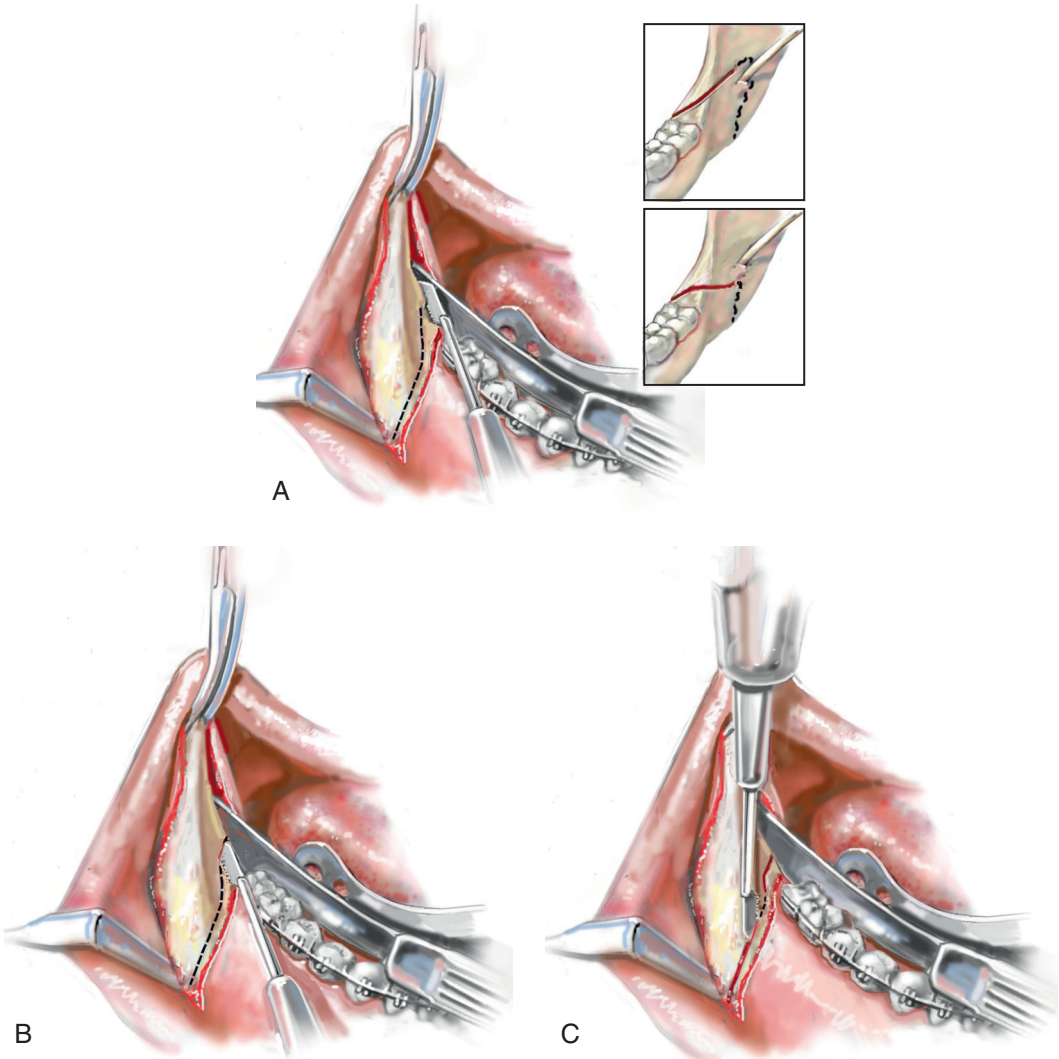
• **Figure 15-3** Step 6.



• **Figure 15-4** Step 11, A, B, and C.



• **Figure 15-5** Step 12.



• **Figure 15-6** Step 14, A, B, and C.

the condyle as a third segment) is greatly reduced.

B. The osteotomy then extends anteriorly and just lateral (buccal) to the molars. It is important that the saw blade penetrate the cortex and enter the medullary cavity in all locations. If the wisdom tooth is impacted, the saw blade will bounce off the wisdom tooth just after penetrating the superior paper-thin cortex (Fig. 15-6, *B*); this will be just lateral to the second molar. The blade will penetrate back into the medullary cavity as the osteotomy progresses anterior past the impacted tooth.

C. The reciprocating saw is then repositioned to complete the vertical cortical osteotomy. The osteotomy location will depend on the planned buccal extension of the proximal segment (i.e., the Dal Point modification.) The vertical osteotomy begins at the inferior border of the mandible, where the inferior alveolar nerve is close to the cortex and prone to laceration (Fig. 15-6, *C*). The osteotomy then continues superiorly to join the cortical osteotomy, which was previously completed lateral to the molars.

COMMENT: When later "splitting" the ramus (see Steps 53 through 58), as the inferior alveolar nerve enters the mandibular foramen, it may remain within either the proximal segment or the distal segment (see Fig. 15-6, *A* inset). Traditional thinking is that the medial osteotomy *must* remain above the mandibular foramen as it extends posterior. I believe that it is more important to keep the medial osteotomy low and short; this is a reliable way to achieve a favorable split with limited risk of the condylar component inadvertently remaining part of the distal segment. After the low and short split is completed, I do not favor the automatic need to completely remove all aspects of the inferior alveolar nerve that may remain within the proximal segment. This is especially true at the mandibular foramen location. I do not drill out or curette the inferior alveolar nerve from the proximal segment unless it would otherwise be under tension after repositioning the distal segment. In my experience, however, this is not usually the case.

Step 15. If the wisdom tooth is erupted and is to be removed, then the extraction is completed now or just before the cortical osteotomies; doing so before the actual split will limit a fracture of the lingual plate that may otherwise occur during dental extraction. A rotary drill with a tapered fissure bur may be used to judiciously remove bone adjacent to the tooth and to section the tooth to facilitate an atraumatic extraction.

Step 16. The wound is irrigated and suctioned. The retractors are removed, and two 1 inch × 3 inch cottonoids are placed for packing (i.e., hemostasis). One is placed along the medial ramus, and the other is placed along the lateral body of the mandible.

Step 17. Attention is turned to the contralateral ramus of the mandible, and identical procedures to those described in Steps 10 through 16 are carried out.

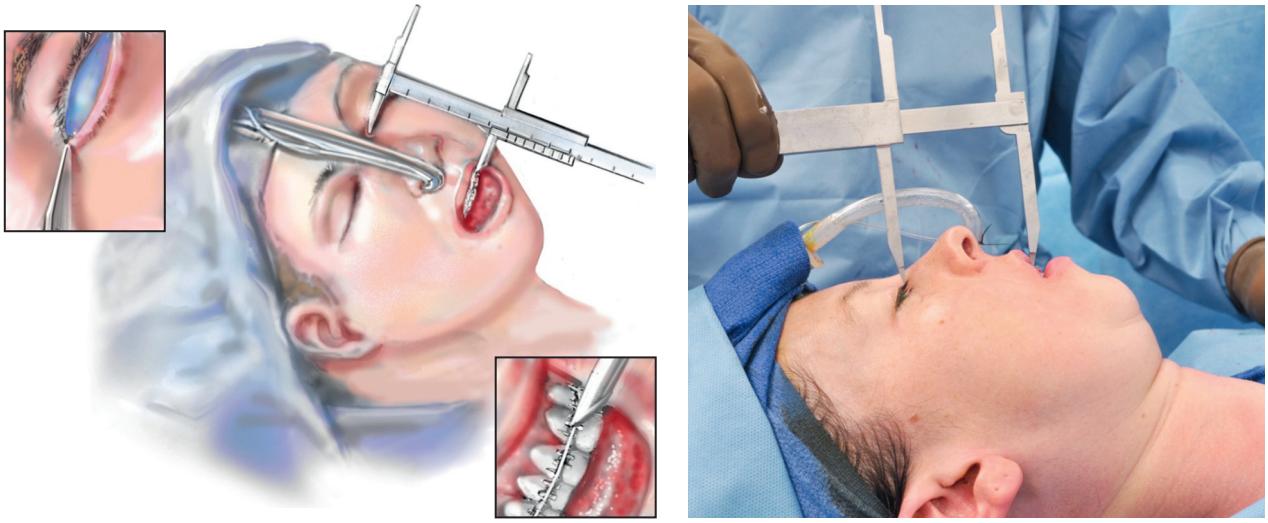
Maxillary (Le Fort I) Osteotomy
(Video 6:3 and 7:3)

Step 18. With the use of a Tessier caliper, the vertical distance (height) between the medial canthus and the mid-maxillary incisor crown is measured on the left and right sides and recorded; this generally measures between 55 mm and 70 mm (Fig. 15-7). This is a reproducible relative measure of the anterior vertical maxillary height (see Chapter 12).

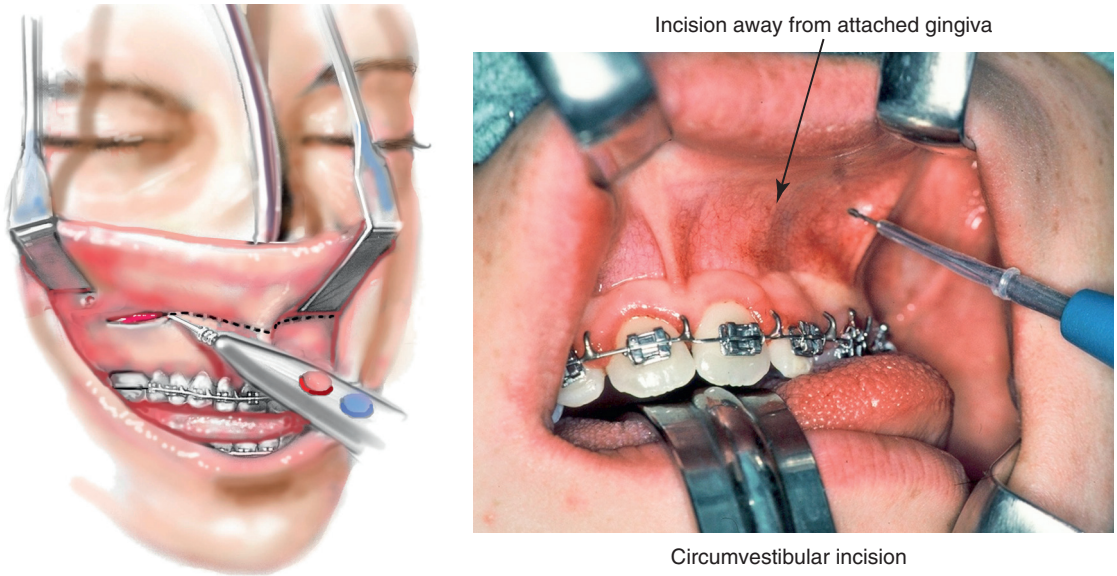
NOTE: If 70 mm is the baseline measurement and 3 mm of vertical intrusion at the incisors is a surgical objective, then, after maxillary repositioning, 67 mm should be the new measurement before the placement of fixation across the Le Fort I osteotomy.

Step 19. Attention is turned to the vestibule of the maxilla on one side. Two medium "toed-in" retractors are used to retract the lip and cheek and thus expose the vestibule. A circumvestibular incision is initiated from the zygomatic buttress region anteriorly toward the midline (Fig. 15-8). It is useful to remain deep in the vestibule and to leave a full cuff of mucosa adjacent to the attached gingiva of the teeth. The most posterior aspect of the incision remains just anterior to and on the dental (vestibular side) of the visualized parotid duct. The incision is made with a Bovie electrocautery device or a knife (#15 blade). Once the incision is made through the mucosa the knife is directed toward the dentoalveolar region and down to bone. This will avoid injury to the infraorbital nerve and limit the exposure of the buccal fat pad.

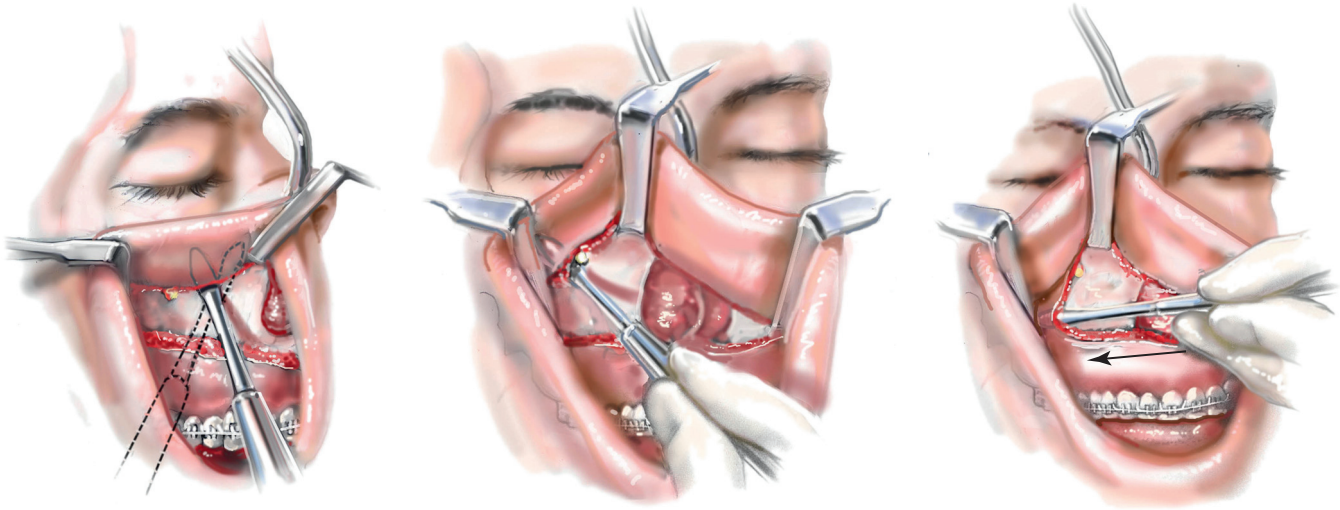
Step 20. Straight and curved elevators are used for the subperiosteal dissection of the anterior maxilla extending to and on either side of the infraorbital nerve; extending medially to expose the pyriform rim, the floor of the nose, and the anterior nasal spine; and extending posterior to the pterygomaxillary junction. A long "toed-out" retractor is placed subperiosteally in the pterygomaxillary space (Fig. 15-9).



• **Figure 15-7** Step 18.



• **Figure 15-8** Step 19.



• **Figure 15-9** Step 20.

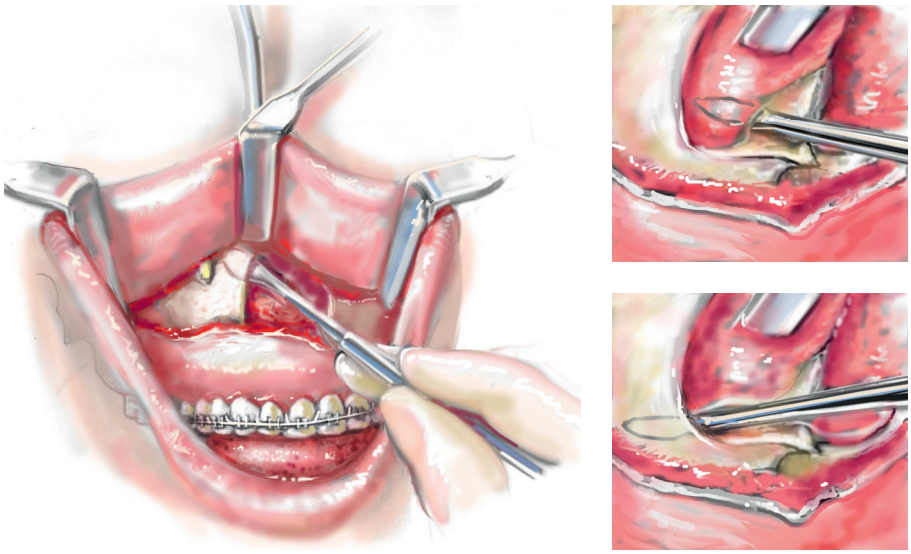
Step 21. The two medium toed-in retractors are shifted to the contralateral side of the maxilla, and the identical incision, dissection, and exposure are accomplished as described in Steps 19 and 20.

Step 22. Straight and curved elevators are used to further separate the nasal mucosa from each lateral nasal wall; the anterior nasal spine from the cartilaginous septum; and the nasal mucosa from the floor of the nose (Fig. 15-10).

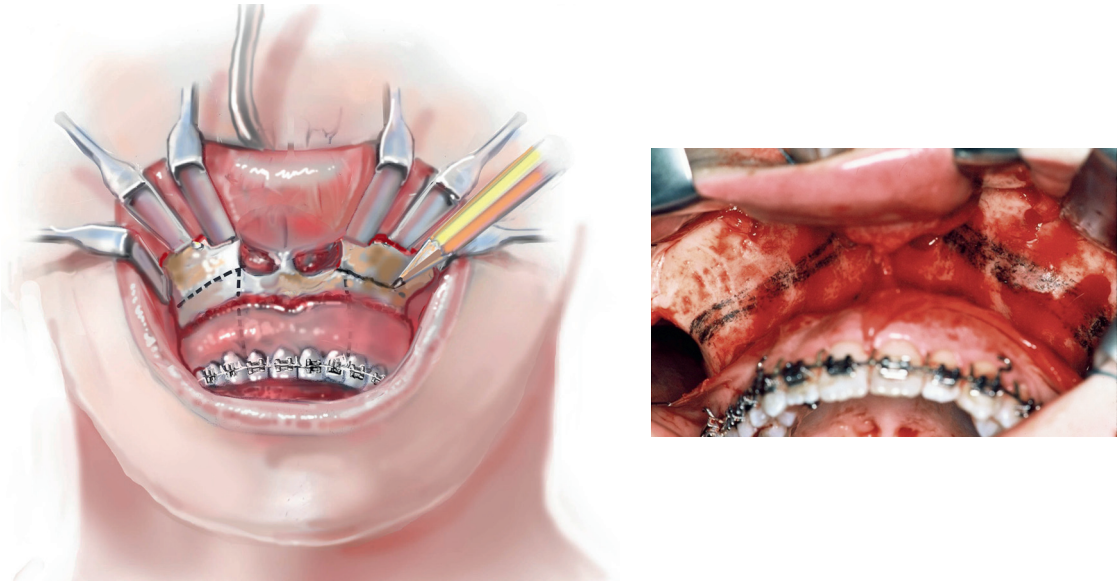
Step 23. With four medium toed-in retractors (one on either side of the infraorbital nerve on each side of the maxilla) and two long toed-out retractors (one in each pterygomaxillary junction) in place, the anterior maxilla is fully visualized. A sterile pencil is used to mark the location for the horizontal Le Fort I osteotomy (Fig. 15-11). If significant maxillary intrusion is planned, a second parallel osteotomy line is marked out. The lower marking (i.e., the osteotomy site) is placed to be within the sinus, above the roots of the teeth, into each lateral nasal aperture, and below each zygomatic buttress. If an ostectomy is to be preformed, the upper marking is made superior and parallel to the lower marking and below the infraorbital nerves (Fig. 15-11).

Segmentation of the Maxilla

Step 24. If segmentation of the maxilla is to be completed, the interdental osteotomies are initiated before down-fracture. This provides a stable "workbench" for the initiation of each interdental osteotomy.



• **Figure 15-10** Step 22.



• **Figure 15-11** Step 23.

The interdental osteotomies are typically made either between the lateral incisor and the canine on each side (Fig. 15-12, *A*) or between the central incisors (Fig. 15-12, *B*). The subperiosteal dissection of the gingival cuff provides adequate exposure of each interdental osteotomy location without the need for direct mucosa incisions. Each interdental osteotomy site is marked with a sterile pencil. The osteotomy is then initiated with an oscillating saw (i.e., a short fan blade on a long shaft) and later deepened after down-fracture with the oscillating saw (i.e., a longer fan blade on a long shaft).

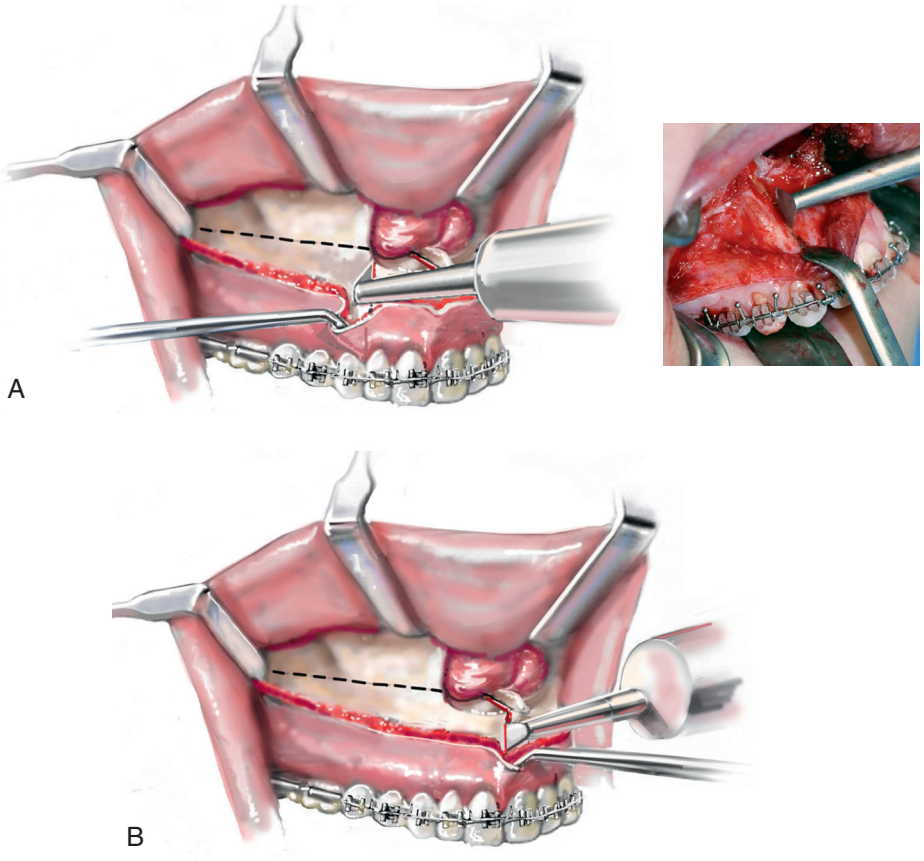
NOTE: The lateral incisor and canine roots—and, to a lesser extent, the two central incisor roots—are naturally divergent. Therefore, there is no advantage or need for the orthodontist to create a gap for the interdental osteotomy. Perforation into the periodontal ligament space with injury to the dental roots when completing these interdental osteotomies should be an uncommon occurrence.

Maxillary (Le Fort I) Osteotomy: Continued

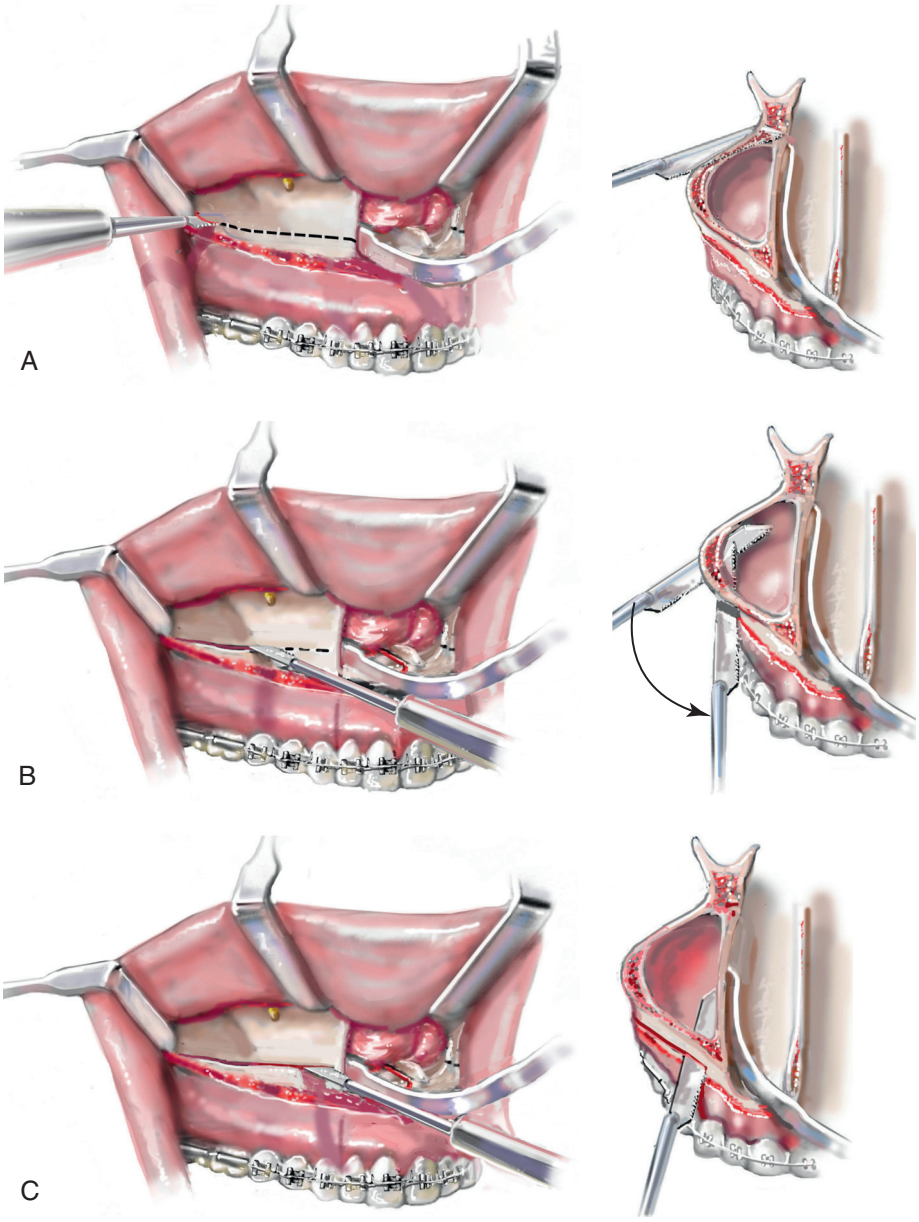
Step 25. With a reciprocating saw (i.e., a long straight blade), the Le Fort I horizontal osteotomy is completed through the lateral, anterior, and medial maxillary walls on each side (Fig. 15-13, *A* through *C*). Before the medial maxillary osteotomy is completed, the lateral nasal mucosa is protected with a small malleable retractor placed between the lateral nasal wall and the nasal mucosa. Before the lateral maxillary osteotomy is completed, the pterygoid fossa soft tissues are protected with a long, toed-out retractor.

Step 26. With a specialized guarded chisel and mallet, the septum of the nose (i.e., cartilage and bone) is separated from the maxilla. Care is taken to avoid penetration past the septum into the nasopharynx (Fig. 15-14).

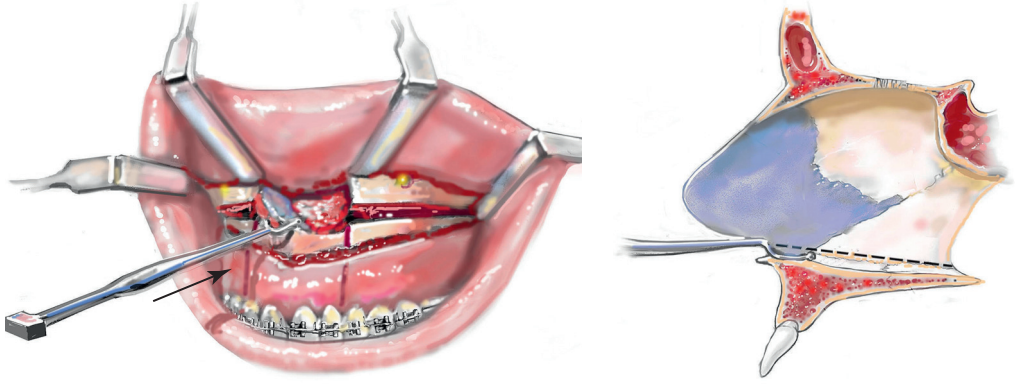
Step 27. With a specialized curved chisel and mallet, the pterygomaxillary suture is separated on each side (Fig. 15-15). Care is taken to avoid injury to the internal maxillary artery or to the pterygoid plexus and to avoid perforation through the palatal mucosa. For a patient with extreme maxillary hypoplasia (e.g., Treacher Collins syndrome), the



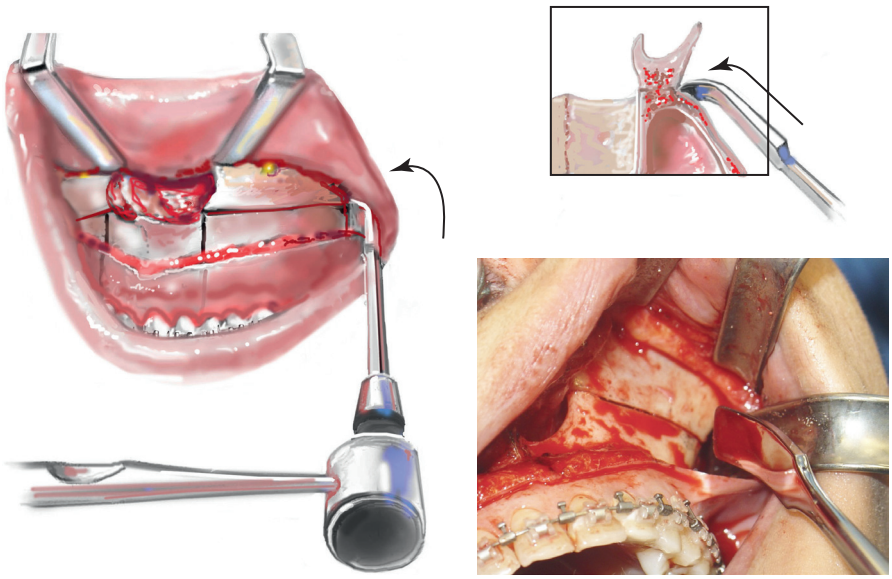
• **Figure 15-12** Step 24.



• **Figure 15-13** Step 25.



• **Figure 15-14** Step 26.



• **Figure 15-15** Step 27.

chisel may be within millimeters of the optic foramen and the optic nerve.

Step 28. With a Tessier (blunt) zygomatic hook placed on the ledge of the nasal sill, force is applied to "down-fracture" the Le Fort I osteotomy unit. While down-fracturing, a Frazier suction tip and a periosteal elevator are used by the assistant to further separate and to limit the tearing of the nasal mucosa (Fig. 15-16).

Step 29. After down-fracture, the maxillary sinus mucosa is generally stripped off of its bony surfaces with a Frazier suction tip and a hemostat (Fig. 15-17). This is done to remove chronic inflammatory tissue (polyps) from the sinus and to reduce hemorrhage.

Step 30. Placing the Tessier (blunt) hook behind the maxillary tuberosity and applying anterior and contralateral directed force to one side at a time assists with the disimpaction of the maxilla (Fig. 15-18). The advantage of the Tessier hook is that it assists with the mobilization of the maxilla without contusion of the palatal mucosa, which may occur with standard (Rowe) nasomaxillary disimpaction forceps. When disimpacting with the Tessier hook behind the tuberosity, the surgeon's other hand is used to grip the anterior maxilla (rather than the teeth) and to apply simultaneous anterior and contralateral force. This is helpful to prevent the fracturing of the tuberosity by the Tessier hook. This is repeated on each side two times to achieve maximum disimpaction. During this process, the assistant to the surgeon stabilizes the patient's head with one hand over each ear. For a maxillary disimpaction that requires more extensive advancement, the Rowe forceps may be required.

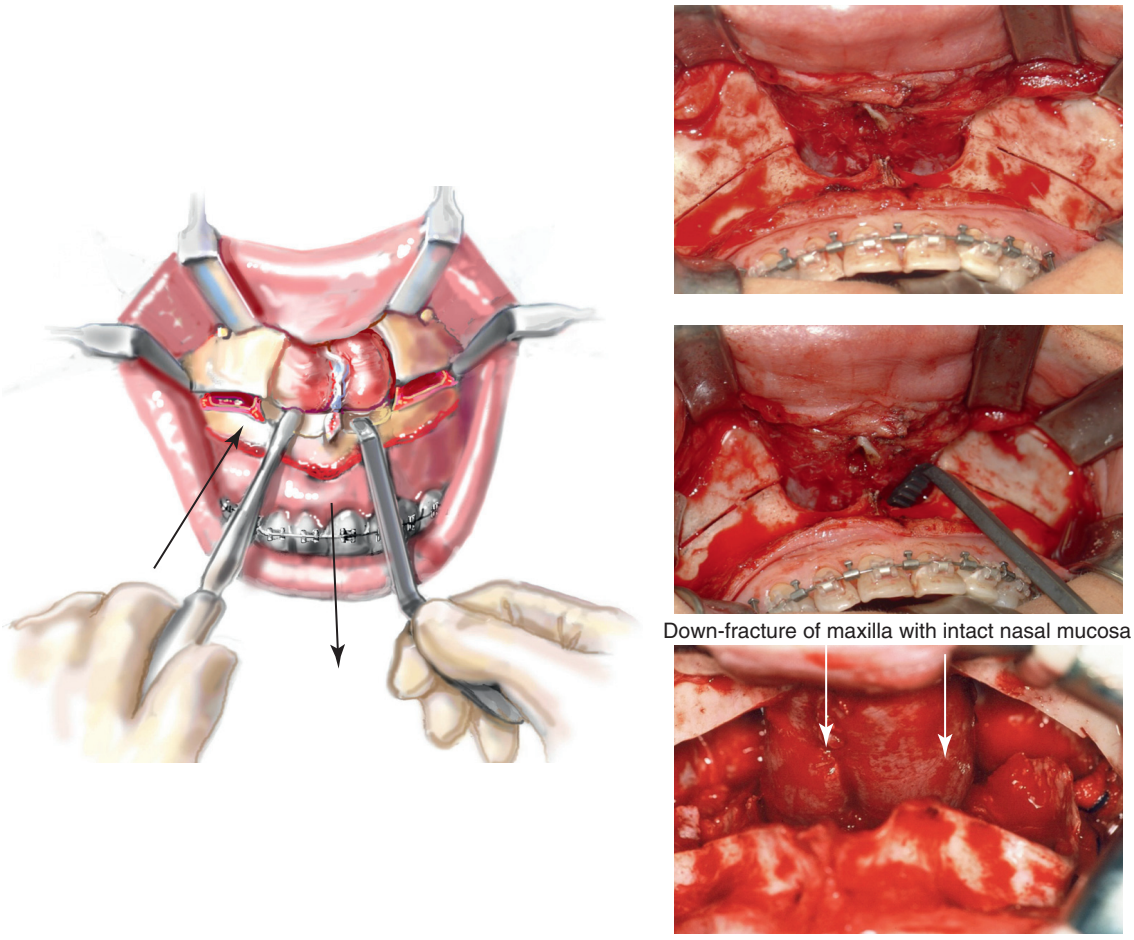
Septoplasty and the Reduction of the Inferior Turbinates (Video 6:4 and 7:4)

Step 31. When indicated on the basis of the patient's history and clinical examination, attention is turned to the removal of the deviated portions of the septum of the nose that are causing blockage of the airway.

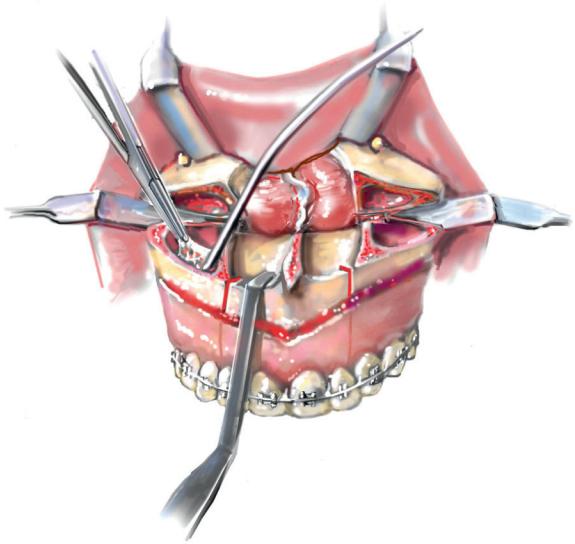
A. With an elevator, the submucosal dissection of the cartilaginous septum (i.e., the quadrangular cartilage) and the bony septum (i.e., the vomer and the perpendicular plate of the ethmoid) is carried out (Fig. 15-19, *A*).

B. The deviated and thickened aspects of the cartilaginous septum and the bony septum are resected with a rongeur (Fig. 15-19, *B*). The more anterior components of the cartilaginous septum that provides needed support to the nasal dorsum and the tip are not disturbed. This includes at least 1 cm of the dorsal and caudal cartilage. When a maxillary intrusion (i.e., for impaction) is planned, even if septoplasty is not required to correct chronic nasal obstruction, the inferior aspect of the septum (i.e., the bone and cartilage) is removed to prevent buckling.

Step 32. When indicated by the patient's history and physical examination, attention is turned to reducing each hypertrophic inferior turbinate that is causing blockage of the airway. An incision is made through the nasal mucosa just below and parallel to each turbinate (Fig. 15-20, *A*). If the nasal mucosa was torn during down-fracture, the laceration is used and may be extended for improved turbinate exposure. Each enlarged



• **Figure 15-16** Step 28.



• **Figure 15-17** Step 29.

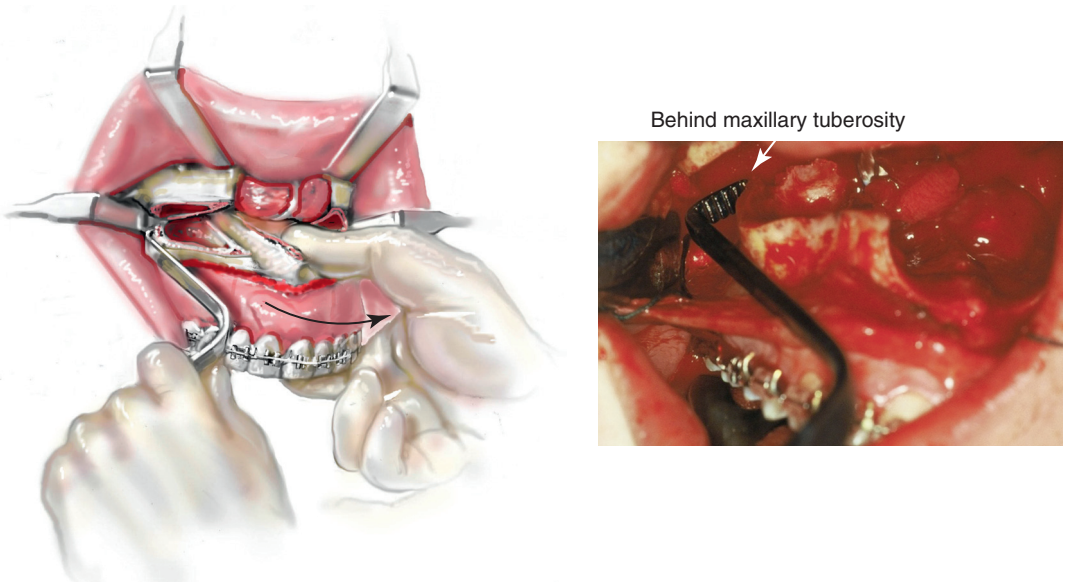
inferior turbinate is visualized (Fig. 15-20, *B*), and the inferior aspect of each is resected with a straight Mayo scissors (Fig. 15-20, *C*). Hemostasis is obtained along the cut edge of each turbinate with the Bovie electrocautery device (Fig. 15-20, *D*). The nasal mucosa is reapproximated with interrupted sutures (3-0 Vicryl [polyglactin 910]) to create a closure that is as watertight as possible (Fig. 15-20, *E*).

NOTE: To prevent a fire in the operative field, if there are any air leaks around the nasotracheal tube, then the electrocautery device is not used.

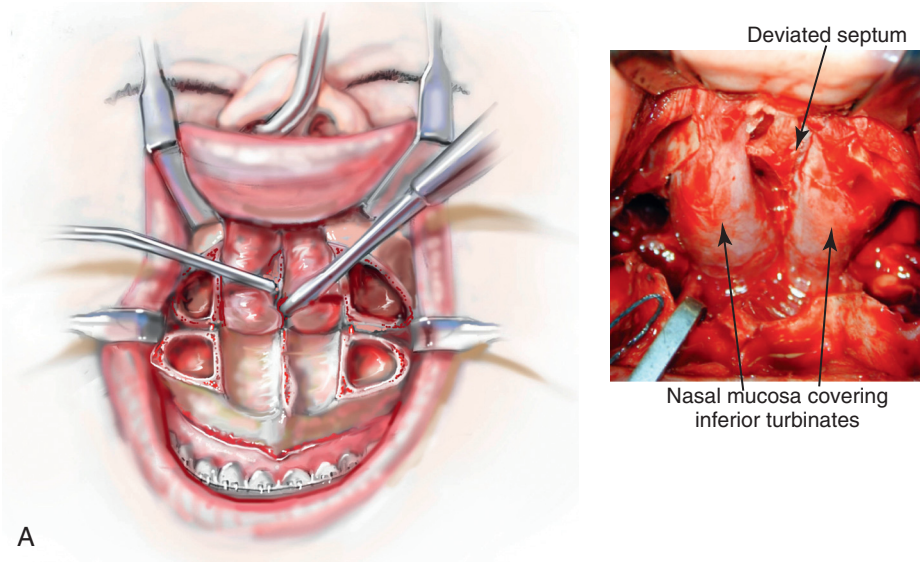
Removal of the Maxillary Wisdom Teeth (Video 6:5)

Impacted Wisdom Teeth

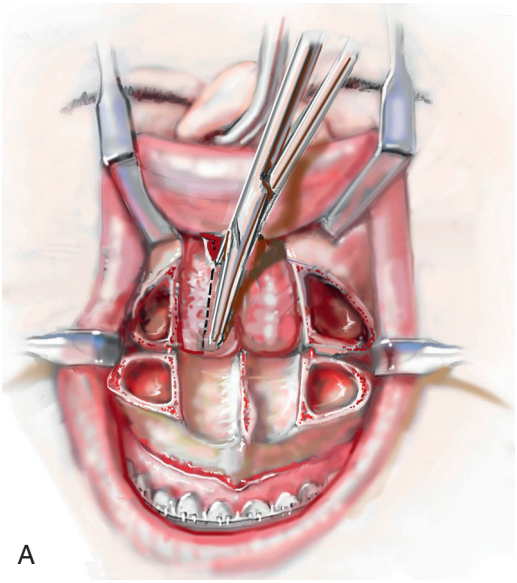
Step 33. A. If indicated, each bony impacted maxillary wisdom tooth is removed through the sinus floor (Fig. 15-21). A rotary drill with a small watermelon bur is used to remove bone from the sinus floor that is directly over the impacted



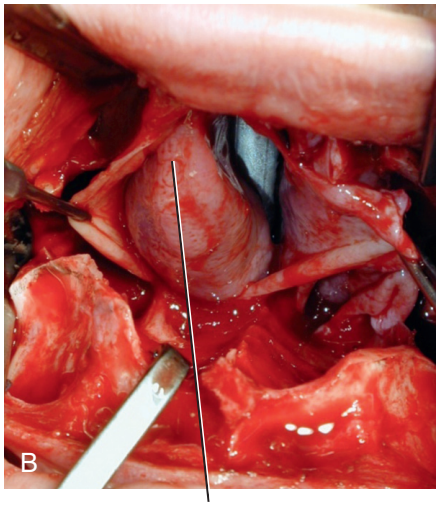
• **Figure 15-18** Step 30.



• **Figure 15-19** Step 31, A and B.

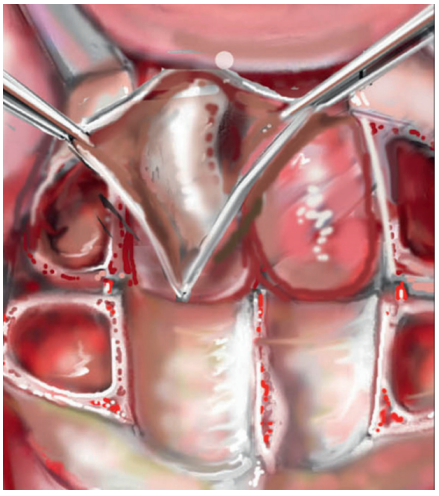


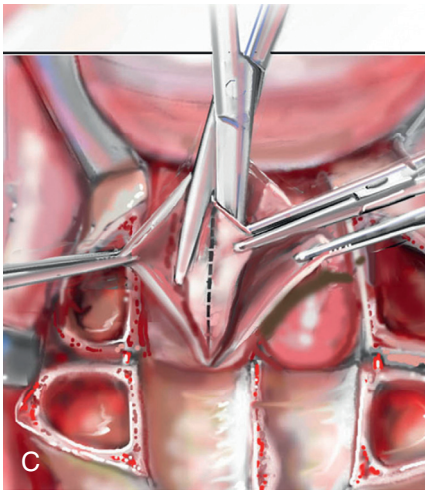
A



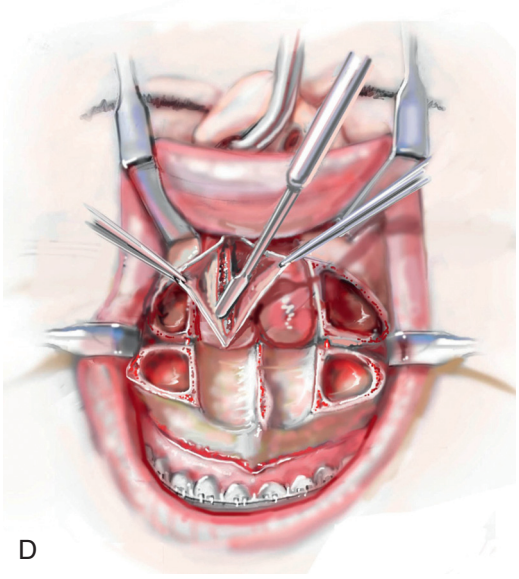
B

Inferior turbinate exposed after elevation of nasal mucosal flaps

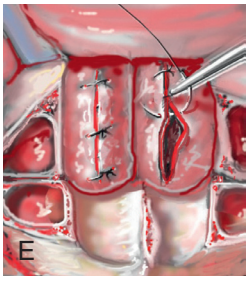




C

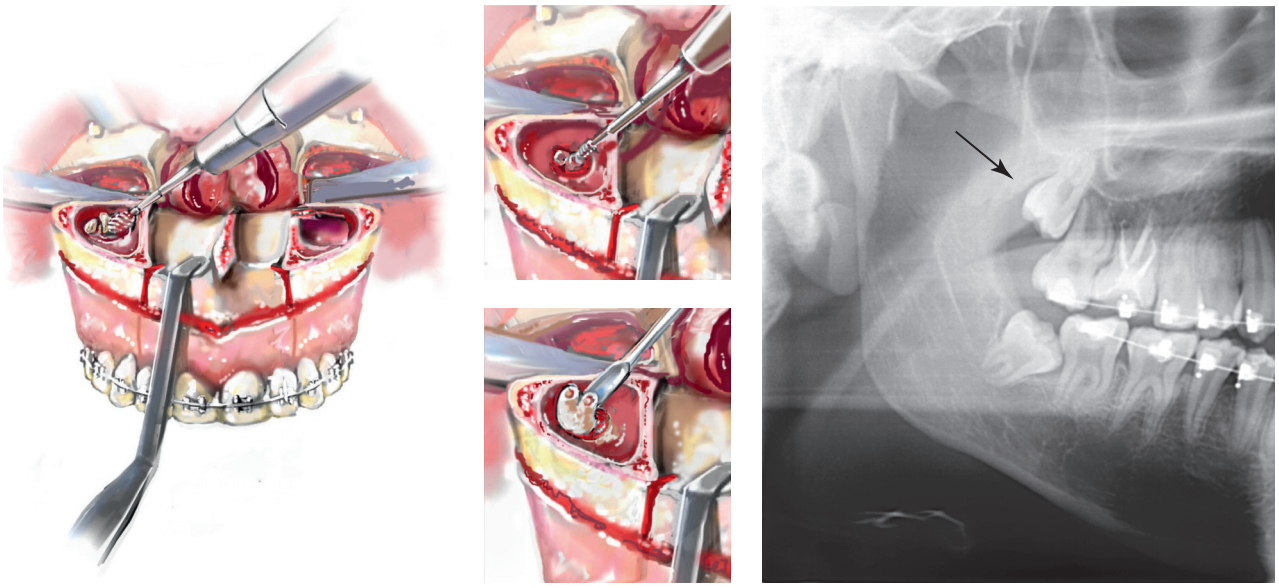


D



E

• **Figure 15-20** Step 32.



• **Figure 15-21** Step 33.

tooth. Next, using a rotary drill with a tapered fissure bur, bone is removed adjacent to the impacted tooth. Depending on the extent of tooth development, the impacted wisdom teeth may also be sectioned to assist with removal. A dental elevator is used to remove the tooth. Perforation through the palatal mucosa is an uncommon occurrence.

Erupted Wisdom Teeth

B. If indicated, each erupted maxillary wisdom tooth is removed before down-fracture. This provides a stable "workbench" at the time of extraction. To preserve maximum circulation to the down-fractured maxilla, it is important to minimize the disruption of the adjacent palatal and labial mucosa at the time of extraction.

Segmentation of the Maxilla: Continued

If segmentation of the maxilla is to be carried out, the interdental cuts that were initiated previously are completed during this stage.

Three Segments

Step 34. A. Description of a three-piece Le Fort I osteotomy with interdental cuts between the lateral incisor and the canine on each side connected to parasagittal osteotomies through the hard palate:

(1) The parasagittal maxillary osteotomy is completed through the down-fracture using a reciprocating saw with a short, straight blade. The osteotomy is from the posterior edge of the hard palate anteriorly to the pre maxillary region (Fig. 15-22, *A-1*).

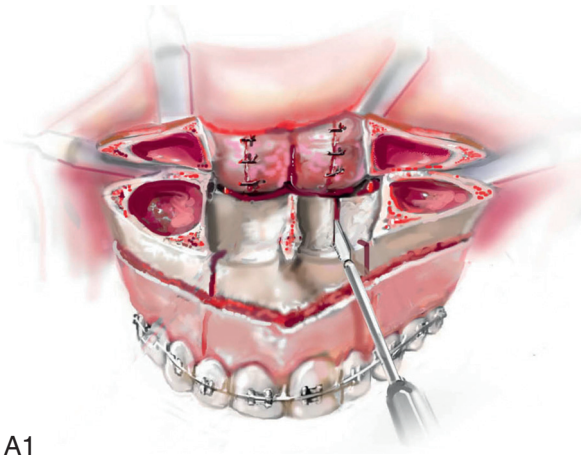
(2) If the planned posterior arch-width expansion is significant, then a second parasagittal osteotomy is also completed on the contralateral side (Fig. 15-22, *A-2*). The two parallel parasagittal osteotomies allow for a more tension-free expansion than a single osteotomy.

(3) The previously initiated interdental osteotomies are deepened using an oscillating saw with a longer, fan blade on a long shaft (Fig. 15-22, *A-3*).

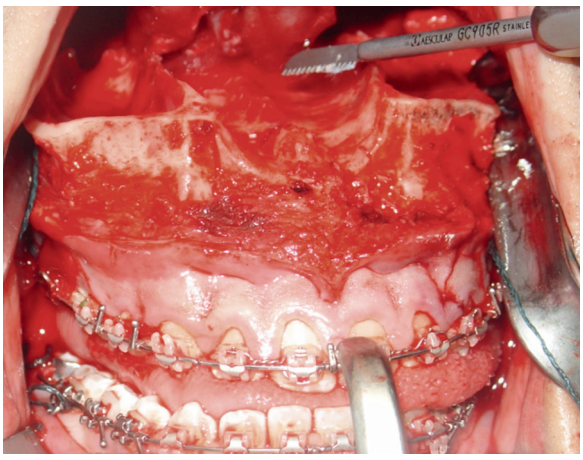
(4) The oscillating saw with a longer, fan blade on a long shaft is also used to connect each interdental osteotomy to the parasagittal osteotomy along the floor of the nose at the level of the incisal foramen (Fig. 15-22, *A-4*).

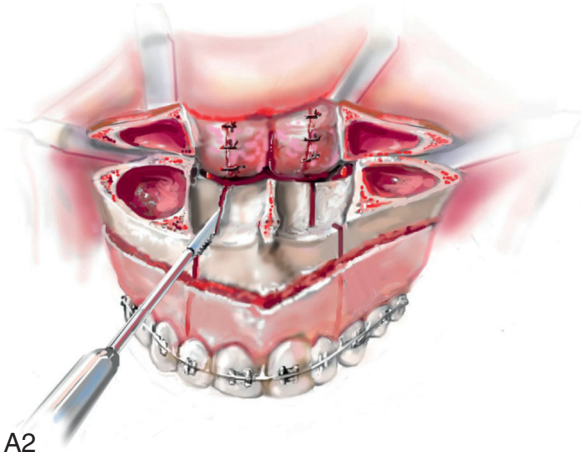
(5) A fine chisel (i.e., 5 mm or 7 mm in width) is placed into the interdental osteotomy on the same side as the parasagittal osteotomy, and an elevator is placed into the parasagittal palatal osteotomy (Fig. 15-22, *A-5*). Twisting the elevator and chisel simultaneously assists with the splitting of the maxilla into segments.

(6) The fine chisel (i.e., 5 mm or 7 mm) is then placed into the other interdental osteotomy site, and an elevator is placed in the nasal floor osteotomy site. With a twisting motion, separation is completed (Fig. 15-22, *A-6*). Perforation through the palatal mucosa (when completing

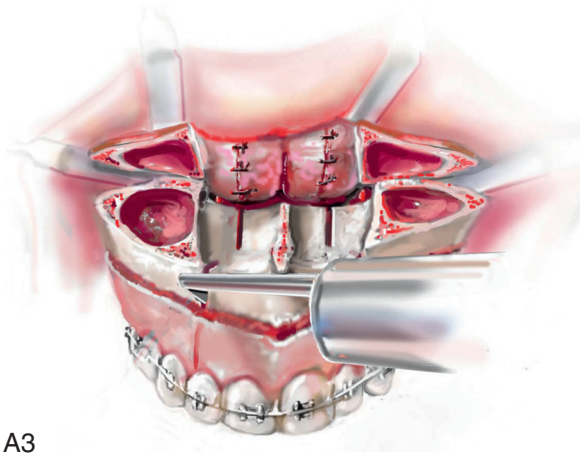


A1

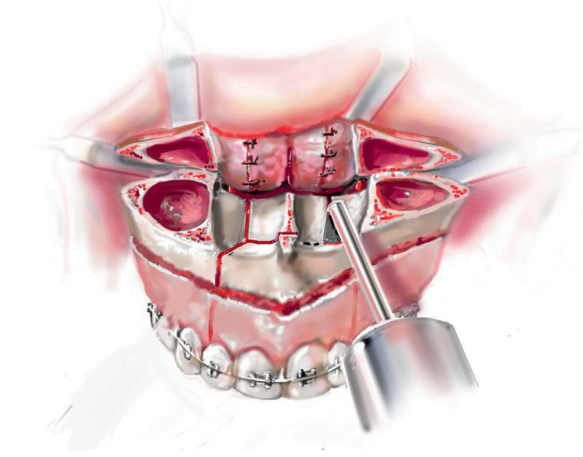




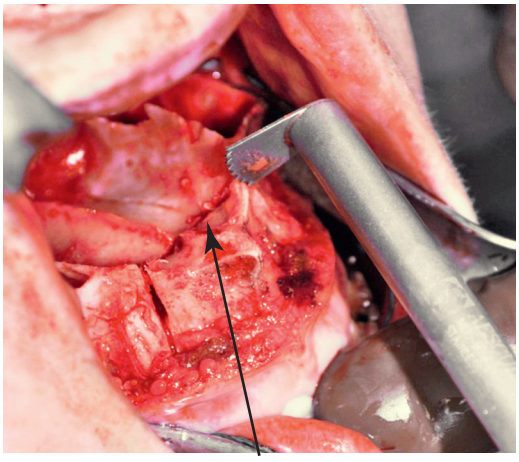
A2



A3

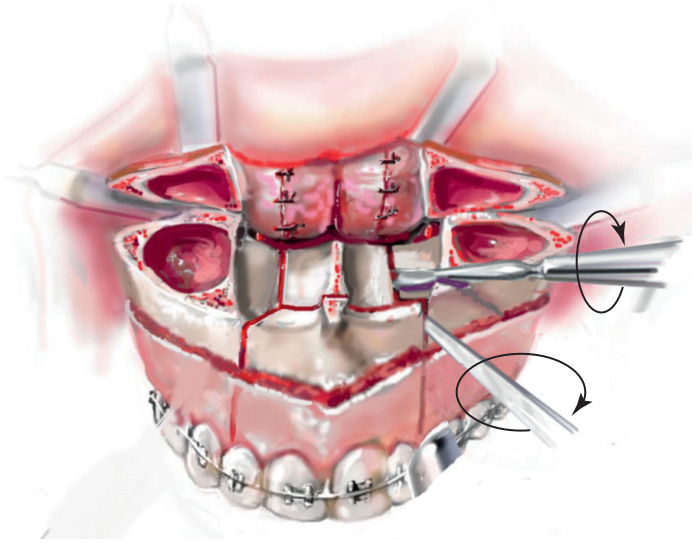


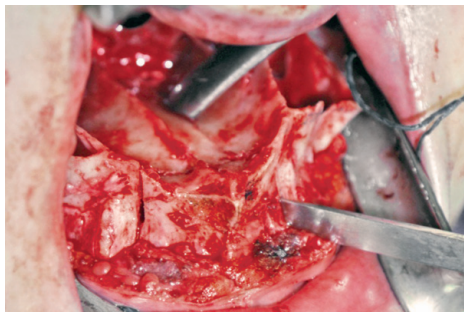
A4

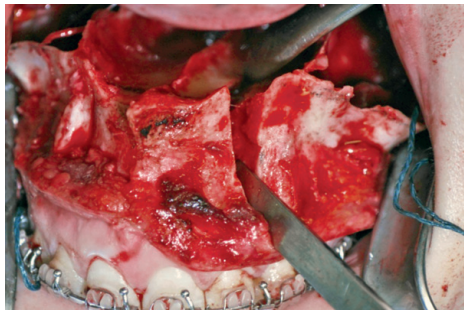


Osteotomy across hard palate to separate premaxillary segment

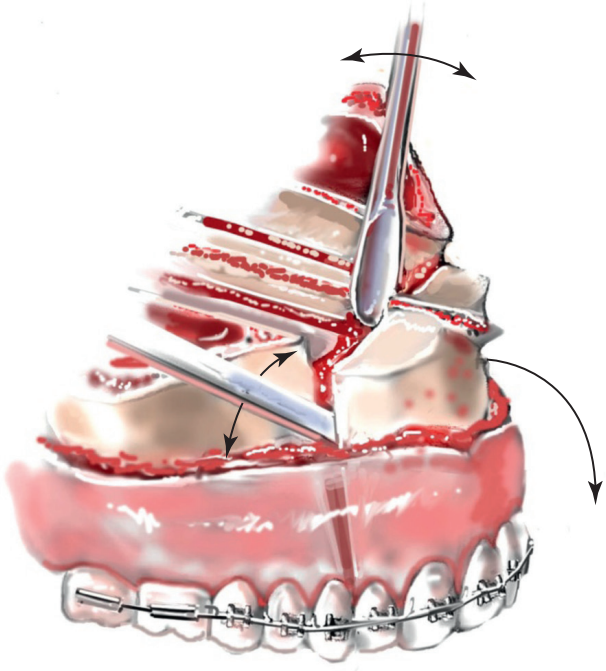
• **Figure 15-22 A,** Step 34, A, Part 1 through Part 4.

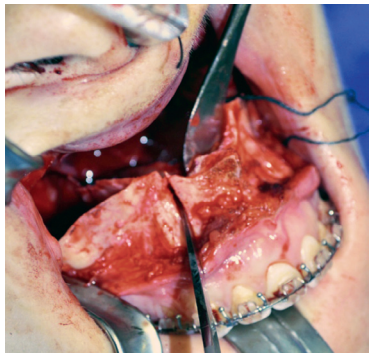


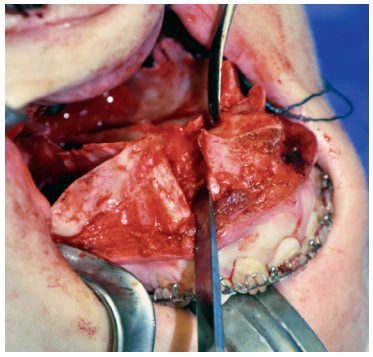




A5



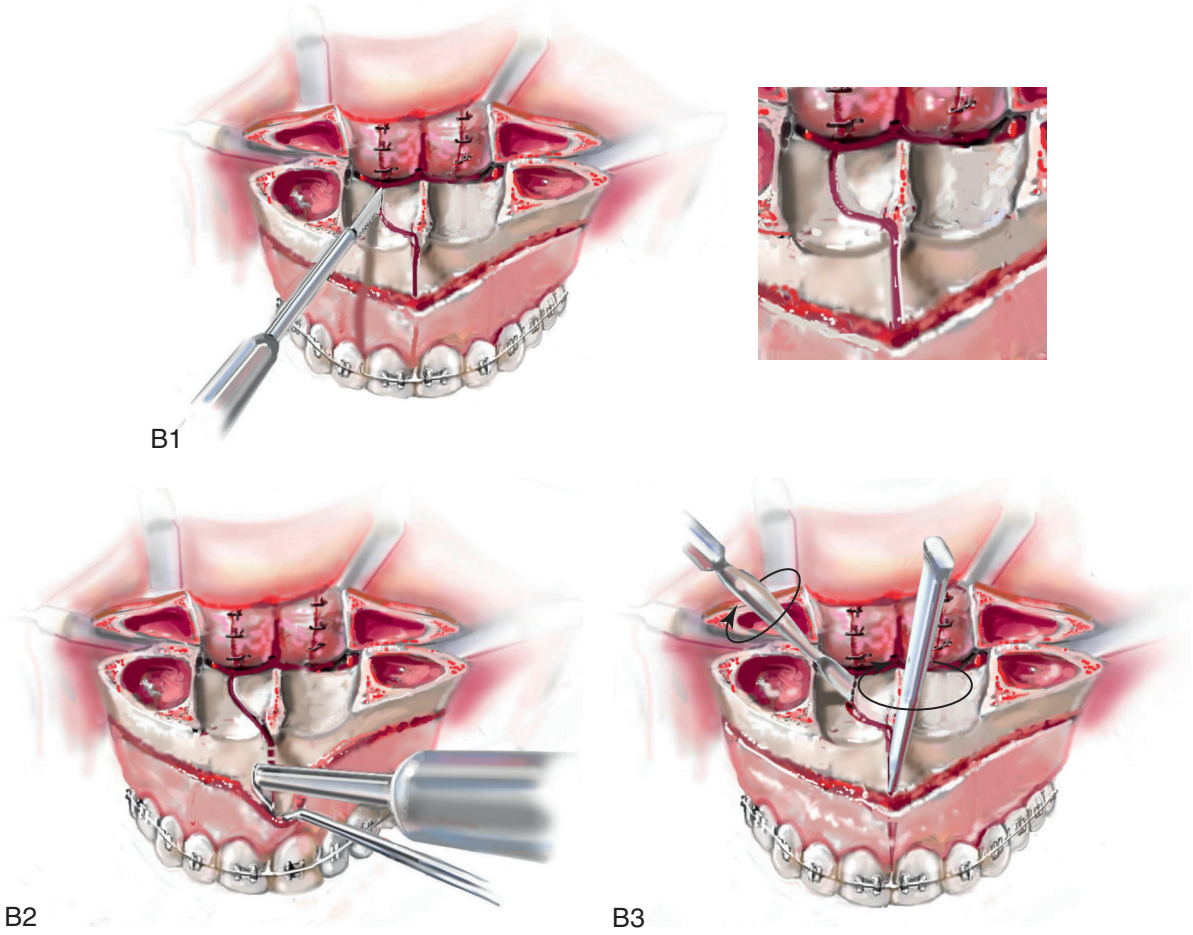




A6

• **Figure 15-22A, cont'd** Step 34, A, part 5, 6.

Continued



• **Figure 15-22B, cont'd** Step 34, B, Part 1 through Part 3.

the parasagittal palatal osteotomy) with a reciprocating saw may occur. The creation of a permanent palatonasal fistula or the compromise of the circulation to the maxillary segments should be an uncommon occurrence (see Chapter 16).

Two Segments

B. Description of a two-piece Le Fort I osteotomy with an interdental cut between the central incisors connected to parasagittal osteotomies through the hard palate:
   (1) The parasagittal palatal osteotomy is completed through the down-fracture using a reciprocating saw with a short, straight blade from the posterior edge of the hard palate anteriorly to the level of the incisive foramen (Fig. 15-22, *B-1*). If the planned posterior arch-width expansion is significant, then a second parasagittal osteotomy is also completed on the contralateral side.
   (2) The previously initiated interdental osteotomy is deepened using an oscillating saw with a longer, fan blade on a long shaft (Fig. 15-22, *B-2*).
   (3) A fine chisel (i.e., 5 mm or 7 mm in width) is placed into the interdental osteotomy, and an elevator is placed into the parasagittal osteotomy. Twisting both simultaneously assists with the splitting of the maxilla into the two segments (Fig. 15-22, *B-3*). Perforation through the palatal mucosa when completing the parasagittal palatal osteotomy may occur. The creation of a permanent palatonasal fistula or the compromise of the circulation to the maxillary segments should be an uncommon occurrence.

Maxillary Le Fort I Osteotomy: Continued
(Video 6: 6-10 and 7: 5-9)

Step 35. The maxillary segments are next secured into the prefabricated acrylic intermediate occlusal splint with the use of 26-gauge wire placed through interdental drill holes in the splint and around the orthodontic arch wire (Fig. 15-23).

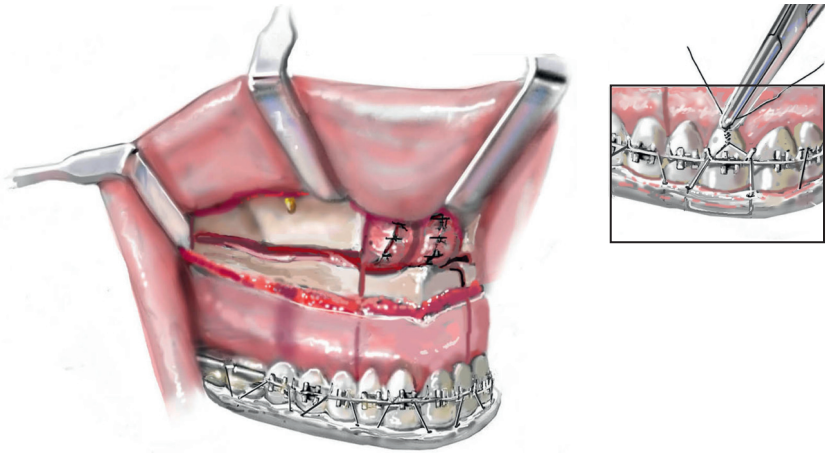
Step 36. The maxillary segments are secured to the mandible in the occlusion established by the "intermediate" splint with the use of 26-gauge wire loops passed around hooks located on the orthodontic arch wires (Fig. 15-24).

Step 37. With the condyles seated in the terminal hinge position (i.e., the same position that they were in when capturing centric relation in the office setting), the maxillomandibular complex is rotated to achieve the vertical dimension that was preoperatively determined (Fig. 15-25, *A*).

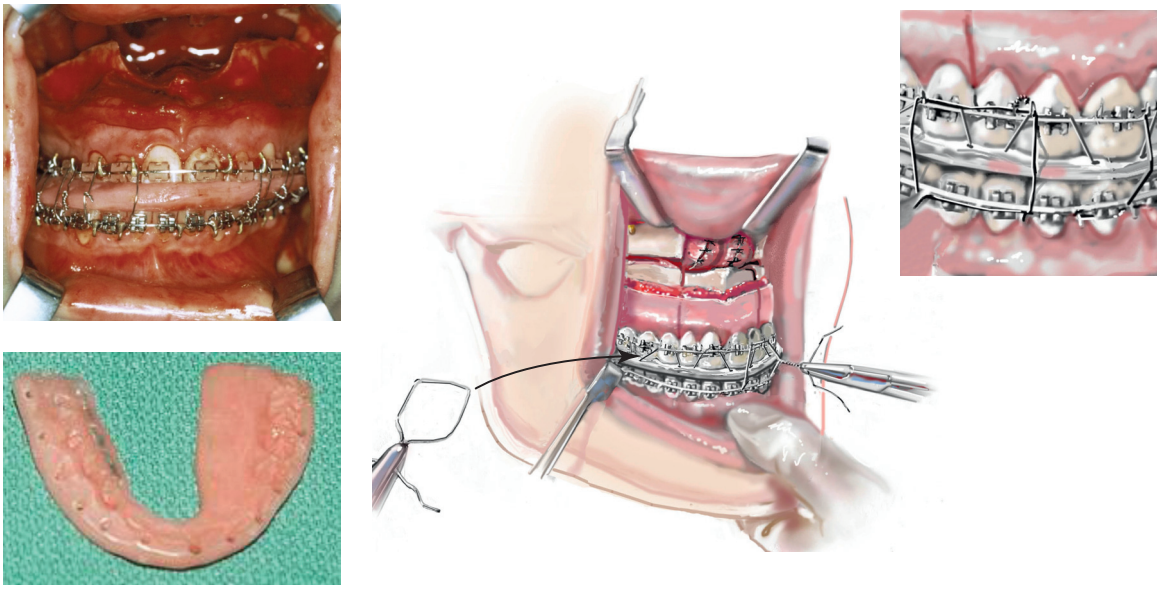
A. If vertical shortening (impaction) of the maxilla is planned, this will require the removal of bone interferences with rongeurs or using a rotary drill with a watermelon bur.

B. The Tessier caliper is used to measure the anterior vertical dimension (i.e., the anterior facial height) of the midface (i.e., the medial canthus to the mid-maxillary incisor on each side) to confirm that the desired vertical height is achieved (Fig. 15-25, *B*).

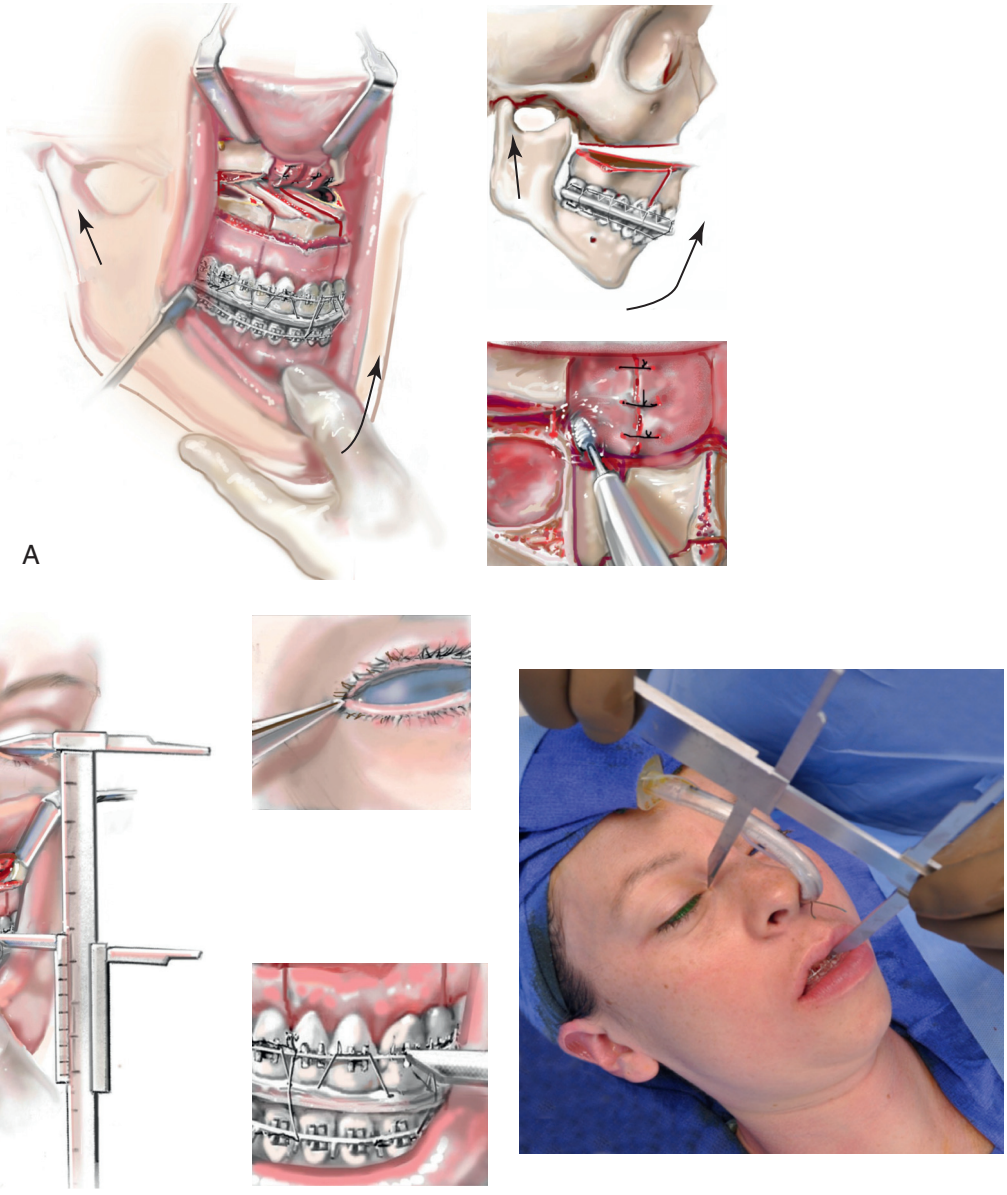
Step 38. With the condyles remaining in the terminal hinge position (i.e., the same position that they were in when capturing centric relation in the office setting) and with the maxilla in the preferred vertical location, appropriately sized and individually contoured L-shaped titanium plates (one by one) are most frequently placed across each zygomatic buttress and at each lateral nasal rim and secured with titanium screws (1.7 mm in diameter and 4 mm in length) (Fig. 15-26).



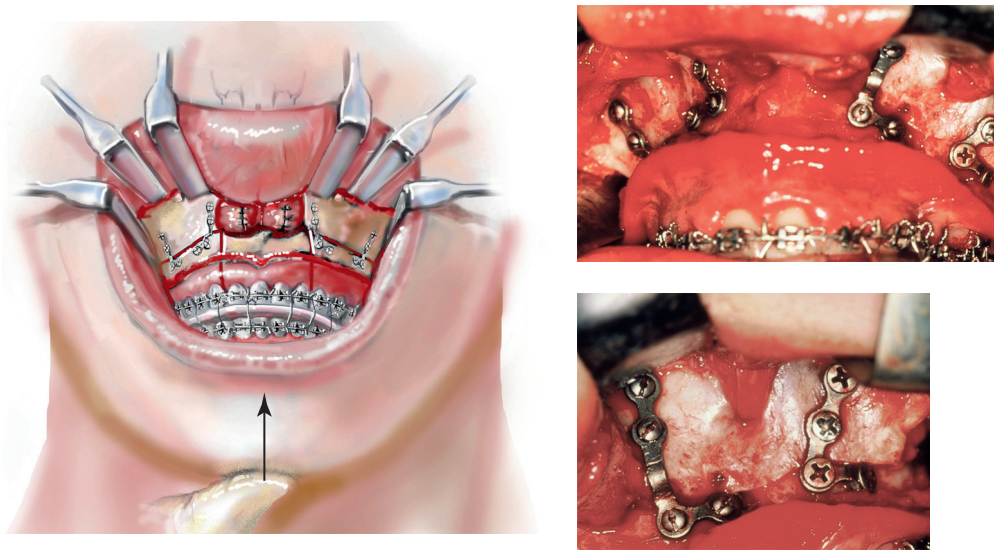
• **Figure 15-23** Step 35.



• **Figure 15-24** Step 36.



• **Figure 15-25** Step 37, A and B.

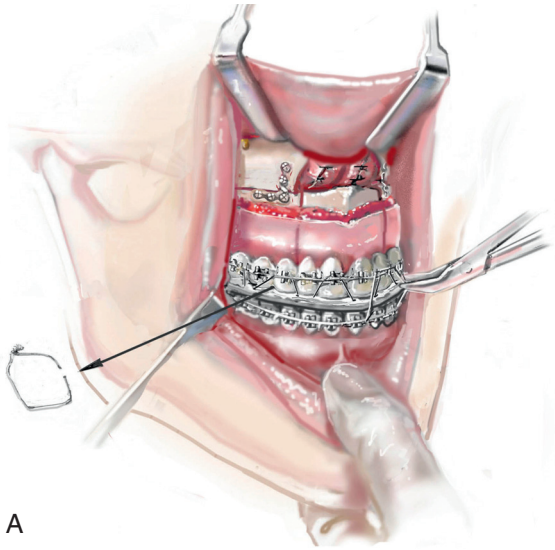


• **Figure 15-26** Step 38.

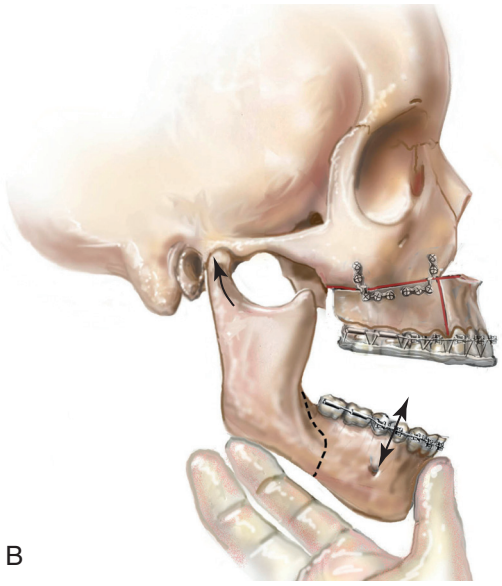
Step 39. A. The jaws are unwired (Fig. 15-27, *A*).
B. With the condyles seated in the terminal hinge position, the mandible is rotated vertically (up and down) to check the occlusion. The occlusion *must* be found to be even within the intermediate splint (Fig. 15-27, *B*).

Step 40. A. After the occlusion is determined to be even, the intermediate splint is removed.
B. The "final" splint is placed on the maxillary teeth and secured to the orthodontic wire (Fig. 15-28, *A*). This is accomplished with the use of 26-gauge wires passed through the interdental drill holes in the "final" splint and around the orthodontic arch wire (Fig. 15-28, *B*).

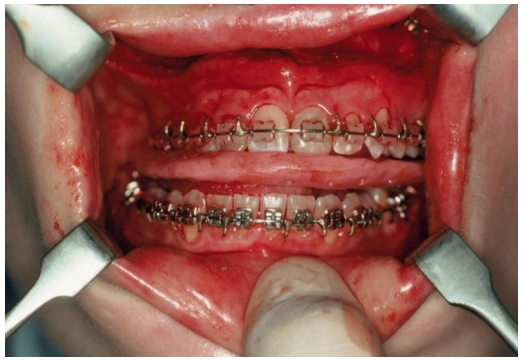
Step 41. When the extent of horizontal advancement and vertical lengthening of the maxilla are judged to be significant, then an interpositional bone graft may be beneficial for the achievement of reliable healing (Fig. 15-29, *A*). In these circumstances, I prefer to use autogenous or allogenic iliac bone graft. A bloc of corticocancellous bone is crafted and tightly wedged into the gap on each side. Each graft is inset in between the pyriform rim and the zygomatic buttress titanium plates (Fig. 15-29, *B*). An additional plate is then contoured to extend from the anterior maxilla above, across the graft, and then onto the alveolar process of the anterior maxilla. Titanium screws (1.2 mm in diameter and 3 mm in length). then secure the plate to the bone (Fig. 15-29, *C*).



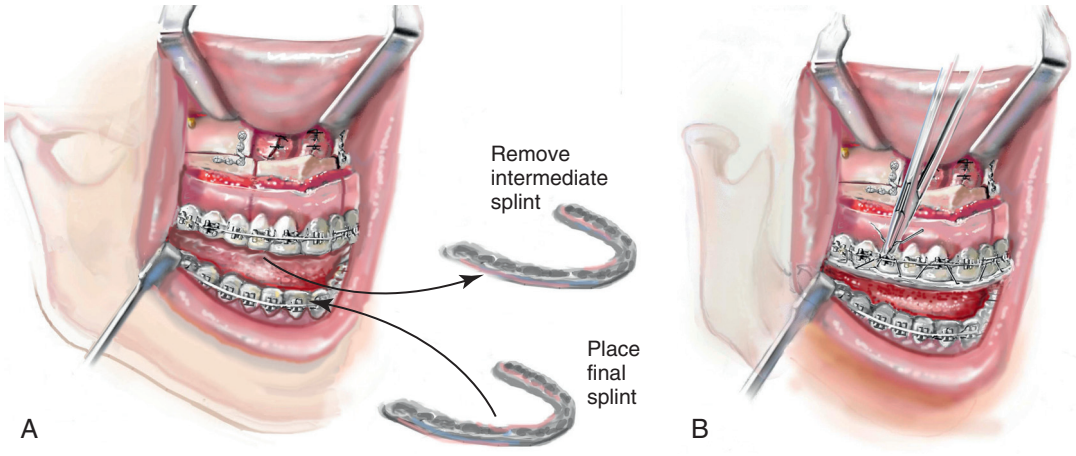
A



B



• **Figure 15-27** Step 39, A and B.



• **Figure 15-28** Step 40, A and B.

Pyriform Rim, Nasal Floor, and Anterior Nasal Spine Recontouring (Video 6:9 and 7:7)

Step 42. The nasal aperture, the floor of the nose, and the anterior nasal spine are inspected. Refinement and recontouring of these structures using a rotary drill with a watermelon bur are frequently beneficial (Fig. 15-30). For example, in the patient with vertical maxillary excess (i.e., long face growth pattern), the deepening and recontouring of the nasal floor, the pyriform rims, and the anterior nasal spine will generally improve nasal airflow (i.e., the correction of a tight nasal inlet and an elevated nasal floor) and also enhance nasal aesthetics (i.e., limit unwanted alar base widening and tip elevation).

Chin Osteotomy (Video 6:11 and 7:10)

Step 43. Attention is next turned to the facial vestibule of the chin. The lower lip is stretched outward to allow for exposure of the vestibule and the visualization of the mental nerve through the mucosa on each side. In the depth of the vestibule, an incision is made with the use of a Bovie electrocautery device or a knife (#15 blade) through the mucosa from cuspid region to cuspid region, stopping just short of the visualized mental nerve on each side (Fig. 15-31). The center two thirds of the incision are next extended down to bone. A full cuff of mucosa and muscle is maintained adjacent to the attached gingiva of the anterior teeth. This should allow for adequate layered wound closure (i.e., involving muscle and mucosa) without resulting periodontal sequelae.

Step 44. Dissection with an elevator exposes the anterior surface of the chin (not completely to the inferior border of the central chin). The dissection continues laterally and remains inferior to the mental nerve, with lateral exposure to the inferior border of the mandible. There is no need to dissect above the mental nerve on either side. If a 360-degree dissection around the nerve is completed, it is more likely that the nerve will be excessively stretched or avulsed.

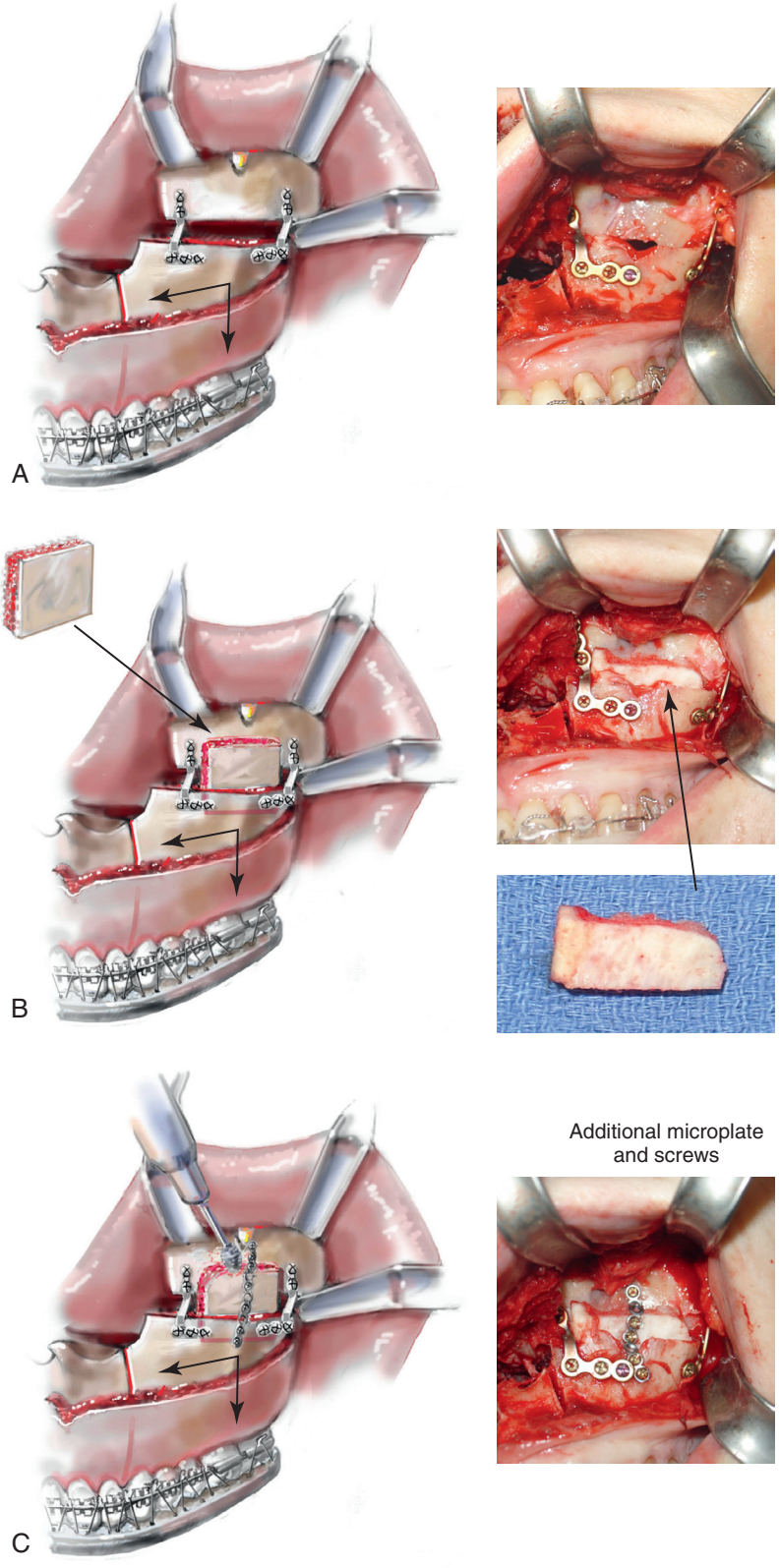
Step 45. A small S-shaped Tessier chin retractor is placed on each side inferior to the mental nerve and laterally to the inferior border. A sterile pencil is used to mark the location of the oblique osteotomy (Fig. 15-32); this should be planned sufficiently below the dental roots and the mental foramen on each side. The exact location of the osteotomy will depend on the presenting chin morphology and the planned reconstruction (i.e., vertical reduction or lengthening and extent of horizontal advancement).

Step 46. Using an oscillating saw with a short fan blade on a long shaft, a vertical groove is made in the midline across and perpendicular to the proposed horizontal osteotomy (Fig. 15-33, *A*). This will help to maintain vertical orientation after the osteotomy and before fixation.

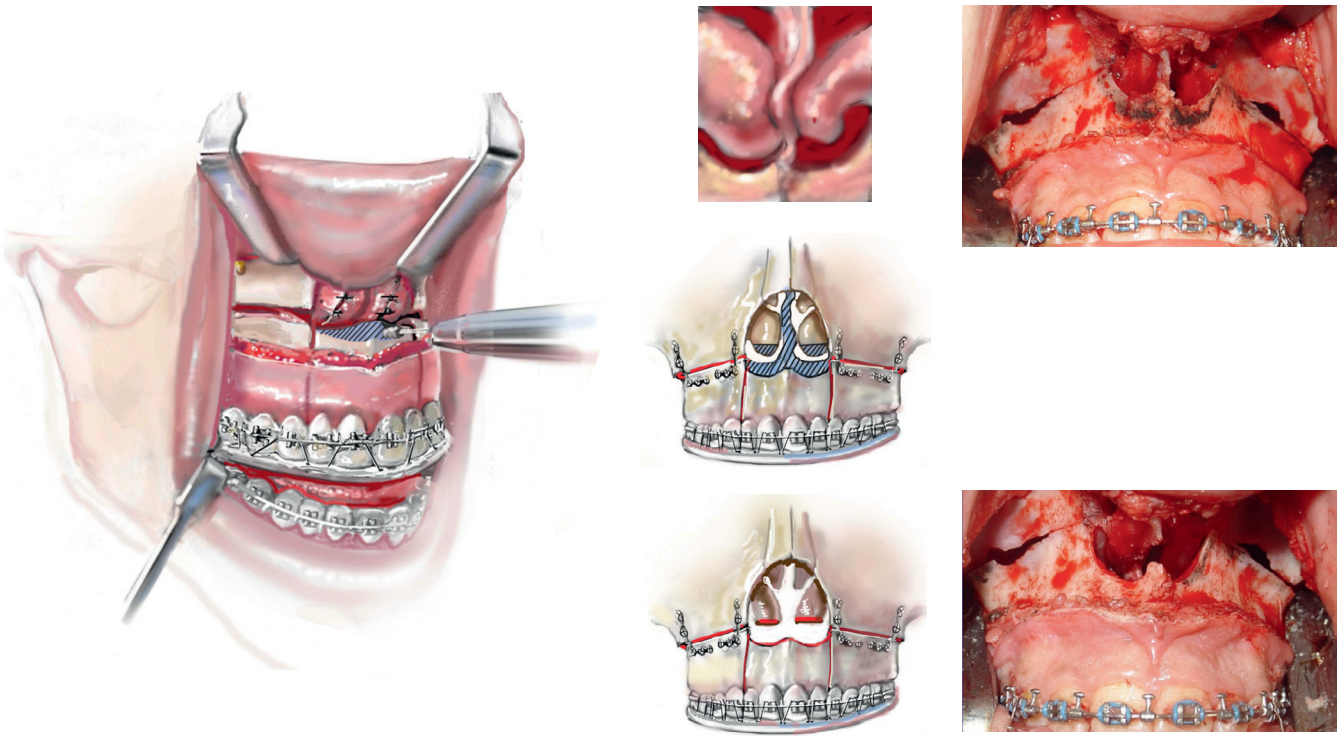
Step 47. A drill hole is placed in the midline within the distal chin (Fig. 15-33, *B*). Later, a screw (1.7 mm in diameter and 8 mm in length) will be partially inserted. The screw is not placed in the distal chin until the osteotomy is completed. It will then be held with a wire twister and used as a retractor and a holder to facilitate the repositioning and orientation of the distal chin unit.

Step 48. The oblique osteotomy of the chin is initiated in the central portion using an oscillating saw with a wide, fan blade on a short shaft (Fig. 15-34). The use of this saw and blade helps to maintain orientation and limit a cant in the osteotomy.

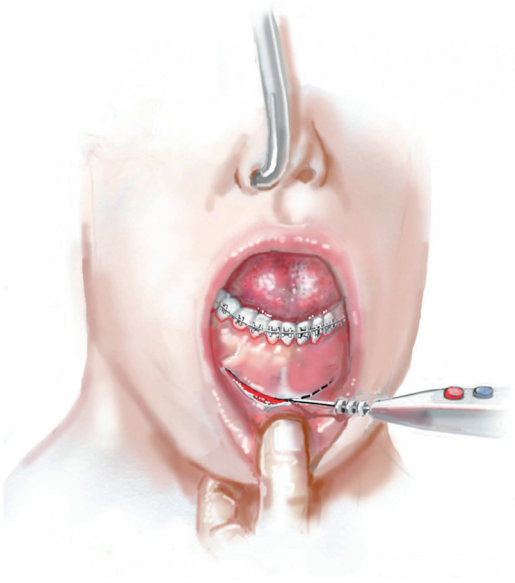
Step 49. The reciprocating saw with a short blade and then with a longer blade is used to complete the lateral aspects of the osteotomy and to go through the



• **Figure 15-29** Step 41.



• **Figure 15-30** Step 42.



• **Figure 15-31** Step 43.

depth of bone on each side (Fig. 15-35). An osteotome may be inserted with a twisting motion to complete the osteotomy separation.

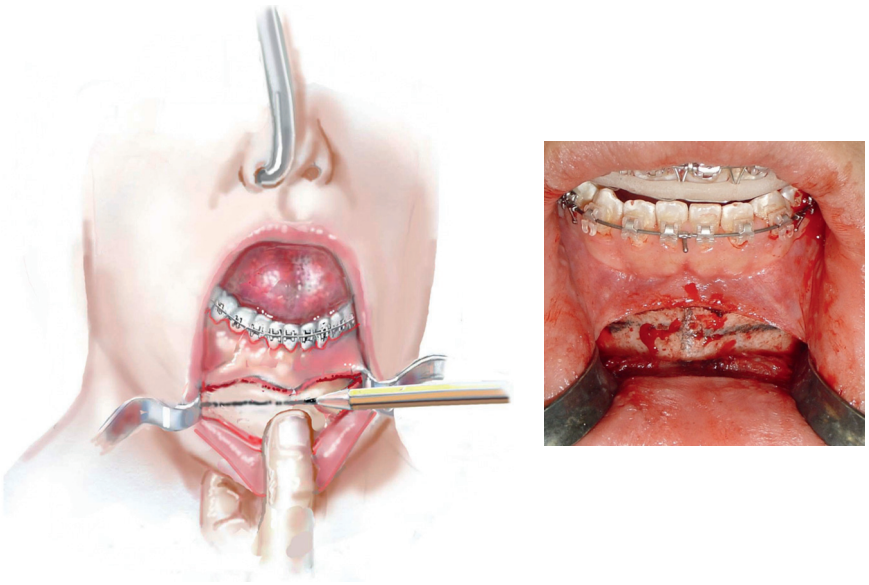
Step 50. When vertical shortening of the chin is planned, there are two options.

A. If only limited shortening is required in combination with horizontal advancement, it may be preferable to use a rotary drill with a watermelon bur to remove bone height at the osteotomy line.

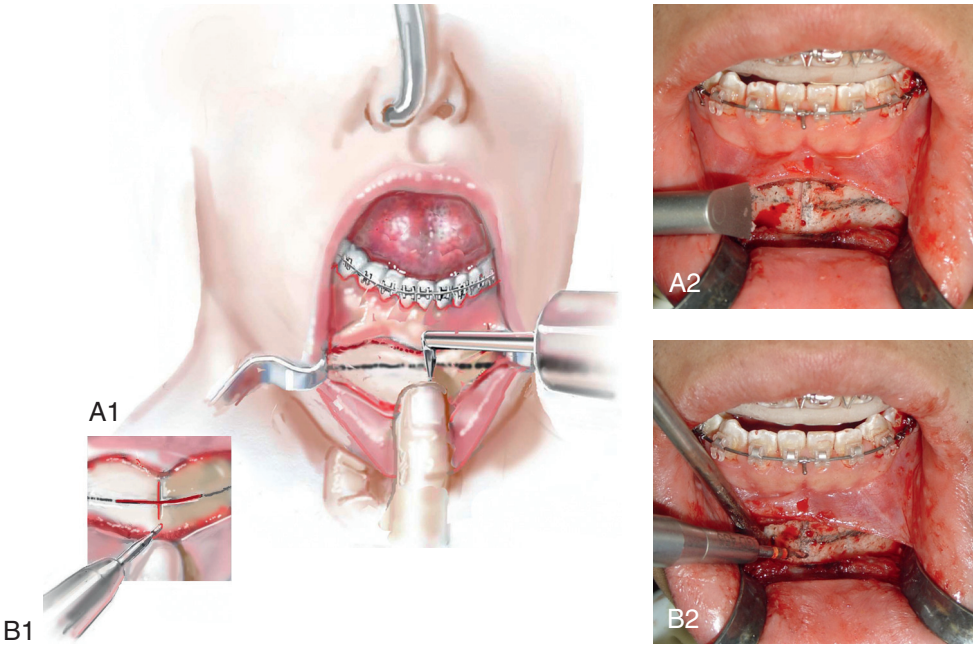
B. If a more significant vertical reduction is planned, a second parallel osteotomy for ostectomy is completed through the proximal chin (below the dental roots) using a reciprocating saw with a long straight blade. Any lateral irregularities are also removed using a rotary drill with a watermelon bur. This is to limit postoperative palpable or visible step-offs.

Step 51. The distal chin is held in the desired position by the assistant with the use of the wire twister secured to the positioning screw. The surgeon custom contours each titanium plate across the osteotomy site and then secures each plate with screws (1.7 mm in diameter and 4 mm in length). Typically, either a three- or four-hole straight plate is contoured with the ends placed on either side of the osteotomy. The superior screw is usually placed in between the lateral incisor and the canine on each side. With the chin secured in its new location, the positioning screw is removed (Fig. 15-36, *A* and *B*).

Step 52. If significant vertical lengthening is carried out, an interpositional graft (i.e., autograft, allograft, or block hydroxyapatite) is crafted and placed in the gap (Fig. 15-37). The graft fills the central gap in between the fixation plates. I do not find it necessary to place graft in the lateral aspect of the gaps.



• **Figure 15-32** Step 45.



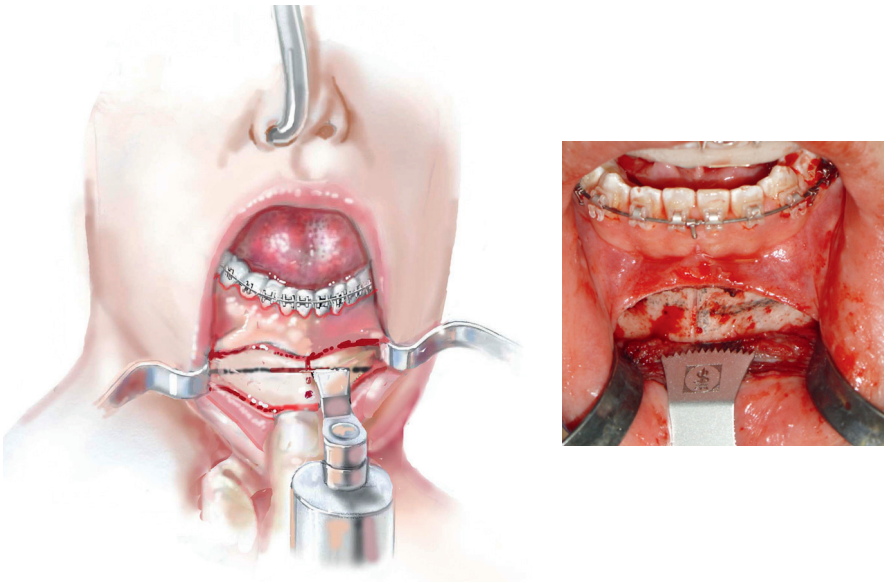
• **Figure 15-33** Steps 46 and 47.

An additional plate with screws is generally placed vertically in the midline across the osteotomy site directly over the graft (see Chapter 18).

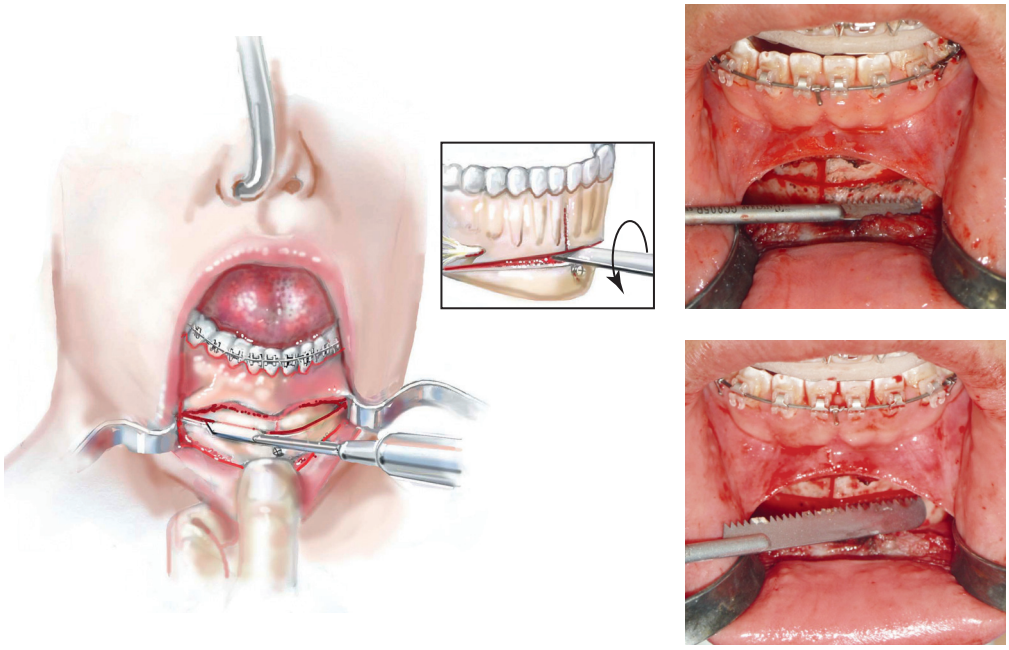
Sagittal Split Ramus Osteotomies: Continued (Video 6:12 and 7:11)

Step 53. Attention is returned to one side of the ramus of the mandible. The previously placed packs are removed and retractors (i.e., tongue, inferior border at buccal extension, toed-in Langenbeck at anterior ramus to retract the cheek, and medium malleable to expose the medial cut) are placed for sufficient exposure to complete the sagittal splitting of the ramus of the mandible.

Step 54. Initially, fine osteotomes (i.e., 5 mm and 7 mm in width) are used with a mallet to confirm that all cortical cuts are complete (Fig. 15-38). When twisting the chisels, a degree of movement should be visualized along the vertical cut down to the inferior border. Care is taken to avoid injury to the inferior alveolar nerve, which is located superficial to the cortex along the inferior aspect of the buccal shelf extension.



• **Figure 15-34** Step 48.

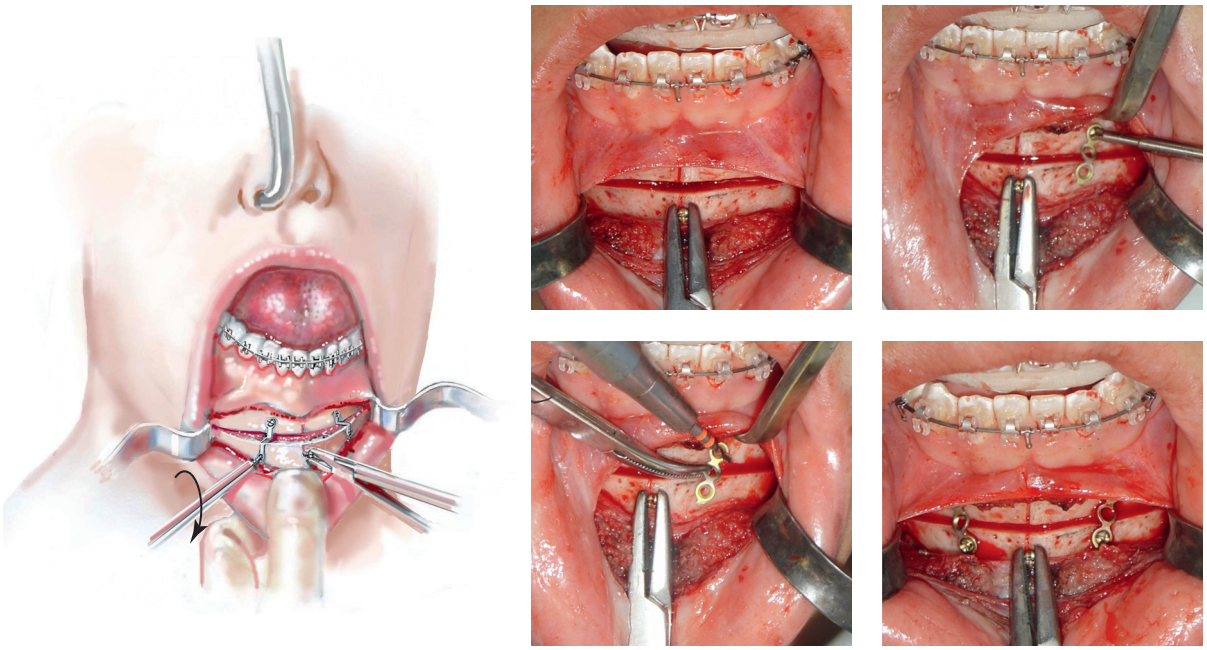


• **Figure 15-35** Step 49.

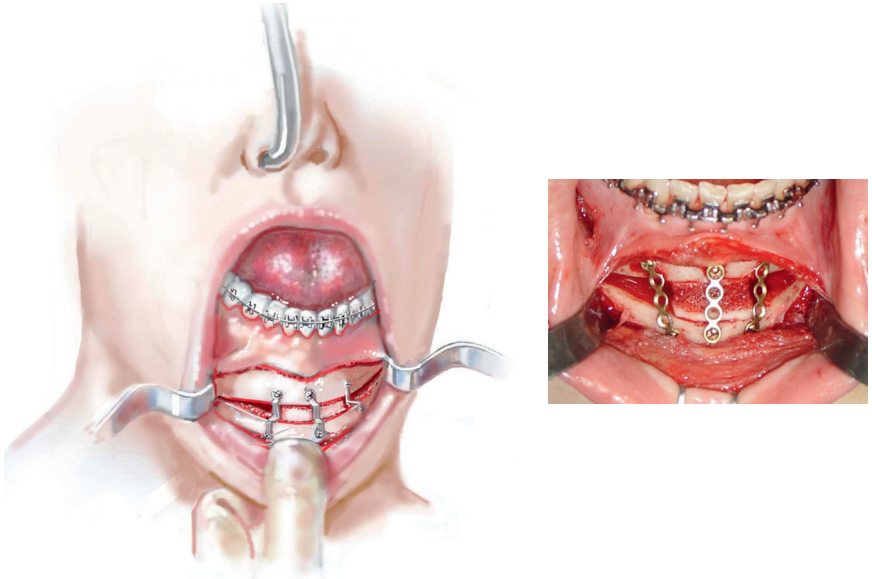
Step 55. A larger osteotome (10 mm in width) is then placed deep into the medullary cavity near the second molar of the mandible. A sagittal split spreader forceps is placed deep into the medullary cavity (i.e., more distal) in the buccal extension region (Fig. 15-39). After both of these are in place, a simultaneous twisting and spreading motion is employed to complete the separation. During separation, the neurovascular bundle is visualized, and the correct separation of the condyle (retained to the proximal segment) is confirmed.

Step 56. With the split complete, further freeing of the neurovascular bundle from the proximal segment is sometimes desirable. A thin periosteal elevator may be used to gently tease the neurovascular bundle free from the proximal segment. A rotary drill with a watermelon bur may also be used to release the neurovascular bundle from the proximal segment.

NOTE: It is not usually necessary to completely free the nerve from the proximal segment. Caution is warranted, because a degree of contusion to the neurovascular bundle will occur when doing so.



• **Figure 15-36** Step 51.



• **Figure 15-37** Step 52.

Step 57. A curved Kocher clamp is placed on the distal aspect of the proximal segment. The clamp is used as a handle to apply force and to ensure the complete separation and freedom of motion of the proximal and distal segments (i.e., soft-tissue release).

Step 58. The identical sagittal splitting of the ramus of the mandible is completed on the contralateral side; see Steps 51 through 57.

Removal of Impacted Mandibular Wisdom Teeth (Video 6:12)

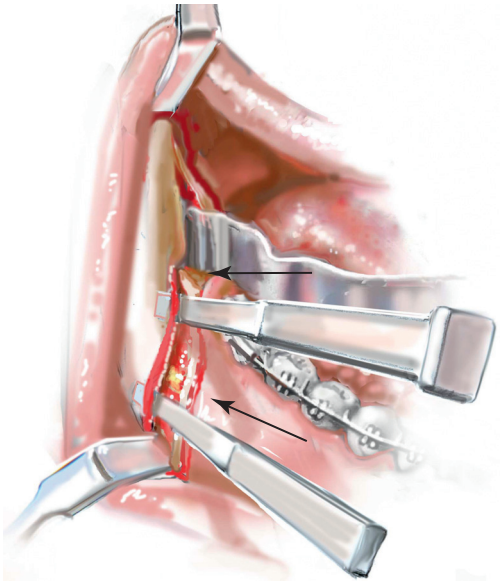
Step 59. If present, each bony impacted mandibular wisdom tooth located within the distal segment is removed with a dental elevator (Fig. 15-40). It may be helpful to use a rotary drill with a tapered fissure bur to remove bone adjacent to the tooth. Sectioning the tooth may be required to facilitate

the atraumatic removal of the tooth. This will minimize the chance for an uncontrolled fracture of the lingual plate.

Sagittal Split Ramus Osteotomies: Continued
(Video 6: 12-15 and 7: 11-14)

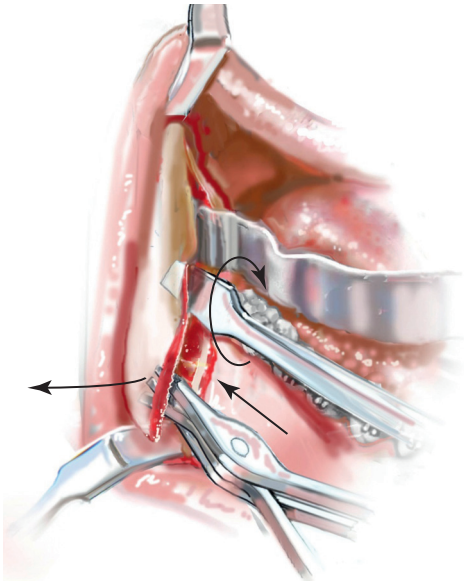
Step 60. The distal mandible is placed into occlusion through the prefabricated final splint, which has already been secured to the maxilla. The jaws are wired together with 26-gauge wire loops placed around the orthodontic interdental hooks (Fig. 15-41).

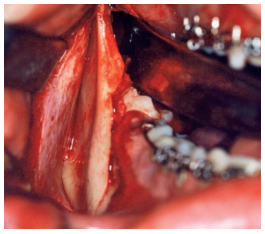
Step 61. Attention is turned to the proximal segment of the mandible on one side. The proximal segment is seated with the condyle in the terminal hinge position. This is accomplished with the placement of a fine curved hemostat to the edge of the buccal extension. The condyle will then rest in a superior and anterior position within the glenoid fossa (i.e., the same position that it was in when capturing centric relation in the office setting). Any interferences that prevent sufficient passive bone contact of the proximal and distal segments along the osteotomy site are managed using a rotary drill with a watermelon bur (Fig. 15-42). If the mandible is set back, an appropriate section of bone is also removed from the distal aspect of the proximal segment (i.e., the buccal shelf) to prevent any cortex overlap. When setting the mandible back, the removal of bone interferences from the lingual plate of the distal segment is also generally required. These tasks are accomplished using either a rotary drill with a watermelon bur or a reciprocating saw with a short, straight blade.

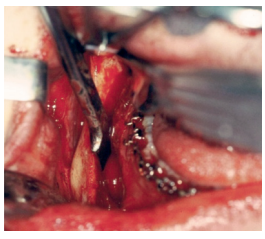


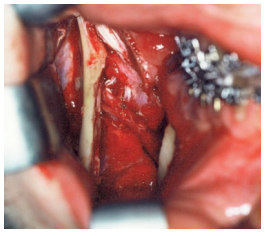
• **Figure 15-38** Step 54.

NOTE: The hemostat is not used to clamp the proximal and distal segments together. Gentle superior and posterior force is applied to seat the condyle in the terminal hinge position.



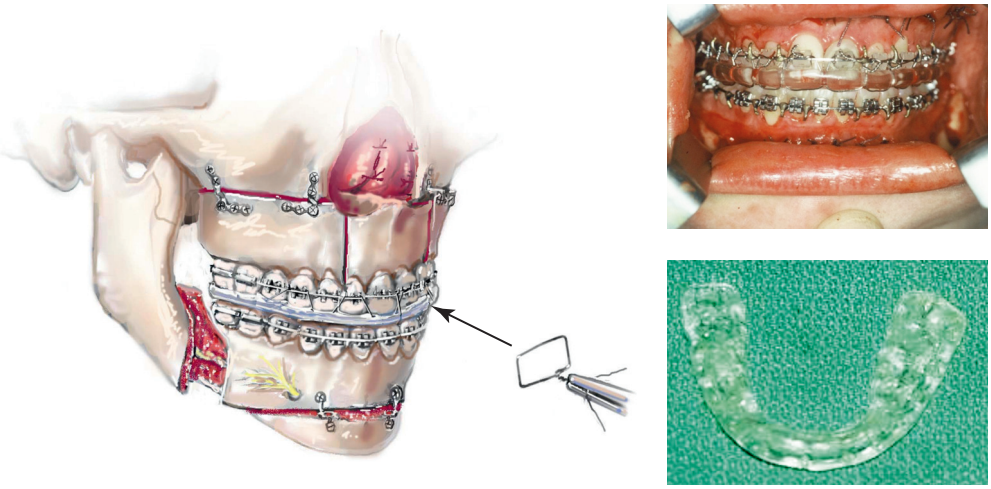




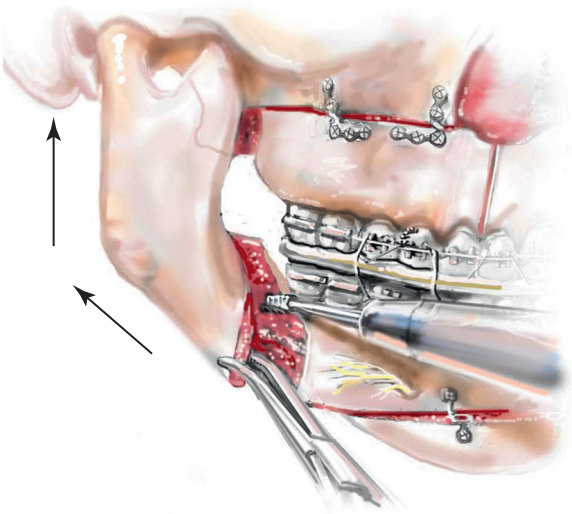


• **Figure 15-39** Step 55.

• **Figure 15-40** Step 59.



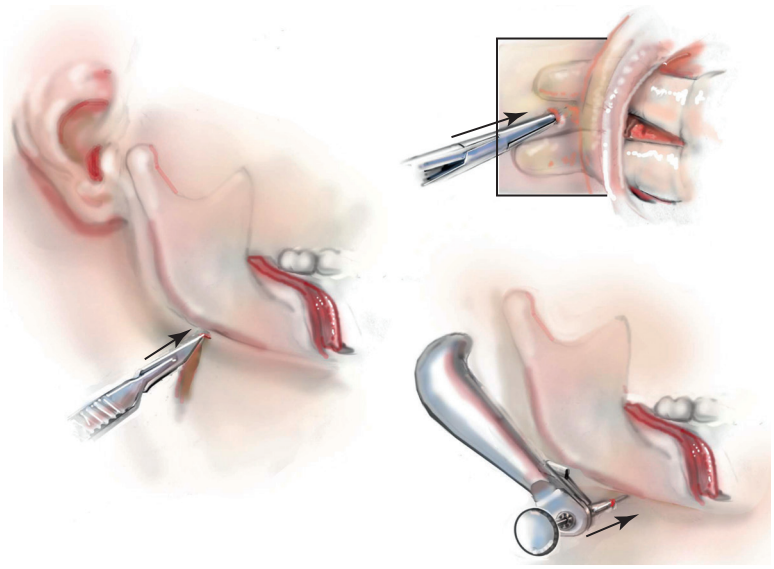
• **Figure 15-41** Step 60.



• **Figure 15-42** Step 61.

Step 62. A 4-mm lateral neck incision (just below inferior border of the mandible) is made through the skin for trocar insertion (Fig. 15-43, *A* through *C*). The location of the incision is based on the best position of the trocar for ideal bicortical screw placement. With the use of a straight hemostat, blunt dissection through the skin incision is accomplished to achieve intraoral exposure. The trocar is then introduced, and the cheek retractor component of the trochar is placed to improve visualization.

Step 63. The proximal segment is seated with the condyle in the terminal hinge position. This is accomplished via the placement of a fine curved hemostat to the edge of the buccal extension. (Note that the hemostat is not used to clamp the proximal and distal segments together.) Gentle superior and posterior force is applied to seat the condyle in the

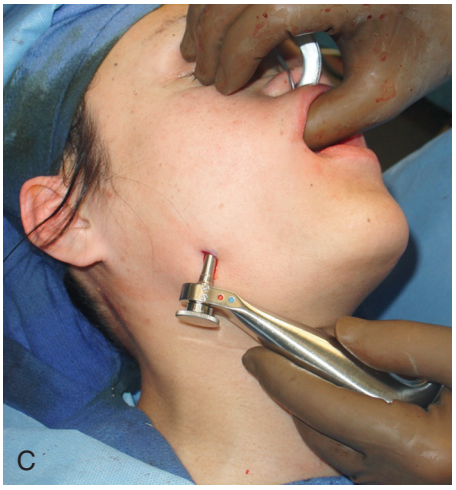


A

B

4-mm skin incision

Insertion of trocar



C

Trocar inserted

Trocar cheek retractor in place

• **Figure 15-43** Step 62.

terminal hinge position. The condyle will then rest in a superior and anterior position within the glenoid fossa (i.e., the same position that it was in when capturing centric relation in the office setting). Three bicortical titanium screws (2.3 mm in diameter and 14 mm to 16 mm in length) are generally sufficient to passively secure the proximal and distal segments together (Fig. 15-44, *A* through *C*). The bicortical screws are placed in locations where bone contact is the best. A fourth screw is sometimes placed. Stability is further improved when the screws are positioned as far from each other as possible. Plates with unicortical screws are occasionally preferred to bicortical screw fixation.

Step 64. Attention is turned to the contralateral side, and identical procedures are carried out; see Steps 60 through 63.

Step 65. The jaws are then unwired. With the application of posterior and superior vectored force to the anterior mandible, the condyles are seated in the terminal hinge position. The mandible is then rotated (up and down) to check that the occlusion is even and passive into the splint (Fig. 15-45). If not, the bicortical fixation screws are removed, and the process is repeated; see Steps 59 through 63.

Wound Closure (Video 6: 16 and 7: 15)

The anesthesiologist is asked to discontinue the deliberate hypotensive anesthesia before wound closure. This should unmask any significant bleeding vessels.

Step 66. The maxillary, mandibular, and chin vestibular wounds are irrigated, suctioned, and checked for hemostasis. The wounds are then closed with interrupted suture (3-0 Vicryl and 3-0 chromic).

A. The mandibular vestibular wounds are generally closed in one layer with interrupted suture (3-0 Vicryl). Running and locking (3-0 chromic) suture may be substituted (Fig. 15-46, *A*). A watertight closure is important to prevent dehiscence followed by increased risk of infection.

B. The maxillary wound is generally closed with running and locking suture (3-0 chromic) (Fig. 15-46, *B*). The risk of dehiscence or infection is low.

C. The wound of the chin is closed in two layers (i.e., muscle and mucosa). Three to five interrupted buried sutures (3-0 Vicryl) are placed for muscle layer closure. Locking and running sutures (3-0 chromic) are used for the closure of the mucosa layer (Fig. 15-46, *C*). A two-layer closure is essential to limit dehiscence and to prevent chin ptosis.

D. The trocar (skin) wounds are closed with two interrupted sutures (6-0 nylon).

Step 67. The mouth is irrigated and suctioned, and the throat pack is removed. An orogastric tube is placed to drain the stomach and then removed. Interdental elastics are applied to secure the occlusion.

Anterior Neck Soft-Tissue Procedures (Video 7:16)

Step 68. If anterior neck soft-tissue management is planned, it is carried out at this stage. This may include cervical flap elevation, supra-platysma and sub-platysma defatting, and vertical platysma muscle plication (see Chapter 40). Additional prepping with povidone–iodine (Betadine) solution is carried out, and a sterile drape is placed over the standard drapes that are already in place. Sterile instruments and a fresh gown and gloves are used. Injection with tumescent solution is completed in the supra-platysma plane.

A. A 3-cm submental incision is made within or just caudal to a natural submental crease.

B. Two double skin hooks are placed (i.e., one on either side of the incision) to provide tension on the flaps.

C. If the separation of the cervical skin from the underlying platysma muscle is planned, it is accomplished with a blunt-tipped scissors. The cervical skin flap elevation off of the platysma muscle may continue laterally over the sternocleidomastoid muscles inferiorly to the thyroid cartilage and superiorly to the inferior border of the mandible.

D. A cannula (no. #7 plastic curved) is used to suction the fat above and in between the platysma muscles.

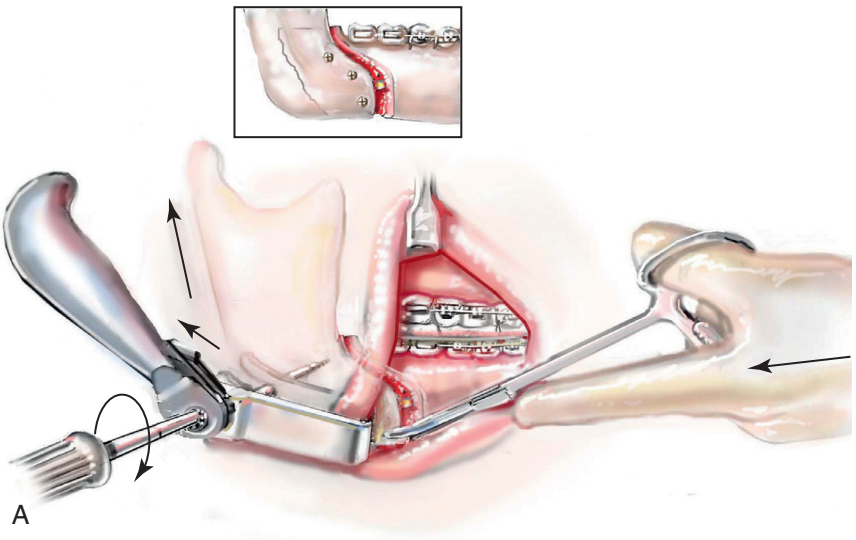
E. Plication of the platysma muscle in the midline (vertically) from above the thyroid cartilage to just below the chin may also be carried out with interrupted suture ties (3-0 Vicryl).

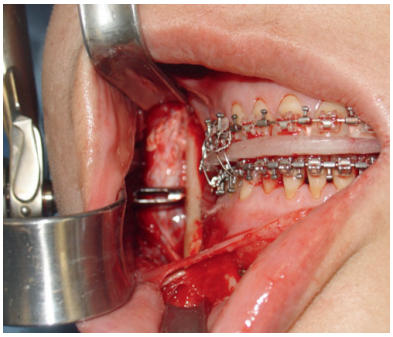
F. After confirming satisfactory hemostasis, the submental skin wound is closed in two layers: dermal (5-0 Vicryl) and skin (6-0 nylon).

G. A light pressure dressing (Jobst type) is generally applied to the neck. In general, I do not find it necessary or useful to place a drain.

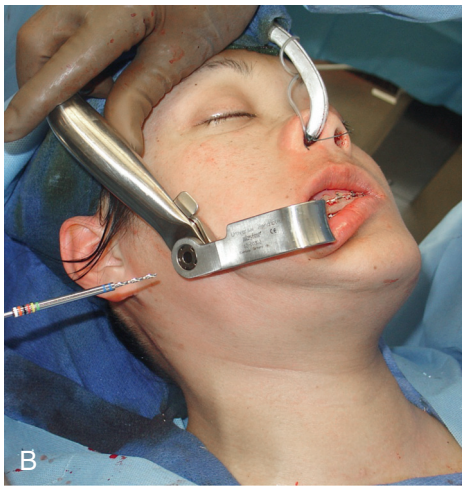
Emergence from Anesthesia

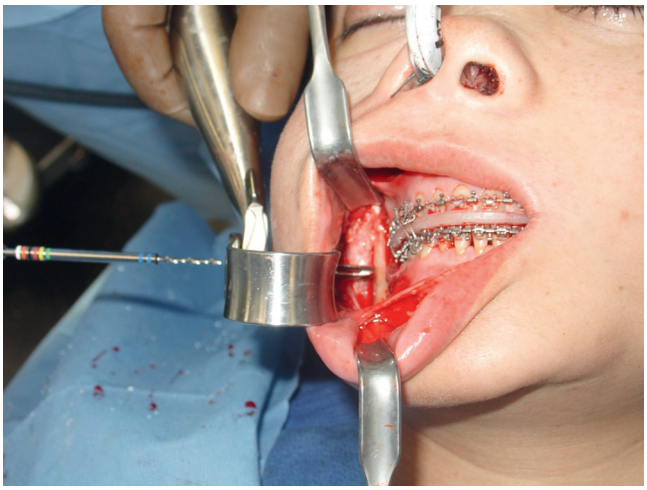
Step 69. The corneal shields or temporary tarsorrhaphies, if used, are removed. The sutures used to secure the endotracheal tube to the scalp and the septum of the nose are released. When the appropriate



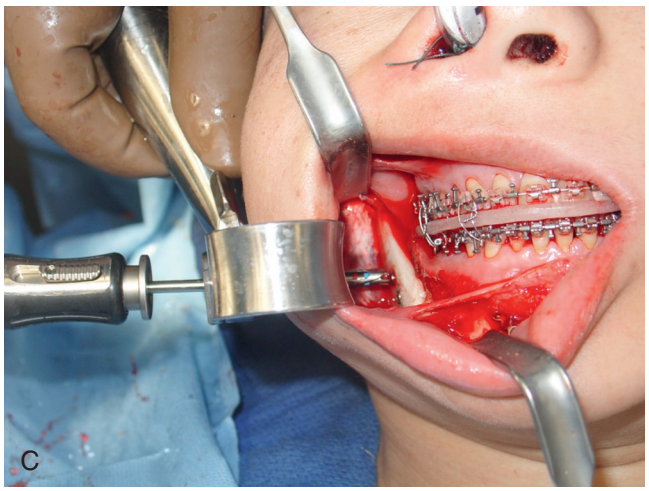


Intraoral view with trocar in place

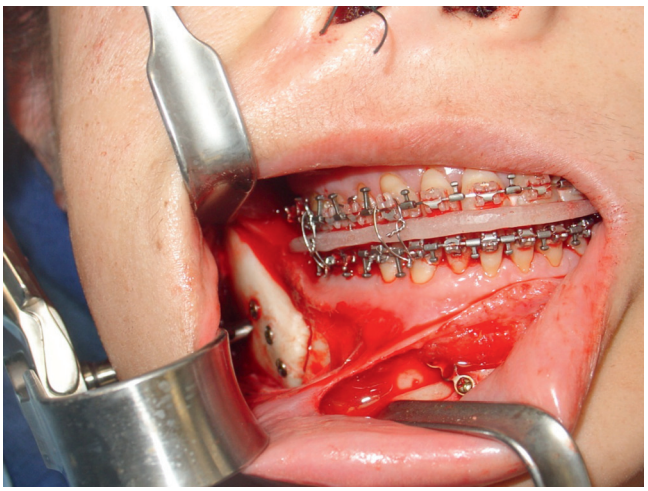




Power drill (2.3-mm bit) inserted through trocar

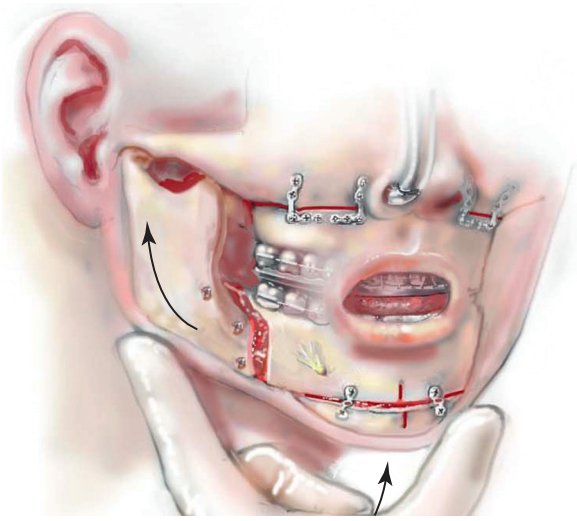


Screwdriver inserted through trocar



3 bicortical screws (2.3 mm in diameter and 14-16mm in length) in place

• **Figure 15-44** Step 63.

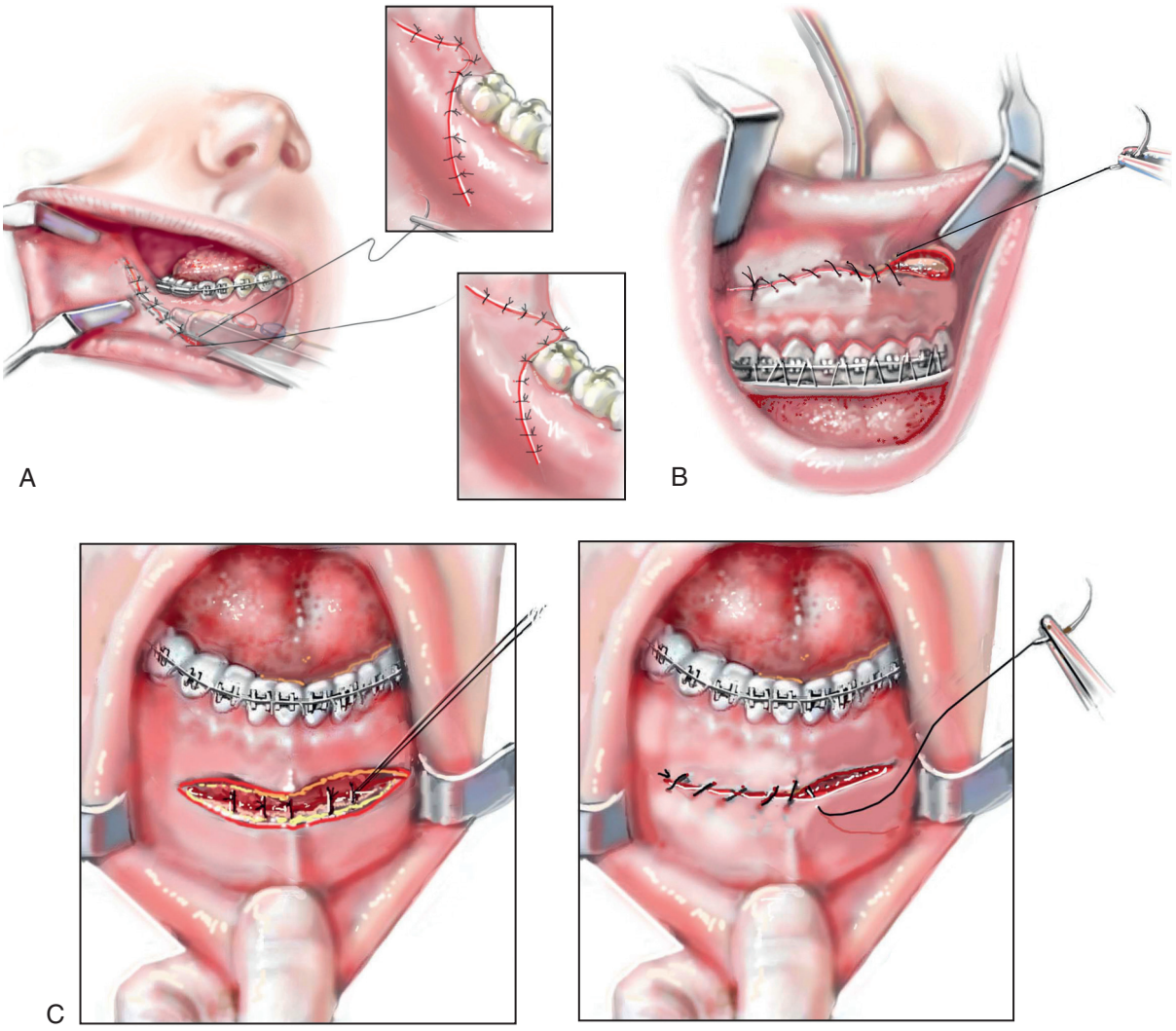


• **Figure 15-45** Step 65.

physiologic parameters are met, the patient is generally extubated in the operating room and then taken to the recovery room breathing spontaneously. If any airway need arises, the interarch elastics are removed without concern (see Chapter 11).

Conclusions

The surgical management of dentofacial deformities has gradually evolved. By understanding the technical details for the safe execution of the maxillary (Le Fort I), mandibular (sagittal splitting of the ramus), and chin (osseous genioplasty) osteotomies, each skeletal unit may be precisely and efficiently repositioned (i.e., in three dimensions) during one operation. Other simultaneous adjunctive procedures (i.e., septoplasty; the reduction of the inferior turbinates;



• **Figure 15-46** Step 66, A, B, and C.

the recontouring of the pyriform rims, the nasal base, and the anterior nasal spine; the removal of wisdom teeth; and soft-tissue neck rejuvenation) are routinely performed, when indicated, to improve head and neck function (i.e. occlusion, chewing, speech articulation, swallowing, breathing, and lip closure) and to enhance facial aesthetics. Special circumstances and unusual anatomy may require variations of the techniques described in this chapter. The development of a consistent approach for most routine cases limits morbidity and increases efficiency of care.

References

1. Luhr HG: Zur stabilen osteosynthese bei unterkiefer–frakturen. *Dtsch Zahnarztl Z* 23:754, 1968.
2. Posnick JC, Fantuzzo JJ, Troost T: Simultaneous intranasal procedures to improve chronic obstructive nasal breathing in patients undergoing maxillary (Le Fort I) osteotomy. *J Oral Maxillofac Surg* 65:2273–2281, 2007.
3. Posnick JC: Le Fort I, sagittal split and genioplasty: historical perspective and step-by-step approach. In Posnick JC, editor: *Craniofacial and maxillofacial surgery in children and young adults*, Philadelphia, 2000, W.B. Saunders Company, pp 1081–1102.
4. Obwegeser HL. In Trauner R, Obwegeser H, editors: Zur operationstechnik bei der progenie und anderen unterkieferanomalien. *Dtsch Zahn- Mund- u Kieferheilk* 23:1–26, 1955.
5. Obwegeser HL: The surgical correction of mandibular prognathism and retrognathia with consideration of genioplasty. I. Surgical procedures to correct mandibular prognathism and reshaping of the chin. *Oral Surg Oral Med Oral Pathol* 10:677–689, 1957.
6. Obwegeser HL: Der offene biss in chirurgischer sicht. *Schweiz Monatschr Zahnhlkd* 74:668–687, 1964.

16

Complications Associated with Orthognathic Surgery

JEFFREY C. POSNICK, DMD, MD

- General Considerations
- Risks and Complications Specific to Sagittal Ramus Osteotomy of the Mandible
- Risks and Complications Specific to Le Fort I Osteotomy
- Risks and Complications Specific to Oblique Osteotomy of the Chin
- Conclusions
- Patient Education Materials

General Considerations

No matter how accurate the diagnosis and how meticulous the surgeon, complications will occur after orthognathic procedures.* Alternatively, the relative safety of orthognathic procedures has been confirmed by a number of retrospective reviews.[13,32,84,93,152,223,245,254,315,353] Panula and colleagues reviewed maxillary and mandibular osteotomy patients at their institution (N = 655) and found only one serious complication (intraoperative bleed). A prospective study of Le Fort I operations by Kramer and colleagues (N = 1000) found a 6.4% incidence of complications that ranged from extensive bleeding (1.1%) to sensory loss in the distribution of the infraorbital nerve.[173] A review by Chow and colleagues of a consecutive series of orthognathic patients (N = 1294) documented a relatively high complication rate (9.7%)[61]; this included mostly manageable "postoperative infections" (7.4%), with no serious or rare events recorded. A large series of orthognathic patients (N = 2049) was reviewed by Van de Perre and colleagues[346]; those authors identified one patient death and 44 other patients with either primary or secondary complications.

*References 2, 23, 25-27, 42, 53, 78, 80, 83, 118, 146, 150, 157, 162, 186, 203, 206, 212, 224, 242-244, 246, 252, 262, 266, 314, 322, 328, 334, 335, 341-343, 345, 347, 355, 362-364.

The definition of what constitutes a complication in orthognathic surgery varies between both researchers and clinicians, thereby making interstudy comparison difficult. By understanding the medical history and physical findings of the individual who presents with a dentofacial deformity including 1) an awareness of any associated conditions or malformations 2) the patient's baseline psychosocial health and 3) any head and neck dysfunctions—patient-specific complications can be minimized. A thorough informed consent process should also focus on patient-specific and family-centered issues. The patient's specific risk factors should be presented to the individual and his or her family for consideration before treatment.

Medical Risk Factors

An essential part of risk assessment includes the surgeon's performance of a thorough review of the patient's medical records and the completion of a history and physical examination. A general clearance examination by the patient's pediatrician or general physician and further confirmation of the patient's health through appropriate laboratory testing is also important. The pregnancy testing of females of childbearing age should be performed routinely the morning of surgery.

For middle-aged adults who are considering orthognathic surgery, additional medical risk factors may include cardiovascular disease; obesity or increased body mass index; obstructive sleep apnea; diabetes; pulmonary disease; smoking history; the potential for deep vein thrombosis or pulmonary embolus; and osteoporosis or the use of bisphosphonate treatment (see Chapter 25).[30,50,172] Evaluation by the patient's primary care physician (and, in many cases, by other medical specialists) and appropriate laboratory work (e.g., electrocardiography, chest radiography, chemistries, hematology, thyroid tests, coagulation studies) help to identify areas of concern. The adult patient with an elevated body mass index who is undergoing surgery is at greater than average risk for intraoperative and postoperative complications. The presence of obstructive sleep apnea in the

patient with a jaw deformity should be considered and ruled out (see Chapter 26).

Wound-Healing Risk Factors

Specific patient factors that may impair wound healing include diabetes mellitus, smoking, bisphosphonate treatment for osteoporosis, poor nutritional status, and chronic glucocorticoid exposure. Type II *diabetes mellitus* is frequently a comorbidity of obesity and increased body mass index. Alpha and colleagues documented a 66% incidence of postoperative infections among diabetic patients undergoing maxillary or mandibular osteotomies, despite adequately controlled glucose levels.[9]

The *inhalation of nicotine* via cigarettes affects both pulmonary function and wound healing.[72,75,95,264,137,149,176] Smoking or nicotine ingestion has been shown to delay the chondrogenic phase of bone (osteotomy) healing and to decrease the circulation of surgically elevated flaps, thereby resulting in impaired healing after wound closure. Cigarette smoke contains nicotine, carbon monoxide, and hydrogen cyanide; each of these chemicals has been shown to impair wound healing by producing relative tissue hypoxia. Nicotine is a vasoconstrictor that diminishes tissue oxygenation, predisposes the individual to microvessel thrombosis (i.e., increased platelet adhesion and direct endothelial cell damage), and diminishes cell proliferation and function. Carbon monoxide reduces oxygen-carrying capacity, and hydrogen cyanide inhibits oxidative metabolism and oxygen transport. These consequences of cigarette smoke tissue ischemia are most evident during healing after surgical procedures, when tissue circulation is at risk for interruption (e.g., after microvascular tissue transfer, during the placement of free [non-pedicled] bone grafts). A number of clinical studies have documented increased morbidity in patients who smoke and later undergo head and neck reconstructive and aesthetic procedures. To minimize postoperative wound difficulties and anesthesia-related pulmonary complications, patients should be counseled to refrain from smoking for at least 6 to 8 weeks preoperatively and for 2 to 4 weeks postoperatively. A serum or urine nicotine concentration can be obtained before surgery to confirm patient compliance.

It is known that *bisphosphonate treatment for osteoporosis* can affect osteoclast activity and that it may negatively impact successful bone healing. Current guidelines concerning bisphosphonate treatment continue to evolve and should be followed when planning jaw or dentoalveolar procedures.[1,85,125,198,209,214,215,220-222,248,283,285,292,294,357,367]

Perioperative Airway Risk Factors

Perioperative risk to the airway and its management should be considered during all phases of surgical care, including before nasotracheal intubation, during surgery, at the time of extubation, and soon after extubation (Fig. 16-1).[53,61,76,170,175,250] The basic clinical preoperative airway assessment should include 1) measurements of mandibular range of motion (i.e., vertical opening and protrusion); neck mobility 2) Mallampati classification 3) the ratio of the patient's height to the thyromental distance 4) neck circumference and 5) body mass index. A Mallampati classification of III or IV or a ratio of height to thyromental distance of more than 23.5 is a strong indicator of a difficult

• **Figure 16-1** Perioperative risk to the airway and its management should be considered during all phases of orthognathic surgery, including before nasotracheal intubation, during surgery, at the time of extubation, and soon after extubation. Individuals who are scheduled for orthognathic surgery and who are recognized to be at high risk for airway compromise should be reviewed in advance with the anesthesia team to arrange for special needs. Three patient examples in which these issues came into play are shown, including an adult with Noonan syndrome, a teenage girl with Treacher Collins syndrome, and a teenager with Klippel–Feil anomaly. Each of these patients also has the potential for cervical spine anomalies with limited neck range of motion. The risk for spinal cord injury should also be evaluated before orthognathic surgery.

airway with conventional direct laryngoscopy. A *neck circumference* of more than 17 inches in men is a risk factor for obstructive sleep apnea. Individuals who are scheduled to undergo orthognathic surgery may have special medical risk factors that include baseline malformations or syndromes and associated abnormalities that carry the potential for difficult airway control (see Chapter 11). In the high-risk patient, advance discussion with the anesthesia team to arrange for an anesthesiologist who is experienced with difficult away management and who is skilled with awake fiber-optic intubation may be indicated (see Fig. 16-1) (Video 5). After the nasotracheal tube is in place, the surgeon secures it and assists with its management during the operation (see Chapter 15). Complications associated with endotracheal tubes that are placed for orthognathic procedures have included herniation of the airway tube cuff leading to occlusion of the tube lumen; inadvertent surgical sectioning of the endotracheal tube; and the occurrence of contact granuloma of the vocal cords.[62,249] Acute intraoperative or postoperative pulmonary edema, postoperative apnea, pneumomediastinum, and pneumothorax have all been reported as complications associated with anesthesia (see Chapter 11).[14,33,34,56,91,117,122,153,217,233,245,259,318,321]

Malignant hyperpyrexia involves an abnormality of muscle metabolism of genetic origin that manifests as widespread skeletal muscle contraction, hypercatabolism, and hyperthermia upon exposure to volatile anesthetic agents or succinylcholine.[192,223,365] It is a serious condition that requires prompt diagnosis and aggressive management. It may occur during an orthognathic procedure with a typical initial intraoperative presentation of tachycardia and noticeably warm skin.

Decisions about the timing of extubation after surgery should be made by clinicians on the basis of extubation criteria and patient-specific risk factors.[159,376] In general, the surgeon is present at the time of extubation in case either *emergency tracheostomy* or reintubation is required. Our preference is to extubate the patient in the operating room when feasible but only when specific parameters are met. For the patient with an ongoing difficult airway, a more delayed approach to extubation maybe taken. The risk of inadequate ventilation after extubation is highest when preextubation parameters are not adequately met and when excess sedation remains (see Chapter 11).

Spontaneous leg compartment syndrome has been reported as a complication after orthognathic surgery.[204] The occurrence is thought to be the result of a drug interaction between anesthetic or postoperative medications or the effect of antidepressive drugs that are superimposed on chronic mild exercise-induced compartment syndrome.

Death is recognized as an exceedingly rare complication related to orthognathic procedures. Of the numerous large case series published, only one death has been described; this was reported by Van de Perre and colleagues from a series of 2049 patients.[210] It occurred in a 17-year-old male patient who was described as being slightly mentally challenged and who underwent bimaxillary osteotomies. Six hours postoperatively, a cardiac arrest occurred, and attempts to resuscitate the patient failed. The cause was thought to be preexisting cardiomyopathy. A death was also reported by Waack and colleagues as part of a limited series of 63 consecutive Le Fort I osteotomies.[216] The death occurred in a healthy 15-year-old female patient on the first postoperative day as a result of unknown causes.

Cervical Spine Risk Factors

As part of the presurgical assessment, the evaluation of the patient's cervical spine is essential. *Congenital malformations*, *previous trauma,* and *arthritis* are occasional etiologies of cervical spine problems in the orthognathic patient. Patients with specific syndromes (e.g., Klippel–Feil anomaly, hemifacial microsomia, Treacher Collins syndrome, Apert syndrome) are at higher risk for a cervical spine malformation that may be undiagnosed at presentation to the orthognathic surgeon (see Fig. 16-1). Referral for evaluation by an orthopedic spine surgeon, a neurosurgeon and a geneticist as well as appropriate radiographic studies (e.g., magnetic resonance imaging, computed tomography) are carried out when indicated. Controlled positioning with limited flexion, extension, and rotation at the time of intubation, intraoperatively, and during recovery may be required. In some cases, *intraoperative neuro-monitoring* (e.g., motor and sensory) is useful to confirm minimal spinal compression during surgery.

Psychosocial Risk Factors

To minimize patient and family disappointment after surgery, an informal (or, in some cases, a more rigorous) *psychosocial assessment* is conducted.[24,36,73,81,167,304,317] All parties involved must accept the risks and limitations of the planned surgical procedures. Unfortunately, neither the preoperative psychosocial assessment nor the individual's biologic wound-healing ability is completely predictable. A small percentage of patients will perceive an unfavorable surgical outcome, even when the clinicians feel that the results are at least satisfactory. Others will not accept a complication, even though the risks were fully explained in advance and all precautions were taken.

Body dysmorphic disorder is a medical condition that can be difficult to recognize before surgery but that will routinely result in postoperative patient dissatisfaction. It is estimated that 7% to 15% of individuals who pursue cosmetic surgery have body dysmorphic disorder (see Chapter 7).

Comprehensive Dental Rehabilitation Needs

Recognizing when a patient who is seeking orthognathic surgery has dental needs that go beyond standard orthodontics is essential to the achievement of an optimal outcome (see Chapter 5). If *complex dental restorations* and *periodontal management* are necessary, then planning should

coordinate care for these needs at the beginning of treatment (see Chapter 6). This is especially true for the individual with a cleft lip and palate or a craniofacial syndrome (see Chapters 28 through 34). It is also true for the adult who has suffered dental and periodontal sequelae from an uncorrected dentofacial deformity or previous treatment (e.g., orthodontics, restorative care) (see Chapter 25).

Functional Assessment of Head and Neck Structures

The orthognathic surgeon should evaluate the patient's baseline head and neck functions, including speech, swallowing, mastication, neck and mandibular range of motion, breathing, hearing, vision, cognition, and psychosocial competence. Any of these functions may be negatively affected by the presenting maxillofacial deformity or malformation and influenced (either favorably or unfavorably) by the treatment being contemplated.[94,113,139,163,228,232,311,313,320,377] A candid discussion with the patient and the family about the potential impact of the planned procedures on any of these functions should take place before treatment.

Operating Room Team Preparation

Limiting risks and complications requires an informed patient and family, coordinated collaborative comprehensive clinical care, an experienced and focused surgeon, meticulous planning, a dedicated operating room team, effective postoperative hospital care, and appropriate outpatient management. The *operating room team* includes the surgeon and his or her assistants, the anesthesiologist and his or her assistants, the operating room personnel (e.g., scrub nurse/technician, circulating nurse), and the full complement of sterilized and organized instrumentation (e.g., inventory, sanitation and sterilization personnel). The *instrumentation* includes power saws and drills, with their associated bits and blades; handheld instruments; and fixation hardware such as plates and screws. Specific *medications, sutures,* and *dressing materials* are also required. The confirmation of the prepared operating room team, equipment, instrumentation, and medications should be undertaken before the patient enters the operating room. The anesthesiologist and the surgeon should discuss the patient's airway and his or her intraoperative and postoperative needs before entering the operating room (see Chapter 11). After the equipment, instrumentation, and medications are set up and checked and the team is briefed, the patient may enter the operating room with the best opportunity for success.

Temporomandibular Disorders

The effects of orthognathic surgery on temporomandibular joint function are often specific to the patient and cannot be fully predicted.[11,82,138,139,156,247] Many clinical studies report improvement in temporomandibular joint symptoms in a majority of patients after successful orthodontics and orthognathic surgery, whereas some of the study patients show deterioration. The favorable effects of orthognathic surgery for patients with preexisting temporomandibular joint symptoms may result from the improved occlusion, the corrected facial height and horizontal projection, and the reduced emotional stress (or the placebo effect). Varied degrees of documented radiographic condylar remodeling after orthodontics and orthognathic surgery have been reported in some patients. Interestingly, TMJ radiographic findings do not necessarily correlate with symptomatic temporomandibular disorders (see Chapters 9 and 37).

Management of Third Molars

Historically, the presence of impacted third molars during a sagittal ramus osteotomy (SRO) of the mandible has been presumed to be associated with increased complications including the following: 1) a higher incidence of "bad splits" 2) a higher incidence of infection 3) issues with the placement of fixation (i.e., osteotomy stabilization) and 4) difficulties maintaining the integrity of the inferior alveolar nerve (IAN). It is for these reasons that, in the past, the removal of impacted mandibular third molars 6 to 12 months before an SRO was routinely recommended. However, the arguments against this approach are that this subjects the patient to two surgeries, two anesthetic treatments, and two recovery periods rather than just one and that this staged approach may in fact increase rather than decrease the previously mentioned complications.

Clinical studies have now documented no increased risk of unfavorable or so-called "bad splits" when SROs are performed in patients with impacted third molars as compared with those without third molars.[90,218,268,276,348] Doucet and colleagues laid this argument to rest with their prospective cohort study that looked at the effects of the presence or absence of third molars during SRO on 1) the frequency of unfavorable fractures 2) the degree of entrapment of the IAN in the proximal segment and 3) the procedural time required to move the teeth.[89] The study included patients who were scheduled to undergo SRO to correct a dentofacial deformity. Study patients were divided into two subgroups: Group I was made up of those with an impacted mandibular wisdom tooth at the time of SRO (n = 331; mean age, 19.6 years); Group II included those without a mandibular wisdom tooth at the time of SRO (n = 346; mean age, 30.4 years). The overall incidence of unfavorable fractures was 3.1% (21 out of 677), with frequencies of 2.4% (8 out of 331) in Group I and 3.8% (13 out of 346) in Group II. Interestingly, the rate of IAN entrapment in

NOTE: When the removal of impacted mandibular third molars is recommended to the patient for long-term dental health, it is generally this author's preference to do so at the time of orthognathic surgery (Fig. 16-2, *A*).

the proximal segment was significantly lower in Group I (37.2%) than in Group II (46.5%). The removal of third molars at the time of SRO increased procedural time by only 1.7 minutes.

As documented in published studies, this author has not experienced increased levels of complications (e.g., infection, bad split, inability to place fixation, increased incidence of inferior alveolar paresthesia) by doing so.

Exceptions to the routine removal of impacted wisdom teeth at the time of orthognathic surgery may include a severely hypoplastic mandible that requires extensive advancement (i.e., in certain patients with hemifacial microsomia or Treacher Collins syndrome). In such cases, maximal ramus region bone stock is needed for osteotomy stabilization and healing. For those few patients, the preoperative removal of the wisdom teeth followed by adequate time for sufficient bone fill (i.e., 12 months) may be preferred.

When the removal of erupted or partially erupted mandibular wisdom teeth is carried out at the time of SRO, the standard vestibular incision for SRO is altered to achieve access to the erupted wisdom teeth and then for watertight wound closure (Fig. 16-2, *B*) (also see Chapter 15). *In review, when considering evidence-based medicine, the routine practice of the removal of wisdom teeth before SRO can no longer be justified.*

When removing impacted maxillary wisdom teeth, access through the floor of the antrum after down-fracture at the time of Le Fort I osteotomy represents this author's usual approach (Fig. 16-2, *C*). The exception is when carrying out segmental osteotomies in a cleft maxilla in conjunction with extensive horizontal advancement of the lesser segment. In these cases, the removal of the wisdom tooth in the lesser segment may significantly diminish bone volume with potential compromise of stabilization and healing.

The removal of an erupted or partially erupted maxillary wisdom tooth is completed through the gingival opening before down-fracture. When this is done, minimal flap elevation is essential so as not to compromise maxillary perfusion after down-fracture.

Fixation Considerations

Need to Remove Plates and Screws

Metal plates and screws have been used to stabilize fractures and osteotomies in the maxillofacial region for more than 40 years.[8,9,12,39,46,47,104,131,151,164,225,281,336] The metals that were initially used were stainless steel and vitalium; these were followed by titanium and its alloys.

There is both laboratory and clinical evidence of the tissue compatibility and the high corrosion resistance of titanium and its alloys in the human body. In 1991, the Strasbourg Osteosynthesis Research Group recommended that "the removal of a non-functional plate is desirable provided that the procedure does not cause undue risk to the patient."[32A,355A] During earlier years, those who were advocating the routine removal of fixation devices after stable bone union felt that the implant had significant potential to cause problems, so its removal was considered preventative. Today, clinicians agree that the medical risk and financial costs incurred by the routine removal of asymptomatic fixation hardware cannot be justified. It is currently standard practice to remove fixation plates and screws only when clinically indicated.

Becelli and colleagues completed a retrospective analysis of complications of bicortical screws used for sagittal split ramus osteotomy (SSRO) fixation.[22] The authors reviewed a consecutive series of patients (N = 241) who were undergoing sagittal ramus osteotomies (N = 482) for skeletal Class III deformities. Complications related to bicortical titanium screws were observed at 3% of the osteotomy sites (Fig. 16-3). The hardware was eventually removed in these patients (15 out of 482), without long-term consequences.

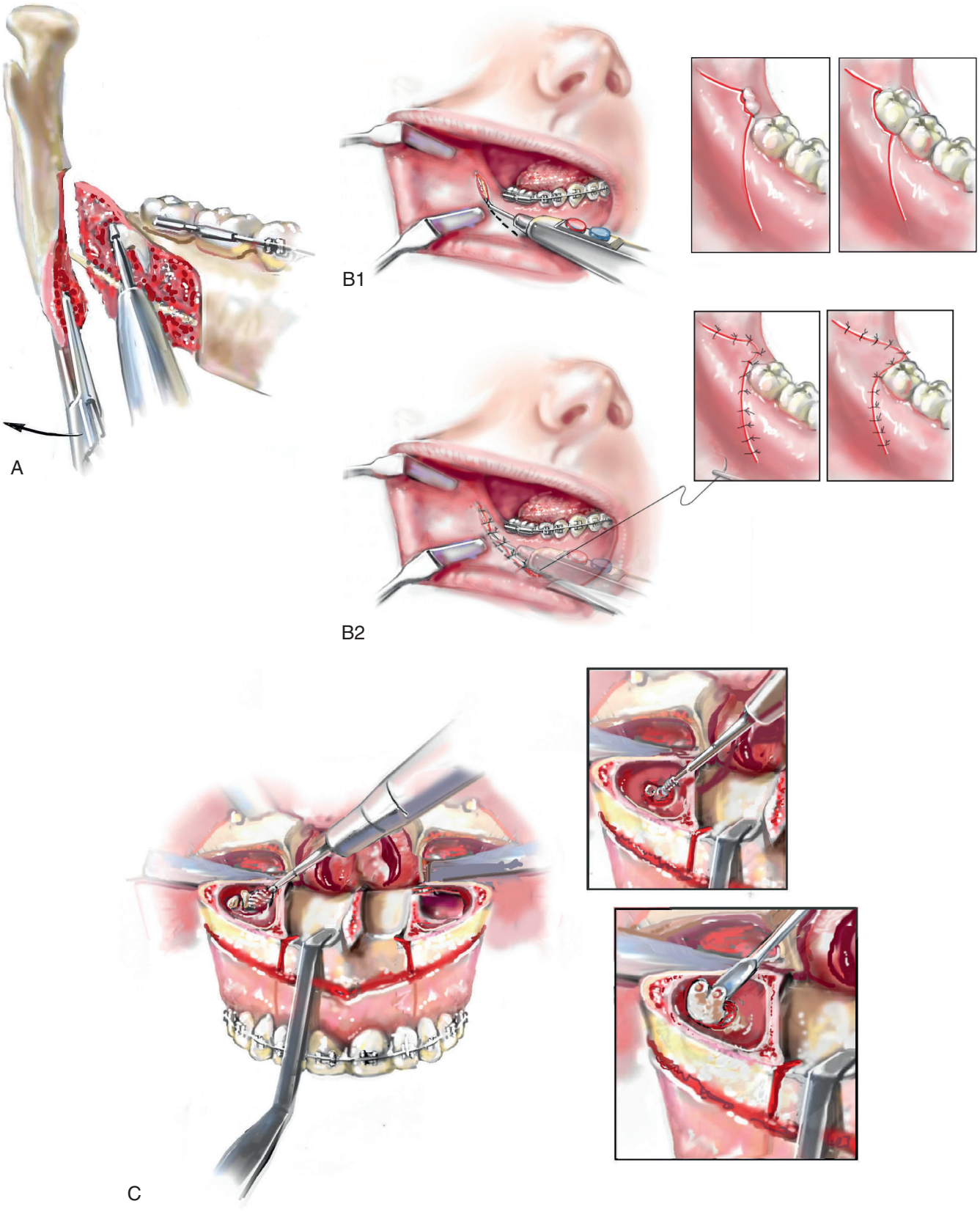
NOTE: In this author's personal series of orthognathic procedures from more than 27 years of clinical practice, the need to or a request by the patient to remove a plate or screw after healing has involved less than 2% of individuals (Fig. 16-3).

The indications for removal fall into the following categories: 1) a palpable and bothersome piriform aperture plate or screw at the Le Fort I osteotomy site when the skin is compressed against it with a finger; 2) a palpable and bothersome zygomatic buttress plate when a food bolus compresses against it while chewing; 3) an exposed bicortical screw head in the labial vestibule at the SSRO site; 4) a screw in close contact with a dental root that is symptomatic; and 5) a bicortical screw that extends through the lingual cortex of the ramus and that causes discomfort or concern, either during movement of the tongue or upon contact with food during swallowing. Under these circumstances, the elective removal of the involved plate or screw is carried out after osteotomy healing has occurred (i.e., preferably 6 to 12 months after surgery).

Use of Titanium versus Resorbable Fixation

The use of self-reinforced biodegradable bone plates and screws has been advocated for use in orthognathic surgery by a limited number of surgeons throughout the world.[92,96,127,130,155,165,178,297,325,344,345] Although this type of fixation is appealing in theory, a greater incidence of postoperative complications has been found with resorbable fixation devices as compared with titanium. Furthermore, no advantages in the achievement of improved skeletal stability have been documented with the use of biodegradable hardware.

Turvey and colleagues reported about 70 patients who were undergoing 194 osteotomies of the maxilla or the mandible and who were stabilized with self-reinforced polylactate bone plates or screws of similar size and configuration to those of titanium systems.[344] The placement of the devices was accomplished transorally and transfacially in a way that was consistent with the osteotomy location. Maxillomandibular elastics were used for each patient after



• **Figure 16-2** **A,** Illustration of an impacted mandibular wisdom tooth being removed in conjunction with sagittal ramus osteotomy. **B,** Illustrations of incision options that are used for ramus osteotomy exposure and then for wound closure. The incision used is dependent on wisdom tooth location. **C,** Illustrations of the removal of an impacted maxillary wisdom tooth through the sinus after Le Fort I down-fracture.

• **Figure 16-3** An 18-year-old with a bilateral cleft lip and palate jaw deformity underwent cleft–orthognathic surgery including maxillary Le Fort I osteotomy in three segments and interpositional iliac bone grafting. Healing was uncomplicated, with the achievement of the planned improvements. One year after surgery, the patient returned with a complaint of a painless ridge in the left buccal vestibule of the maxilla. Examination confirmed an exposed titanium plate and screw. There were no associated symptoms or signs of infection. During an office procedure, the exposed plate was sectioned with a wire cutter and removed. Healing was uneventful.

surgery to control the position of the jaws. Three out of the 70 patients (4%) experienced immediate postoperative loosening of the resorbable bone screws. Each of the patients required a return to the operating room for more rigid stabilization at the involved osteotomy site. Although all patients eventually achieved acceptable occlusion, a number of fixation sequelae occurred during the healing phase, including prolonged mobility of the maxilla; sterile abscess requiring debridement; swelling with sterile fistula tract formation; and intranasal inflammation requiring fixation removal at the time of secondary septoplasty. Although the authors demonstrated that biodegradable plates and screws could be used for orthognathic surgery, there was no advantage when this was compared with titanium fixation devices, and the complication rate is increased.

In a second study, Turvey and colleagues reported about 69 patients who were undergoing sagittal split ramus osteotomies for mandibular advancement.[345] Thirty-four of these patients underwent fixation with self-reinforced biodegradable screws, whereas 35 patients underwent fixation with 2-mm titanium screws. Postoperatively, one of the patients with biodegradable screws required a return to the operating room for improved fixation. Again, no advantage could be demonstrated with the use of biodegradable fixation, whereas a degree of diminished levels of stabilization and increased complications were shown to be present.

Ahn and colleagues completed a study to evaluate the complications of resorbable versus nonresorbable plate and screw fixation in orthognathic surgery.[3] Patients were enrolled in the prospective study (N = 272). Titanium plates and screws were used at the osteotomy sites in Group I (N = 152), and resorbable plates and screws were used at the osteotomy sites in Group II (N = 120). In the 152 patients with titanium plates and screws, there was a complication rate of 8.6% (N = 13). Of the 120 patients with resorbable plates and screws, 18.3% (N = 22) developed complications. There was a greater degree of postoperative infections and malocclusions and a trend toward relapse observed in patients with resorbable plate and screw fixation. Three of the patients with resorbable plate and screw fixation required reoperation for residual malocclusion as compared with zero in the titanium group.

Infection Associated with Orthognathic Procedures

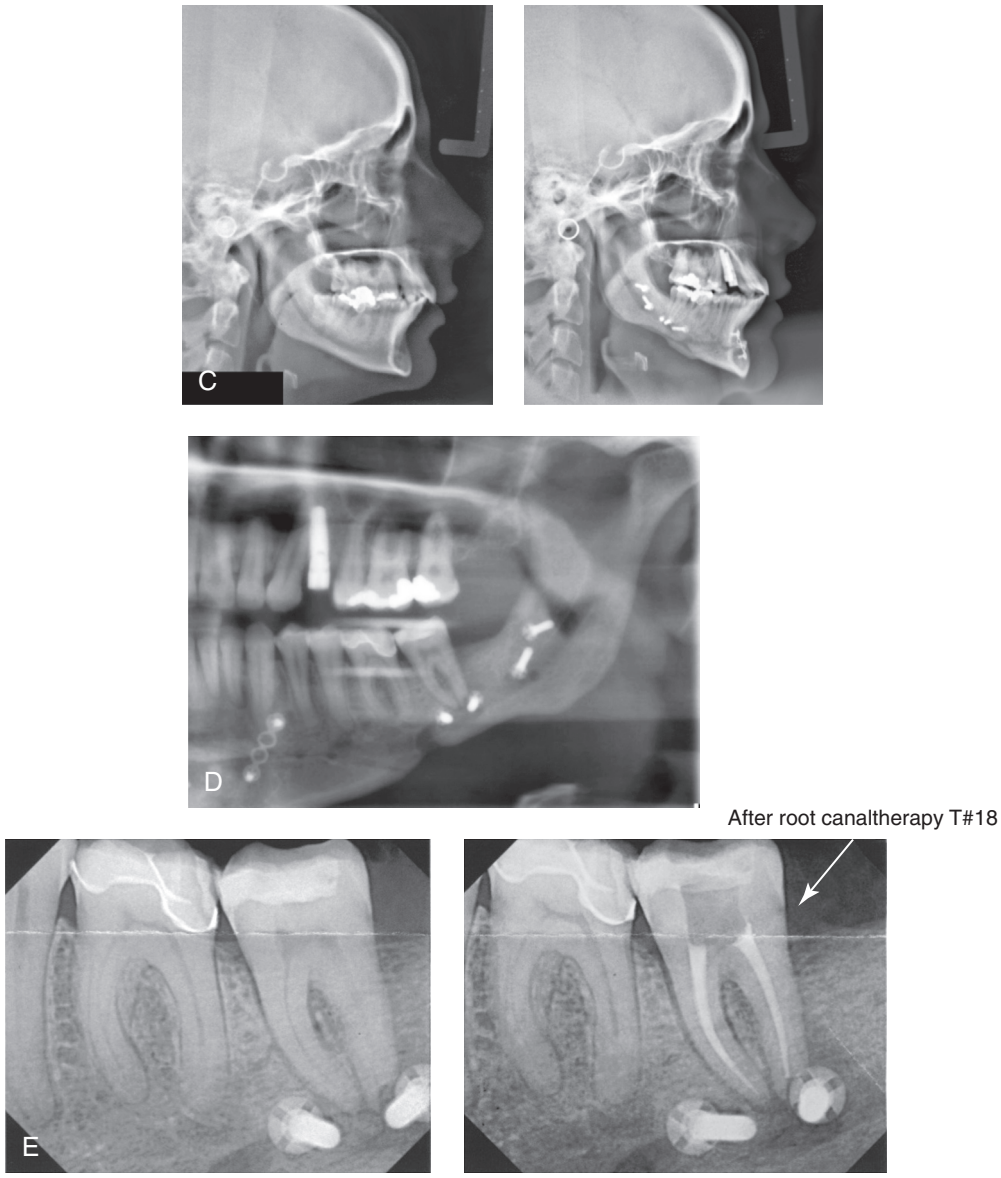
Infection after orthognathic surgery is considered by most to be uncommon. However, a review of the literature indicates a wide range in the reported incidence of this complication.* A critical review of these studies provides insight into the true incidence of infection, clinical pitfalls to consider, and methods to use to best limit these sequelae.

All transoral wounds must be considered "clean-contaminated wounds," with an anticipated theoretical infection rate of as high as 15%.[352] It is said that this incidence of infection in a clean-contaminated site can be reduced to as low as 1% with good surgical technique and the appropriate use of prophylactic antibiotics. The question remains of how to best provide this type of antibiotic coverage for the orthognathic surgery patient. According to the infectious disease literature, antibiotic prophylaxis should provide adequate drug levels in the tissue before, during, and for the shortest possible time after surgery to provide a reduced infection rate; be an effective agent against the bacteria that are most likely to cause the infection; and be bactericidal while at the same time being the least toxic agent available.

Spaey and colleagues reported a 4% prevalence rate of infection after orthognathic procedures in their cohort.[312] Among the infections that were sustained, 92% occurred at the SSRO site, 1% occurred at the Le Fort I osteotomy site, and only 0.5% involved the chin osteotomy location (Figs. 16-4 through 16-7). In their pilot study group, after completion of an SSRO, the authors placed a drain through the mandibular vestibule mucosa incision; this was removed the following morning. The prophylactic antibiotic regimen that was initially used did not have a drug level in place while the drain remained in or after its removal. Because of the high infection rate observed in the pilot study, the researchers changed the protocol to no longer involve the placement of a drain; to achieve a more meticulous mucosal wound closure; and to extend the time of prophylactic antibiotic use to 5 days after surgery. As a result, the infection rate at the SSRO site was dramatically decreased. The authors concluded that, in addition to an extended use of prophylactic antibiotics, the achievement of a sealed vestibular wound at the SSRO is essential to limit the infection rate.

*References 19, 20, 29, 48, 49, 77, 103, 105, 107, 108, 110, 111, 119, 133, 158, 171, 201, 213, 257, 279, 282, 284, 312, 319, 350, 375, 379.

• **Figure 16-4** A woman in her early 30s with a primary mandibular deficiency growth pattern underwent a comprehensive orthodontic and surgical correction. This required the removal of dental compensation, including opening upper bicuspid extraction spaces for dental implants. Surgery included bilateral sagittal split ramus osteotomies, an osseous genioplasty, and anterior neck soft-tissue procedures. In-hospital and early at-home convalescence proceeded without incident. Five weeks after surgery, the patient returned to a more normal diet, to regular physical activities, and to her orthodontist's care. Two months after surgery, pain in the left mandibular second molar region with chewing was persistent. The tooth was also tender to percussion. A periapical radiograph revealed the proximity of a bicortical fixation screw to the distal root, with possible apical root resorption. The removal of the involved screw was offered. However, the patient chose root canal therapy followed by the relief of pain and the resolution of percussion tenderness. **A,** Profile and lateral cephalometric views before treatment. **B,** Oblique facial views before and after treatment.



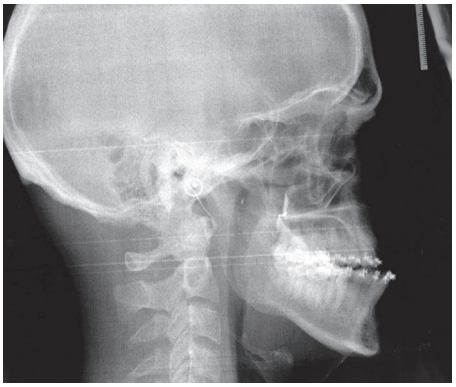
• **Figure 16-4, cont'd C,** Pretreatment and posttreatment lateral cephalometric radiographs. **D,** Left side Panorex image 2 months after surgery, with left mandibular second molar pain with chewing. **E,** A periapical radiograph is shown before and after successful root canal therapy (see Fig. 19-8).

In a randomized, double-blind study, **Ruggles and colleagues** tested two different antibiotic regimens in patients undergoing orthognathic surgery.[284] Both groups received preoperative and intraoperative antibiotics, whereas only one group continued 48 hours of postoperative coverage. The group that received no postoperative antibiotics had a 15% infection rate, whereas no infections were found in the group with extended antibiotic coverage.

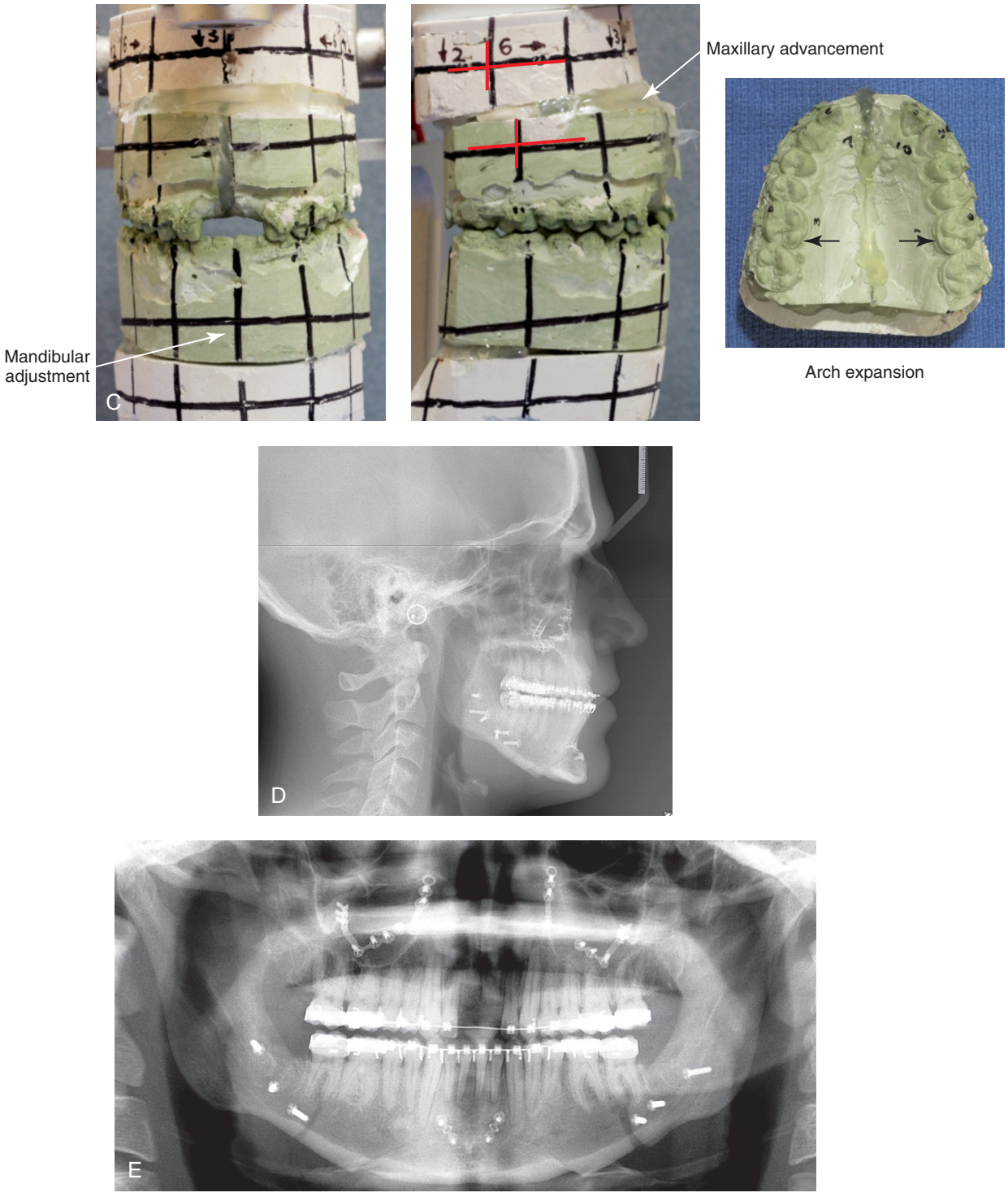
Danda and colleagues completed a prospective study to evaluate the use of prophylactic antibiotics among patients who were undergoing orthognathic surgery.[78] The study patients (N = 150) were divided into two groups. Group I (N = 75) received a single dose of antibiotic prophylaxis. Group II (N = 75) received a full day of antibiotic prophylaxis. In Group I, 9.3% of patients (N = 7) developed infection, whereas only 2.6% (N = 2) of Group II patients did. The results indicate a clinically significant increased rate of infection with single-dose antibiotic prophylaxis as compared with a full day of antibiotic prophylaxis.

Kublefelt and colleagues reported on smoking as a significant risk factor for infection after orthognathic surgery.[176A] This was a retrospective cohort study of individuals (n = 286) who were undergoing one-jaw orthognathic surgery (i.e., bilateral SSRO [BSSRO] or Le Fort I osteotomy) during a 7-year period. All patients received a 1-week course of antibiotics. Patients ranged in age from 17 to 56.5 years (mean, 34.8 years). The overall infection rate was 9.1%. The only statistically significant identified risk factor for infection in these study groups was smoking history. The incidence of infection was 14.4% among

Orthodontic decompensation in progress



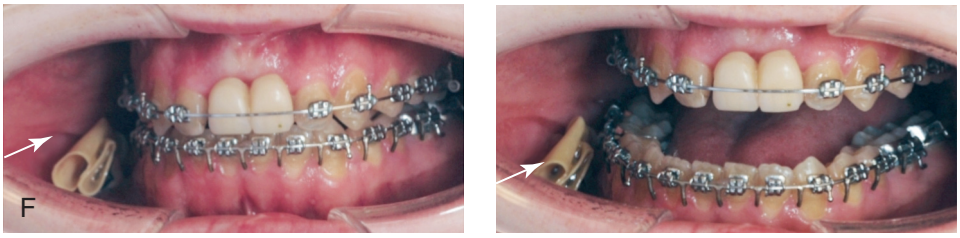
• **Figure 16-5** A 23-year-old man with a developmental jaw deformity and malocclusion was referred for surgical evaluation. He had also recently sustained trauma to the maxillary anterior region that involved a loss of the central incisors. The jaw deformity was characterized by maxillary deficiency in combination with relative mandibular excess. The patient agreed to a comprehensive surgical and dental rehabilitative approach. He also had lifelong difficulty breathing through his nose. After periodontal and restorative evaluations, the patient underwent orthodontic (dental) decompensation. Surgery included maxillary Le Fort I osteotomy in segments (arch expansion, horizontal advancement, and cant correction); bilateral sagittal split ramus osteotomies (asymmetry correction); osseous genioplasty (horizontal advancement); and septoplasty and inferior turbinate reduction. In-hospital and initial at-home convalescence proceeded without difficulty. Three weeks after surgery, increased swelling and tenderness along the right ramus region were appreciated, and intraoral drainage through the lateral ramus incision was recognized. The patient underwent exploration with Penrose drain placement as an office procedure under local anesthesia, and he was restarted on antibiotics. The drain was removed 1 week later, and the 10-day course of oral antibiotics was completed. Healing at all osteotomy sites remained on track. Five weeks after surgery, the patient returned to a more regular diet and physical activity without delay. The orthodontic appliances were removed 6 months after surgery. **A,** Presurgical facial and occlusal views with orthodontics in progress. **B,** Profile and lateral cephalometric views before surgery.



• **Figure 16-5, cont'd** **C,** Articulated dental casts that indicate analytic model planning. **D,** Three week postoperative lateral cephalometric radiograph. **E,** Three week postoperative Panorex radiograph.

Continued

Right facial swelling and intra-oral drainage 3 weeks after surgery



Incision and drainage buccal space abscess/drain in place 3 weeks after surgery

• **Figure 16-5, cont'd F,** Facial and occlusal views 3 weeks after surgery at the time of persistent right facial swelling and intraoral drainage. The patient underwent exploration and Penrose drain placement.

smokers and 7.0% among non-smokers. The site of infection was primarily the ramus region followed by the Le Fort I osteotomy region.

Interestingly, six cases of actinomycosis after orthognathic procedures have been reported.[216,293] **Ozaki and colleagues** reported a case of actinomycosis that occurred in the left submandibular area after SSRO.[249] The infection resolved after 2 months of antibiotic therapy.

NOTE: Current literature confirms that, for orthognathic surgery that includes SSRO, the following are true: 1) prophylactic antibiotics (preoperative, intraoperative, and postoperative) reduce infection rates 2) effective wound closure (without occurrence of dehiscence) will diminish infection rates 3) intraoral drains placed at the osteotomy site are counterproductive and 4) the incidence of infection is higher among smokers.

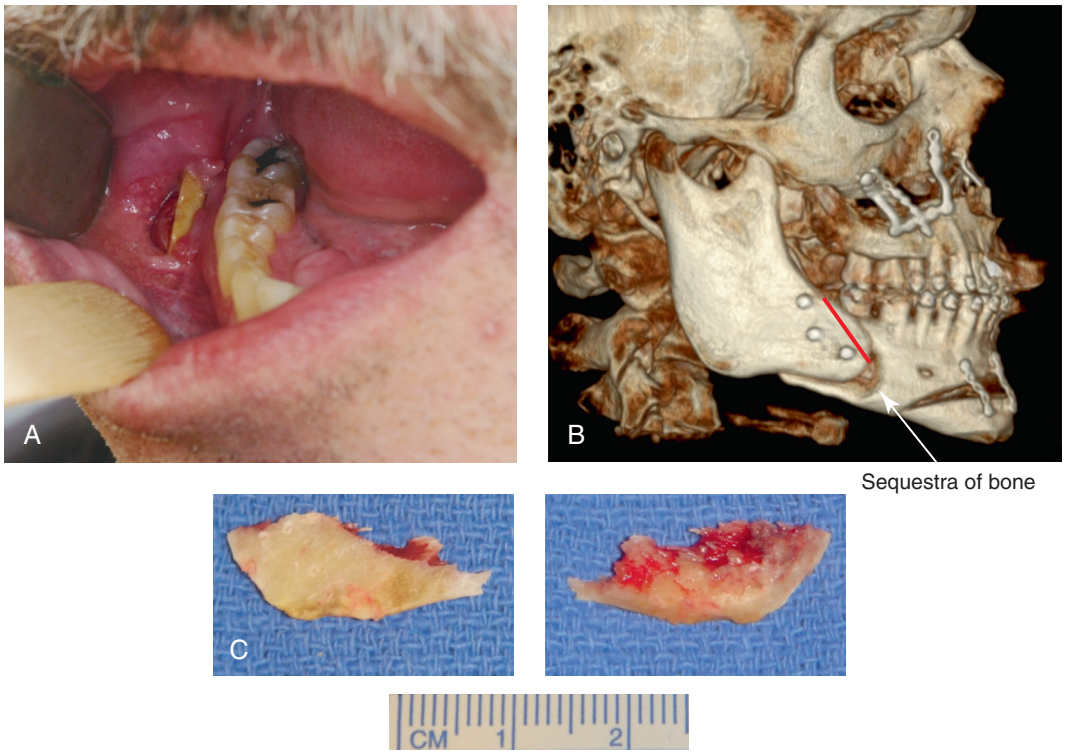
Instrument Breakage: Foreign-Body Reaction

Manikandban and colleagues reviewed the incidence and consequence of bur breakage within the surgical wound during orthognathic surgery.[210] The authors found that surgical bur breakage was not uncommon (5 out of 76 patients; 6.6%). They concluded that bur retrieval was not always advisable, because collateral damage to the hard and soft tissues may occur in the process. They recommended radiographic confirmation immediately after surgery and then clinical follow up for at least 1 year to confirm that no foreign body reaction or infection has occurred (Fig. 16-8).

Risks and Complications Specific to Sagittal Split Ramus Osteotomies of the Mandible

Unfavorable Sagittal Split Ramus Osteotomy

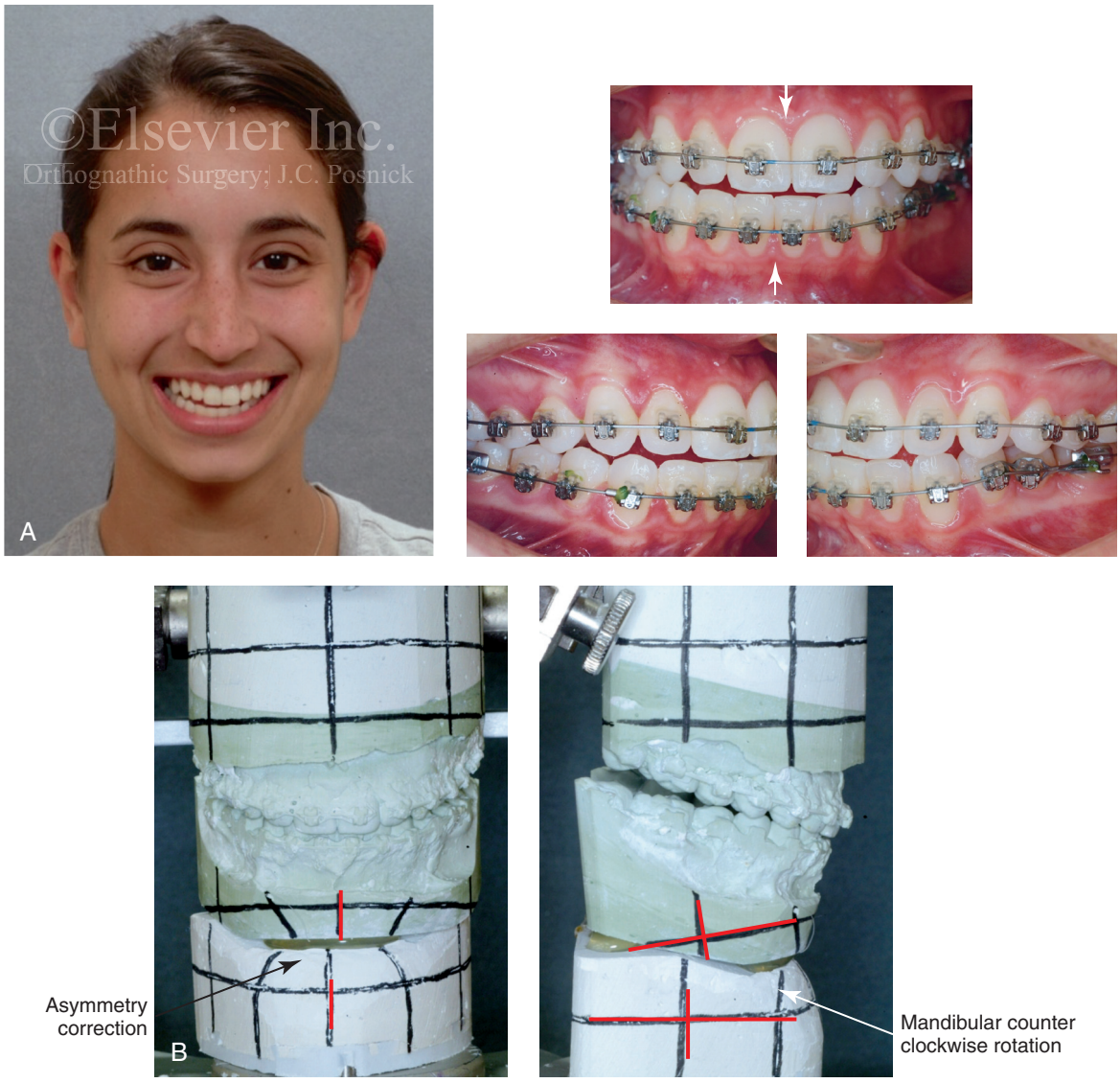
When completing an SRO, an unfavorable fracture (i.e., a "bad split") can occur.[23,42,139,162,206,243,334] The definition of a bad split varies from clinician to clinician. This author reserves the term *bad split* only for those SROs in which the condyle either remains with the distal segment, thus requiring a separate osteotomy (i.e., third piece), or shatters into a separate component (i.e., third piece) on its own (Fig. 16-9). Other less than ideal splits may include the following: 1) a separate fracture or piece of the buccal plate 2) a



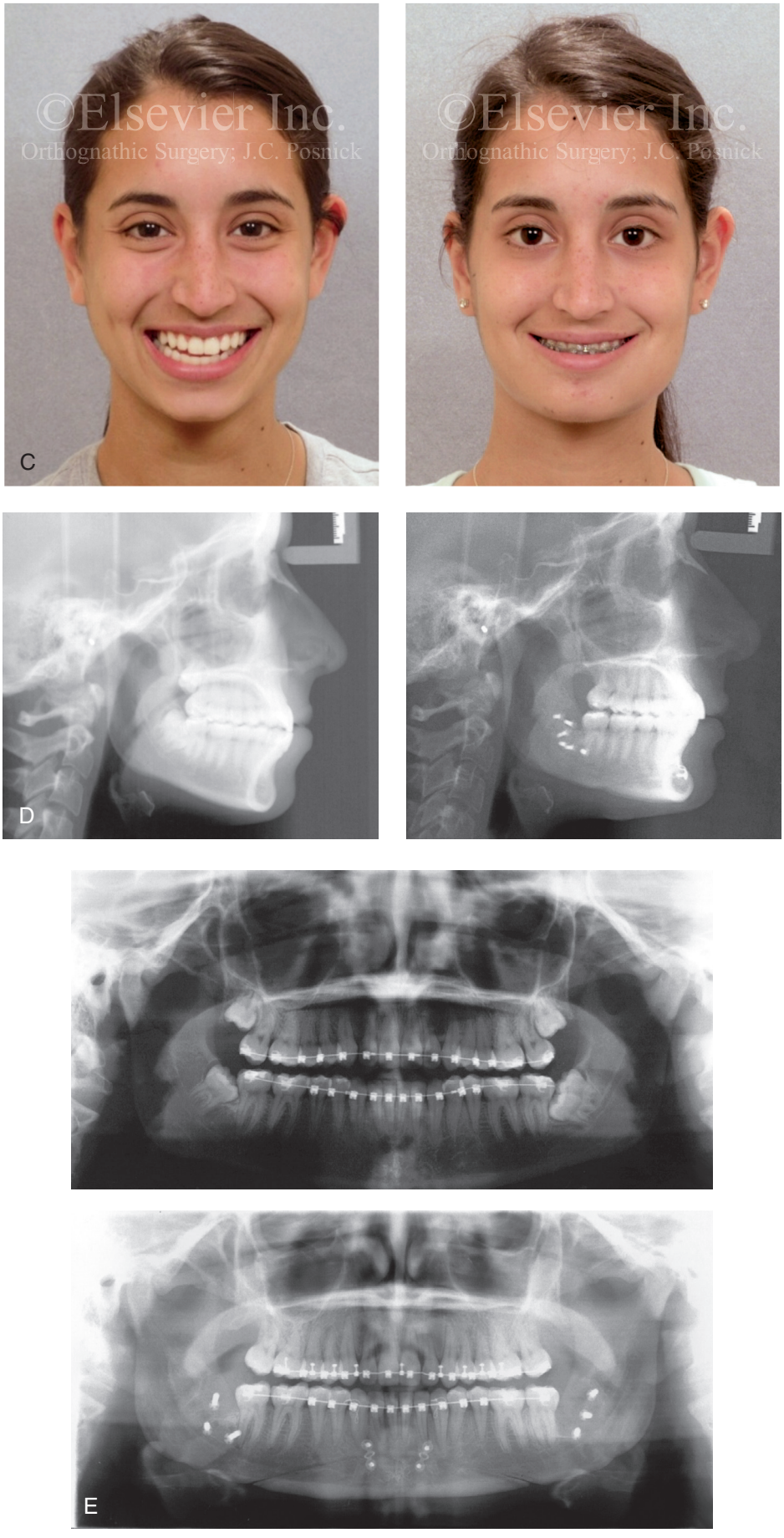
• **Figure 16-6** A 50-year-old man with a short face growth pattern and obstructive sleep apnea underwent orthognathic surgery, including maxillary Le Fort I osteotomy; bilateral sagittal split osteotomies of the mandible; and an osseous genioplasty. Interpositional allogenic grafting was required in the maxilla and chin regions. At the 2-week postoperative visit, partial dehiscence of the right mandibular vestibular wound was recognized. The distal superior aspect of the proximal segment was visualized through the wound. There was minimal associated facial swelling and no abscess or fluid collection. In the office setting, a rongeur was used to remove the exposed bone, and the patient was restarted on antibiotics. The wound spontaneously closed over the next 10 days. Healing at the osteotomy site remained on track, and there was no long-term sequelae. (Note: If the inferior borders had been more closely aligned before the bicortical screw fixation, then dehiscence with the loss of the bone fragment may not have occurred.) **A,** Intraoral view of the right mandibular vestibular wound at 10 days after surgery with dehiscence and exposure of the superior aspect of the proximal segment. **B,** Computed tomography scan demonstrating the portion of the buccal shelf and the proximal segment that was resected with the rongeur. **C,** Specimen removed with a rongeur.

separate fracture or piece of the lingual plate posterior to the second molar; or 3) a separate fracture or piece of the coronoid process. All three of these splits are generally manageable without alteration of the expected postoperative recovery. When the buccal plate separates from the proximal segment, the fragment is either removed or additional plate and screw fixation is used to secure it. When the lingual plate separates from the distal segment, no additional fixation is generally required. The lingual bone fragment is either removed or left in place. When the coronoid process separates from the proximal segment, it is usually removed to limit the chance of ankylosis during the healing process.

Text continued on p. 498



• **Figure 16-7** A 20-year-old college student with an asymmetric mandibular excess growth pattern (Hemi-mandibular elongation) arrived for surgical evaluation. She had previously undergone orthodontic attempts to neutralize the occlusion without success. She agreed to a combined orthodontic and surgical approach. With the removal of orthodontic (dental) compensation, surgery was carried out. The procedures included bilateral sagittal split ramus osteotomies (asymmetry correction) and osseous genioplasty (horizontal advancement). By 5 weeks after surgery, postoperative swelling had generally subsided. A painless "lymph node" was palpable in the right submandibular region. It was not fixed to the skin or bone. Three months after surgery, what appeared to be a localized lymph node became tender with erythema and then with drainage through the skin. Cultures were carried out and showed no specific growth. An infectious disease consult was obtained, and Gram staining showed gram-positive rods. The patient was placed on amoxicillin and then Augmentin. The localized infection involved intermittent drainage but without a connection down to the bone. Four months after surgery, the patient agreed to undergo excision of the soft-tissue mass with primary closure and the reinstituting of antibiotics. Histopathology was unremarkable, and the cultures remained negative. The wound healed without complications. **A,** Facial and occlusal views with orthodontics in progress. **B,** Articulated dental casts that indicate analytic model planning.



• **Figure 16-7, cont'd C,** Facial views with smile before and 5 weeks after surgery. **D,** Lateral cephalometric radiographs before and 5 weeks after surgery. **E,** Panorex radiographs before and 5 weeks after surgery.

Continued

• **Figure 16-7, cont'd F,** Right submandibular localized infection demonstrated by 3 months after surgery. **G,** Four months after surgery, the soft-tissue infection was excised and sent to the pathology and bacteriology departments. **H,** Frontal views with smile before and after successful treatment.

Prior to retreatment

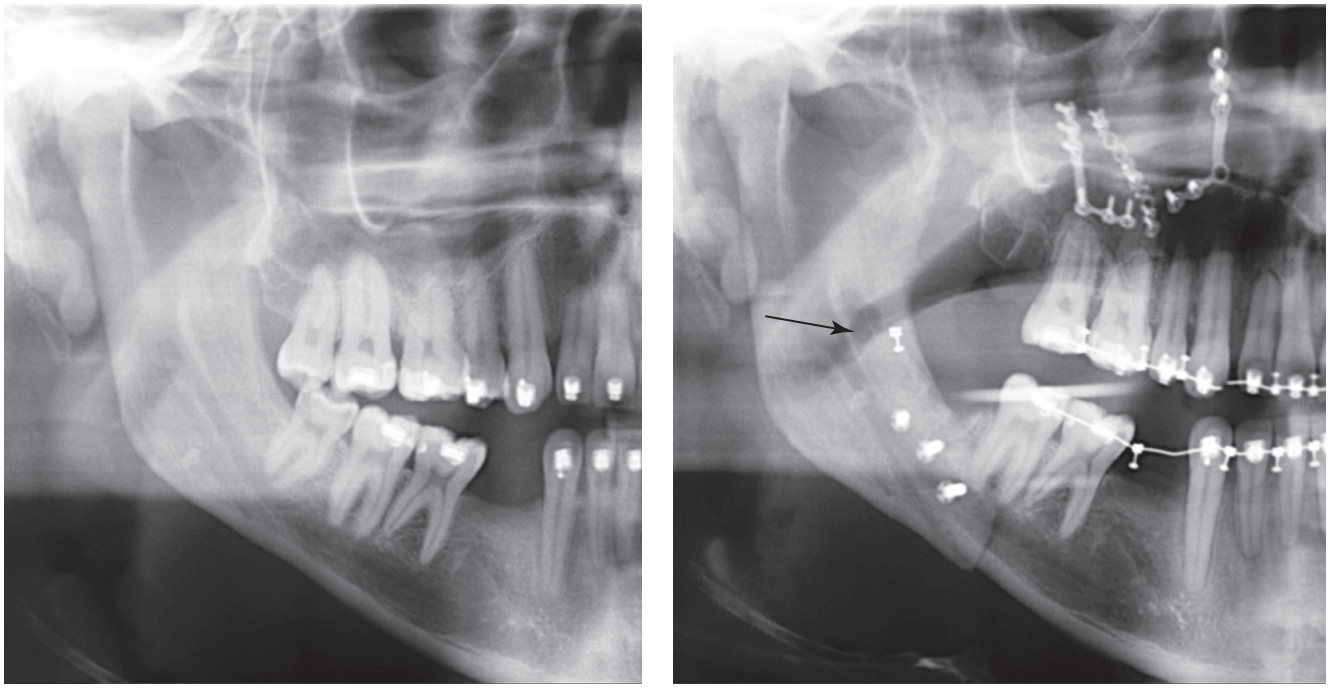
Pre surgery

After treatment

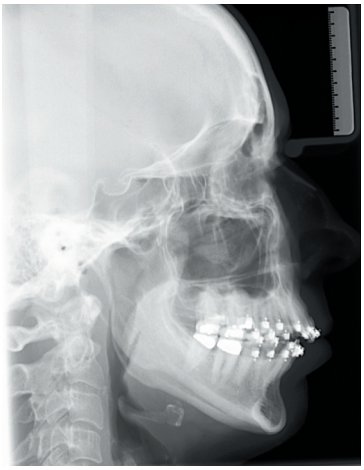
• **Figure 16-7, cont'd I,** Occlusal views before retreatment, just before surgery, and then after successful treatment. **J,** Right oblique facial views before and after successful treatment.

Continued

• **Figure 16-7, cont'd K,** Right profile views before and after successful treatment.

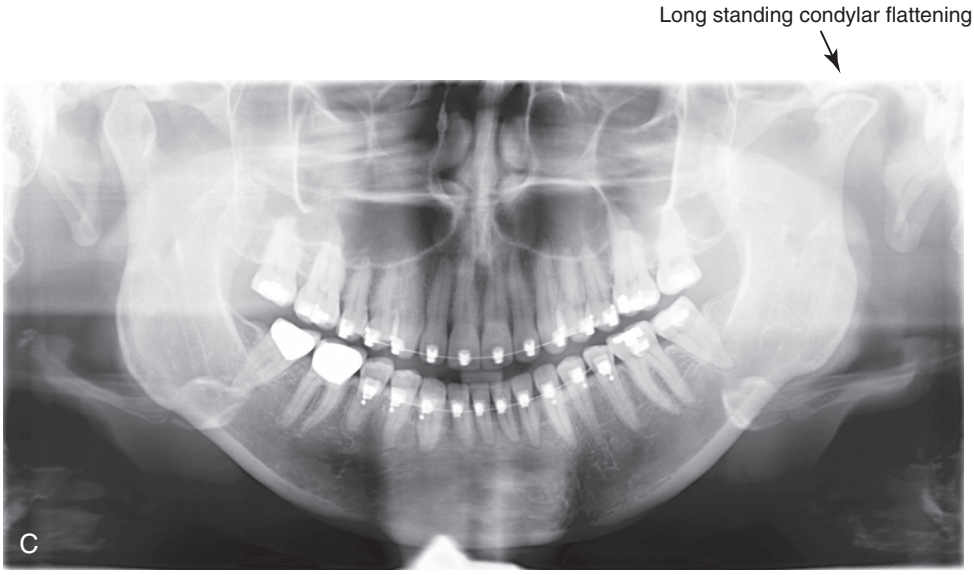


• **Figure 16-8** Individuals who are undergoing orthognathic surgery may experience intraoperative bur breakage. It is also not uncommon for orthodontic brackets, hooks, or ligatures to loosen with the potential for loss in the surgical field. Because of the small size of the lost objects within deep wounds, retrieval is not always advisable or possible. These foreign bodies can be visualized radiographically. Clinical follow up for at least 1 year is recommended to confirm that no foreign body reaction or infection has occurred. A Panorex radiograph is shown before and after orthognathic surgery. An orthodontic hook was lost in the right mandibular vestibular wound and can be seen on the Panorex radiograph. No negative sequelae resulted.

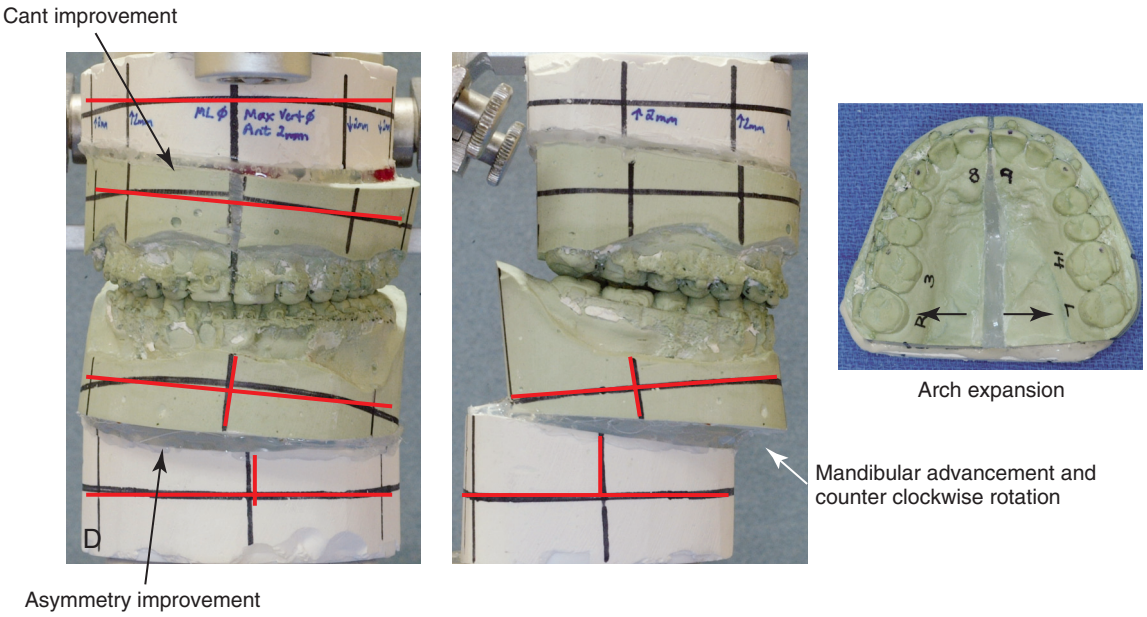


• **Figure 16-9** A woman in her mid 40s was referred for the surgical evaluation of asymmetric mandibular deficiency with secondary deformities of the maxilla. A longstanding flattening of the left condylar head with a loss of posterior facial height was felt to be responsible. There were no current symptoms of temporomandibular disorders, and the condyle was felt to be stable. The patient had an asymmetric Class II excess overjet anterior open-bite malocclusion. An orthodontic and surgical approach was selected. With orthodontic (dental) decompensation complete, surgery included maxillary Le Fort I osteotomy in segments (arch expansion and cant improvement); bilateral sagittal split ramus osteotomies (minimal horizontal advancement and asymmetry improvement); osseous genioplasty (horizontal advancement); redo septoplasty; and neck liposuction. During surgery, a "bad split" was appreciated on the left side. Nevertheless, the ramus osteotomy was secured with three bicortical screws. Radiographs the day after surgery confirmed a separate condylar component in the left ramus region. Posteroanterior and lateral cephalometric and Panorex radiographs were again taken 10 days after surgery, and these were suggestive of adequate segment approximation. At 5 weeks after surgery, left lateral mandibular facial swelling with mild tenderness persisted. At 8 weeks after surgery, a left posterior mandibular vestibular fistula was appreciated. In the operating room, the wound was opened to remove a buccal shelf and coronoid fragment as well as the fixation screws. Cultures confirmed *Escherichia coli, Enterobacter,* alpha-hemolytic *Streptococcus,* and *Candida.* Oral antibiotics were initiated, including clindamycin, ciprofloxacin, and Diflucan. At 11 weeks after surgery, there was a functional occlusion with good vertical mouth opening and a mild shift to the left. At 9 months after surgery, facial morphology and occlusion remained acceptable, and the orthodontic appliances were removed. **A,** Facial and occlusal views with orthodontics in progress. **B,** Profile and lateral cephalometric views before surgery.

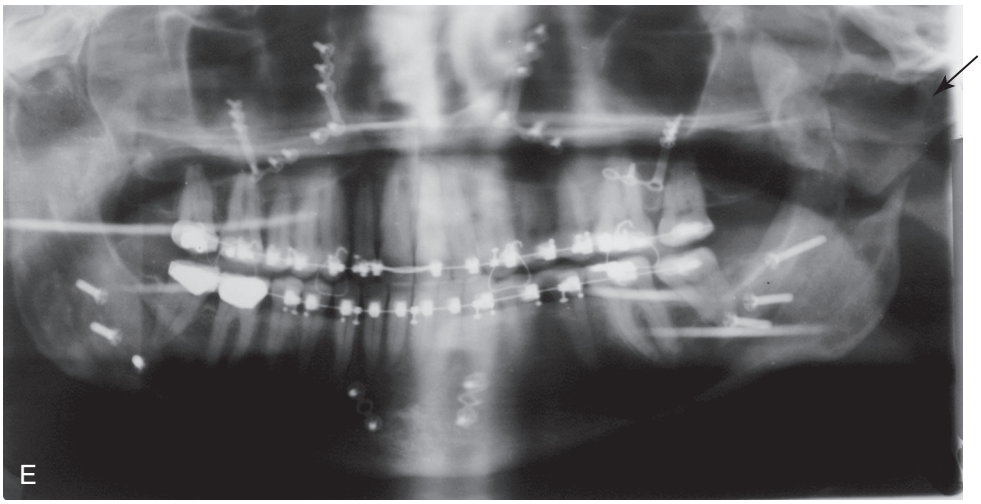
Continued



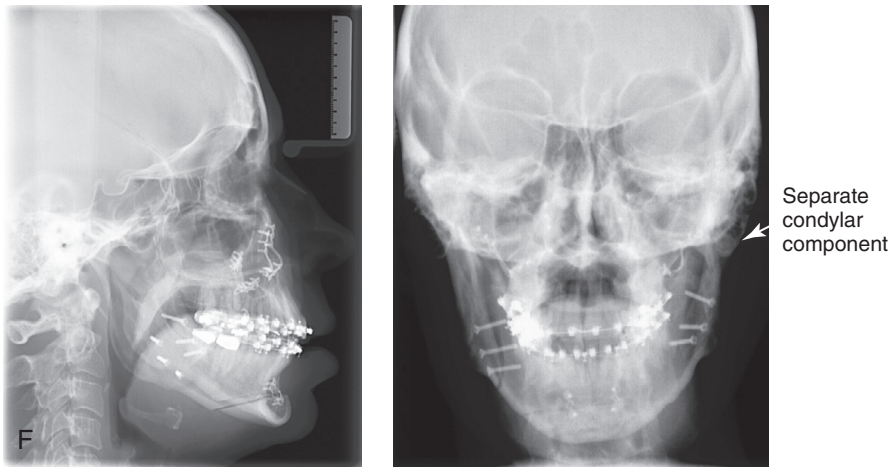
Mandibular deficiency with shift to left



• **Figure 16-9, cont'd** **C,** Panorex radiograph before surgery that indicates longstanding left condylar head flattening with an intact cortical rim. There is mandibular deficiency and a shift to the left. **D,** Articulated dental casts that indicate analytic model planning.



1 day postoperative panorex

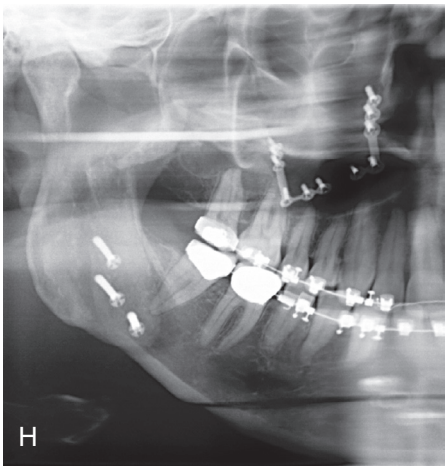


10 day postoperative lateral and PA cephalometric radiographs

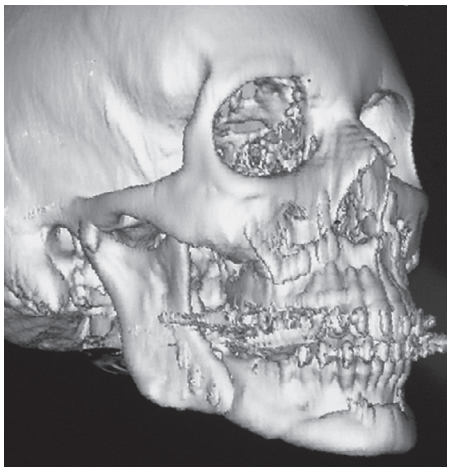
Before and 5 weeks after orthognathic surgery

• **Figure 16-9, cont'd** **E,** Panorex radiograph 1 day after surgery suggests a separate condylar component from the left ramus. **F,** Lateral and posteroanterior cephalometric radiographs taken 10 days after surgery indicate the same. **G,** Facial views before and 5 weeks after surgery. Persistent left side facial swelling is appreciated.

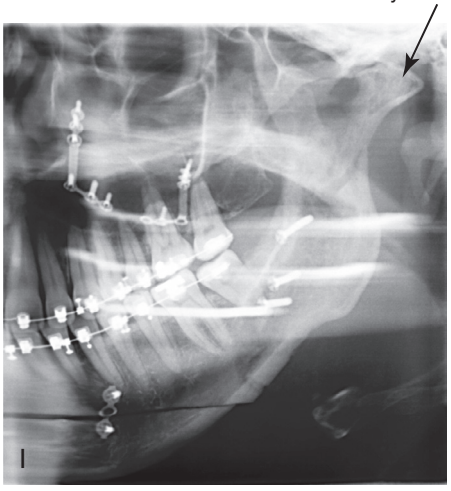
Continued



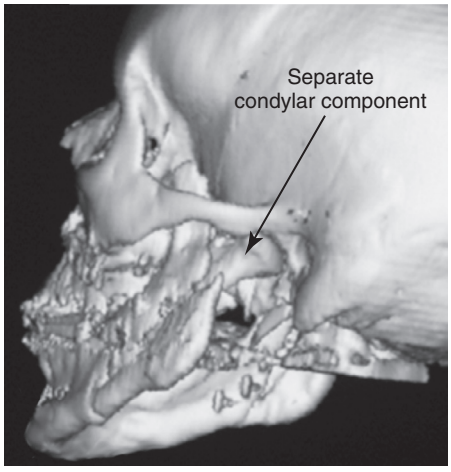
5 weeks postoperative panorex



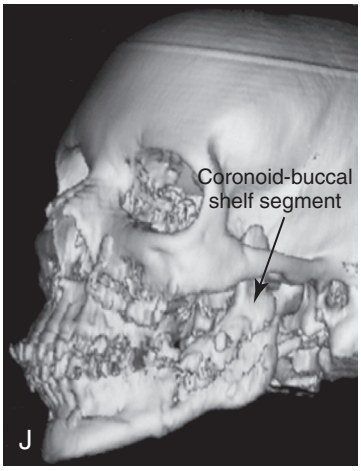
Right side - "good" split



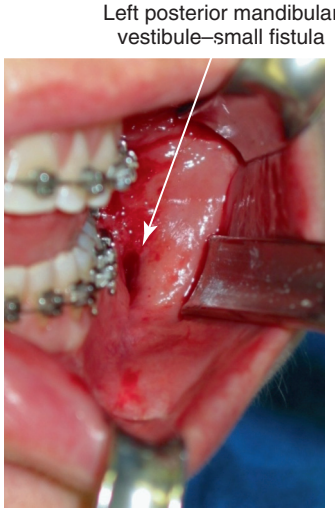
5 weeks postoperative panorex



Left side - "bad" split



8 weeks postoperative CT



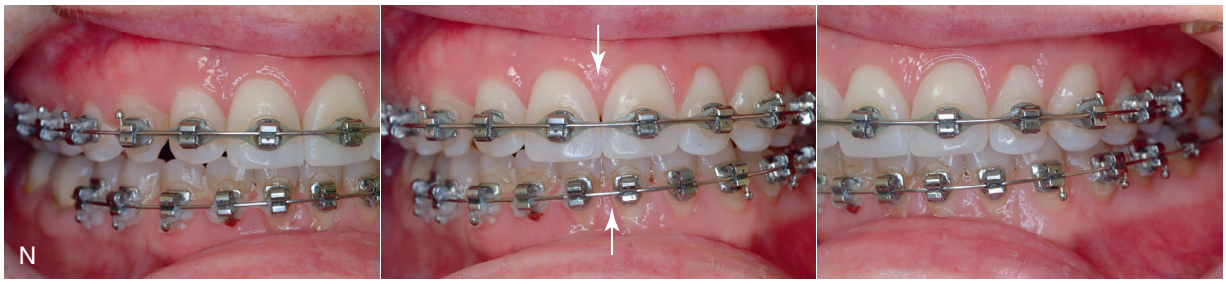
Coronoid-buccal
shelf segment
sequestrectomy
8 weeks postop

• **Figure 16-9, cont'd H,** Panorex and computed tomography scan views of the right ramus 5 weeks after surgery. **I,** Panorex and computed tomography scan views of the left ramus region 5 weeks after surgery confirm a "bad split" that demonstrates the expected anatomy. **J,** Left ramus computed tomography scan, left intraoral vestibular view, and sequestrectomy specimen with removed fixation screws.

• **Figure 16-9, cont'd K,** Frontal views in repose before and 11 weeks after surgery. **L,** Left facial oblique views before and then 11 weeks after surgery. **M,** Left profile views before and 11 weeks after surgery.

Continued

Pre surgery



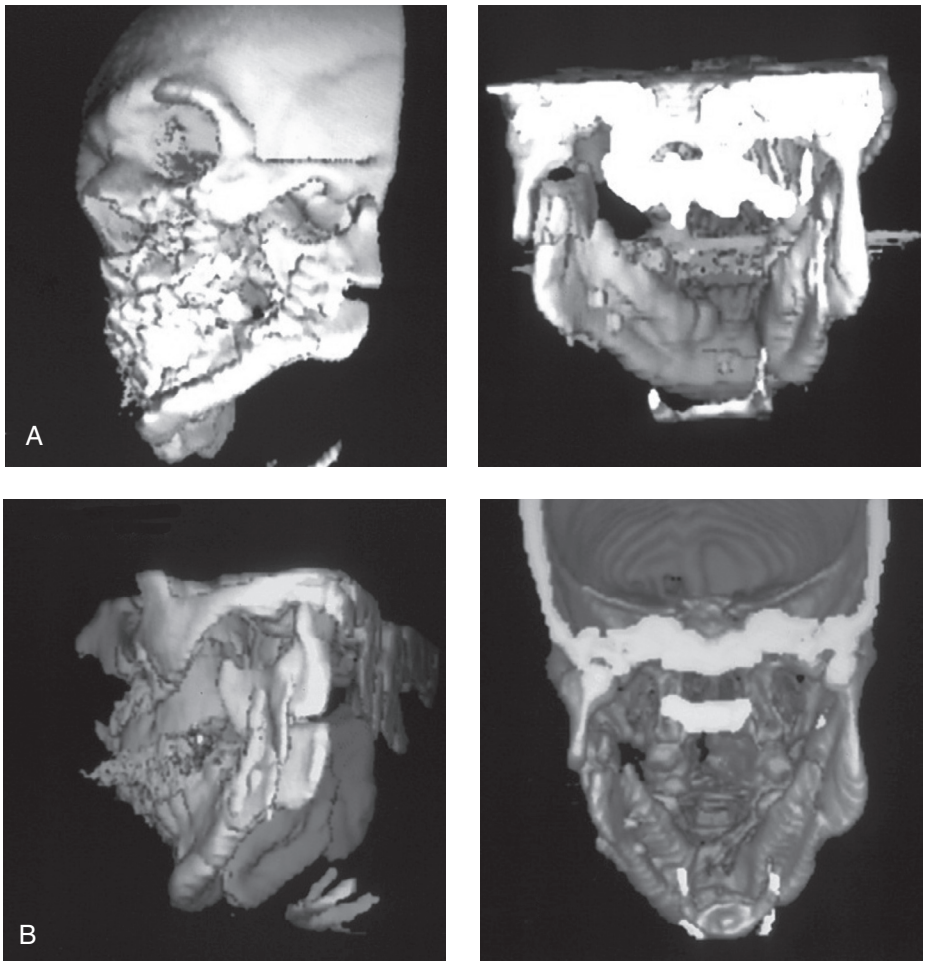
After initial healing

9 months after surgery and finishing orthodontics

• **Figure 16-9, cont'd N,** Occlusal views before surgery and then 11 weeks postoperatively. **O,** Nine months after the completion of treatment, frontal facial and occlusal views demonstrate the results.

When the condyle separates as a third piece, intraoperative decisions will affect the postoperative recovery and occlusion (see Fig. 16-9). From the perspective of the occlusion, if the condyle or the medial pole remains with the distal segment, it will be as if no osteotomy on the ipsilateral side of the mandible was carried out. If no further action is taken, an intraoperative malocclusion should be recognized. To fully complete the ramus osteotomy, the surgeon must then separate (i.e., osteotomize) the condyle from the distal segment (i.e., create a third piece). The osteotomy should be carried out, with as much ramus left on the condylar segment (i.e., the third piece) as possible. Even when this type of bad split occurs, unless significant mandibular advancement is required for the correction of the jaw deformity, the successful healing of the segments will generally occur (see Figs. 16-9 and 16-10).

If the condyle separates (i.e., if a third piece is created) "high up," the result is similar to that of a high condylar neck fracture. There will not be a stable posterior stop unless the surgeon is able to carry out plate and screw fixation of the condylar component (i.e., the third piece) to the rest of the proximal segment. This may not be practical, and it will depend on the morphology of the condylar piece. The incidence of postoperative malocclusion will also be



• **Figure 16-10** A teenager with a cleft jaw deformity underwent orthognathic surgery that included a maxillary Le Fort I osteotomy in segments; bilateral sagittal split osteotomies of the mandible; and an osseous genioplasty. The sagittal split on the left side separated unfavorably. The proximal segment includes a condyle coronoid upper ramus component; an angle ramus component; and a buccal shelf component. The three components of the proximal segment were secured together and fixed to the distal segment with a combination of titanium plates and screws. Fortunately, the reduction was adequate, so healing occurred with favorable facial aesthetics, occlusion, and temporomandibular joint function. **A** and **B,** Three-dimensional computed tomography scan views of a "bad split" fixed into acceptable alignment during orthognathic surgery.

dependent on the directional change planned for the distal mandible. If the mandible is to be advanced a significant amount, there is a greater probability that the minimally stabilized condylar segment (i.e., the third piece) will not heal in an ideal location relative to the residual proximal and distal segments. Alternatively, if the mandible is to remain relatively neutral or is set back, there is a higher probability that the condylar segment will heal to the residual proximal and distal segments in a favorable way (see Fig. 16-9). As long as healing is associated with adequate mobility (i.e., mouth opening), then any residual malocclusion can typically be managed 6 to 12 months later with redo SROs, if desired (see Chapter 35).

Unfortunately, there is no consensus with regard to what combination of factors predisposes an individual to a bad split. The literature shows the incidence of bad splits to vary from 1% to 23%. A buccal plate fracture of the proximal segment, a lingual plate fracture of the distal segment, and a coronoid process fracture of the proximal segment are the most commonly reported "bad splits". Authors who consider these types of less than ideal osteotomies to be bad splits provide an explanation for the high incidence reported in some series. As previously stated, the majority of less than ideal separations (fractures) at the time of SSRO result in fixation challenges, most are not responsible for long-term negative sequelae.

The occurrence of a bad split is greatly reduced by keeping the medial osteotomy "short (not to far back) and low" (close to the occlusal surface of the mandibular molars) (Fig. 16-11). Using this technique a bad split as described should be a rare event (see Chapter 15).

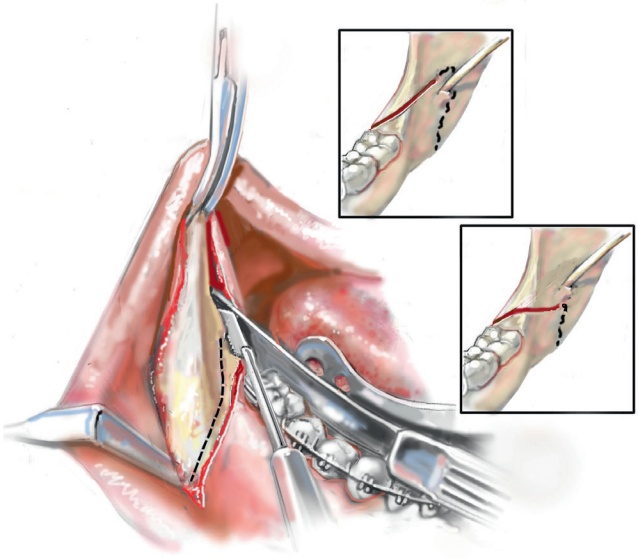
Veras and colleagues evaluated risk factors and the functional and radiographic long-term results of a bad split.[350] They also completed detailed temporomandibular joint

examinations on patients with bad splits. Interestingly, they did not find the simultaneous extraction of a mandibular third molar in conjunction with an SSRO to be a risk factor for the development of a bad split. This is consistent with the research reported by Kriwalsky and colleagues,[174] Precious and colleagues,[268] and others (see discussion earlier in this chapter).

Inferior Alveolar Nerve and Mental Nerve Injury

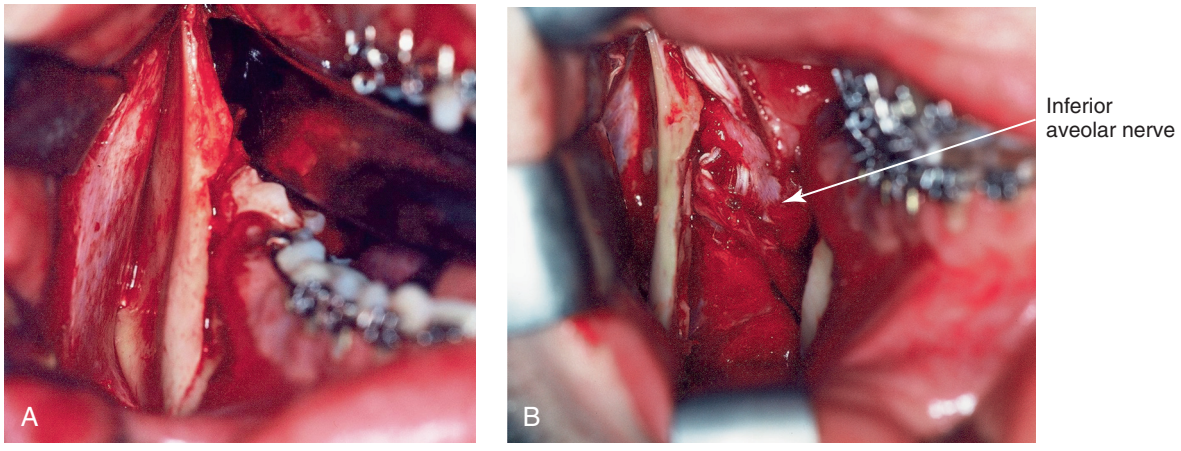
Facial sensibility of the area affected by either an SSRO or a chin osteotomy occurs through the *mandibular nerve,* which enters the mandibular foramen at the medial surface of the ramus.* This area is adjacent to and in front of the inferior alveolar artery and vein and just inferior to the occlusal plane of the mandibular molar teeth. The mandibular nerve becomes the *IAN,* and it runs with its vessels within the canal and supplies sensation to the three molars and the two premolar teeth. It then divides into two terminal branches. The *incisive branch* supplies sensation to the canine and incisor teeth, whereas the *mental nerve* exits from the foramen to supply sensibility to the chin, the lower lip mucosa and skin, and the adjacent gingiva. Damage to the IAN during either the ramus osteotomies or when completing an oblique osteotomy of the chin may occur. During SSRO of the mandible, the IAN can be damaged by a bur on a rotary drill, a saw blade on a reciprocating saw, an osteotome, or during the placement of plate and screw fixation (Fig. 16-12).

Posnick and colleagues completed a study to clarify normal chin, lower lip, and gingival sensibility in adolescents and then compared the normal values with sensibility measurements in three distinct groups of adolescents 1 year after these individuals had undergone mandibular osteotomy.[261,263] One hundred fifteen subjects (230 mental nerves) were included in the study and divided into four groups. The first group included 67 individuals (Group I: n = 134 nerves; mean age, 18 years) who were undergoing orthodontic treatment; none had undergone orthognathic surgery. This group served as controls to determine the normal sensibility values of the chin, the lower lip, and the gingiva in adolescents. The remaining three groups were made up of adolescents who had previously undergone



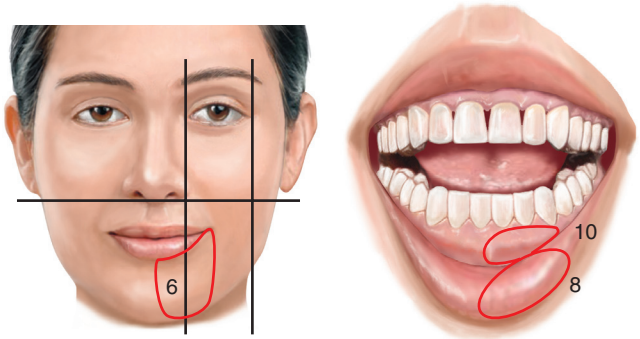
• **Figure 16-11** Illustration of this author's preferred "short and low" medial osteotomy of the sagittal split to minimize an incidence of a "bad split."

*References 3, 5, 6, 10, 12, 17, 18, 21, 37, 45, 51, 52, 55, 57, 65, 66, 69, 71, 88, 97-102, 106, 109, 112, 114, 121, 141-144, 160, 161, 166, 169, 193, 195, 196, 197, 227, 229-231, 234, 239, 258, 263, 265, 275, 277, 295, 296, 299, 301, 305, 327, 329-334, 344, 351, 354, 358-361, 369, 370, 368, 374, 376, 378, 380.



• **Figure 16-12** Intraoperative views of a right sagittal split ramus osteotomy. The demonstration of cortical cuts completed **A,** before and **B,** after the splitting (separation) and mobilization of the segments. The inferior alveolar nerve is visualized in the surgical field.

BSSRO of the mandible (Group II: n = 14 nerves; mean age, 19 years), osteoplastic genioplasty (Group III: n = 40 nerves; mean age, 19 years), or a combination of BSSRO and osteoplastic genioplasty (Group IV: n = 42 nerves; mean age, 19 years). Testing of Groups II, III, and IV was performed 1 year after the patients had undergone the indicated mandibular procedures. At the time of the 1-year postoperative examination, each patient was also asked to detail subjective impressions of current altered sensibility in the mandibular vestibular mucosa, the gingiva, the lower lip, and the chin region. The subjective questioning and objective testing were carried out by an occupational therapist who was trained in facial sensibility testing and familiar with the surgical procedures carried out. Fixed and reproducible coordinates in the area corresponding to the mental nerve were used for objective testing (Fig. 16-13). Three sensory modalities were tested at each coordinate for each involved nerve. Static two-point discrimination was measured with a MacKinnon–Dellon Disk-Criminator (Richardson Productions, Inc, Frankfort, Ill); vibratory thresholds were determined with a biothesiometer; and cutaneous pressure thresholds were measured with a set of Semmes–Weinstein monofilaments. Sensibility values at 1 year after operation for Groups II, III, and IV were compared with the mean normal control values established for Group I. A subjective awareness of alterations in the sensibility of the chin, lip, or gingiva region 1 year after BSSRO was reported in 2 patients in Group II (29%), in 2 patients in Group III (10%), and in 14 patients in Group IV (67%). Interestingly, only 2 of these 18 patients considered the residual sensory loss problematic. The mean objective sensibility values of the three sensory modalities tested were highest among patients from Group IV. Significant differences were found only between the mean two-point discrimination of Group IV patients as compared with the control group in the chin skin area. Thus, 1 year after BSSRO, only 29% of patients had residual subjective awareness of sensory alteration along the distribution of the mental nerve. After an osseous genioplasty, there was only a 10% incidence of long-term subjective diminished sensibility. One year after a combination of BSSRO and osteoplastic genioplasty, 67% of patients experienced varying degrees of residual subjective loss of sensation in the chin and lip region.



• **Figure 16-13** Illustration of sites for sensibility testing in the inferior alveolar and mental nerve distribution. In a study carried out by Posnick and colleagues, coordinates 6, 8, and 10 located within each of the circled areas were tested bilaterally.

Espelan and colleagues completed a 3-year postoperative survey of patients who had undergone SSRO (N = 583 subjects) and found that that 36.8% of the study patients reported impaired sensation over the long term.[96A] Westermark and colleagues completed a retrospective study of patients who had undergone SSRO and concluded that nearly 100% of the patients experienced impaired sensation and sensory function.[358] This is also consistent with the findings of **Essick and colleagues**[97] and **Teerijoki-Oksa and colleagues.**[332,333] The exact reasons why, over time, most patients experience only a degree of simple loss of sensation—whereas some continue to experience active sensations that are not normally present and an even smaller percentage of those with active sensations subjectively describe them as uncomfortable or painful (dysesthesia)—are not known. **Zuniga and colleagues** reported a 5% incidence of dysesthesia of the IAN after SSRO.[380,381]

Turvey reported a 5.5% incidence of IAN lacerations at the time of SSRO.[343] **Van Merkesteyn and colleagues** documented a visible injury in the IAN at the time of SSRO in 7 out of 124 patients (6%).[347] The term *traumatic neuroma* is used to describe the formation of a bulbous mass that develops at the end of a proximal nerve stump after partial or complete nerve transection. The lesion represents an exaggerated relative hyperplasia (i.e., healing) response to a nerve injury. Neuroma symptoms range from paresthesia to severe paroxysmal or persistent pain. Despite the incidence of partial or complete transection of the IAN at the time of SSRO, few cases of traumatic neuroma have been documented. This is likely because the healing transected nerve remains deep in the medullary cavity of the mandible. It is known that a healing nerve stump within bone is rarely painful. The few reported cases of neuroma after SSRO have confirmed the location to be within the soft tissues along the inferior border of the mandible.[120,121]

Essick and colleagues completed a clinical trial that determined that the magnitude and duration of the patient-reported *burden of altered sensation after SSRO* was lessened when facial sensory retraining exercises were performed in conjunction with standard mouth-opening exercises as compared with the group of patients who performed only mouth-opening exercises.[97] The authors defined the term *burden* as the person's reporting of subjective impressions of numbness or unusual sensations (i.e., dysesthesia) in the face, the perioral region, and or oral region. One interpretation of the study findings is that patients who perform facial sensory retraining become more introspective with regard to their altered sensation, which leads to a greater acceptance of their sensory loss by 6 months after surgery. Consistent with this interpretation was the finding that sensory retrained patients initially viewed numbness and decreased sensitivity as more problematic early during recovery but as less problematic later during recovery than did the group of patients who did not undergo retraining.[258]

The retraining sessions were held at 1 week, 1 month, and 3 months after surgery. The levels of instruction for the sensory retraining were designed to increasingly challenge patients' sensory discrimination abilities in a manner similar to that of the early and late phases of sensory retraining commonly used after injuries to the sensory nerves of the hand.[120,121] Through sensory retraining, individuals learn to discriminate moving touch from non-moving touch; the orientation of moving touch; and the direction of moving touch (see Chapter 11). **Poort and colleagues** completed a literature review of methods that are used to test for sensory loss and recovery after IAN injury and recommended that clinicians and researchers use the following two measurement tools: 1) a light touch test with Semmes–Wienstein monofilaments for grading sensation; and 2) a visual analog scale–based questionnaire to evaluate the individual's subjective sensibility.[261A] If clinicians were to adopt consistent measuring techniques, improved communication with regard to sensory loss and level of recovery after interventions would likely follow.

A dilemma that surgeons continue to face is what to do in the operating room when the laceration of the IAN is witnessed. The question raised asks if there is any significant long-term advantage for the patient to undergo either primary or delayed micro repair of the lacerated IAN. Although the concept of the immediate repair of a witnessed IAN transection is appealing, the practicality of this maneuver and the patient's long-term improved results (i.e., greater sensory return and decreased dysesthesia) have not been statistically confirmed. The increased operative time and the potential risks of losing focus on the primary orthognathic objectives must also be considered. **Tay and colleagues** reported four cases of immediate micro-neural repair of an IAN that was transected during SSRO.[334] This represented 4 out of 260 (2%) SSROs completed at their center. One of the four patients who sustained IAN laceration was apparently lost to follow up. Interestingly, the location of IAN transection in the remaining three study patients was at the anterior and inferior aspect of the osteotomy along the buccal shelf between the first and second molars; this is a recognized "danger zone" for IAN transection during SSRO. When there is a moderate to severe degree of mandibular hypoplasia with the need for buccal shelf extension of the proximal segment to achieve successful approximation of the bone after advancement, this risk may increase. In the study by Tay and colleagues, the three study patients underwent immediate repair of the IAN (i.e., neurorrhaphy) by a trained microsurgeon and then received follow for 1 year.[334] The microsurgeon's technique included the complete surgical release of the nerve from the mandibular foramen to the mental foramen followed by approximation and repair with the use of 6-0 microfilament suture under microscope magnification. The increased time of the surgery was approximately 3 to 4 hours. Although the authors felt favorable regarding the outcomes of their patients, the level of sensory return that was documented was not proven to be significantly better than expected after the simple approximation of the nerve endings within the ramus of the mandible.

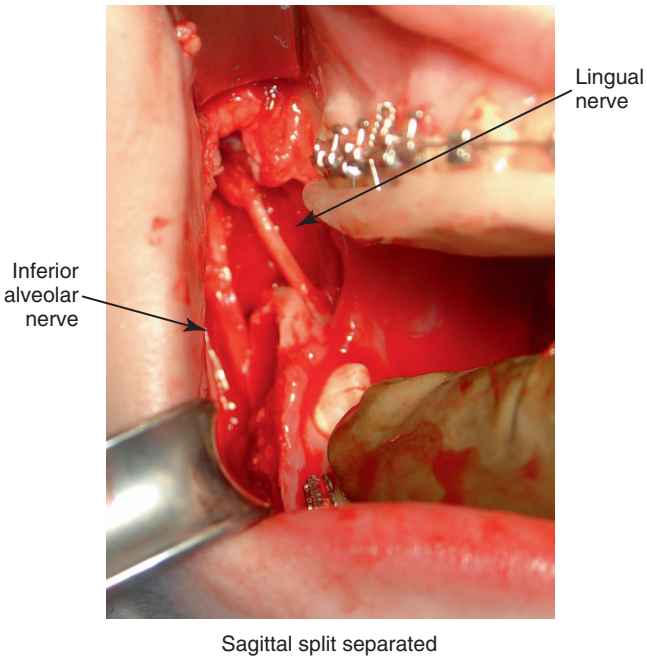
NOTE: This author believes that a controlled study documenting significantly better sensory recovery after direct micro-neural repair that occurs via the simple approximation of the cut edges of the IAN in the setting of an SRO would be required to justify the added time and risk of the repair.

Lingual Nerve Injury

The incidence of injury to the lingual nerve during SSRO is not precisely known, but it is assumed to be rare.[35,132,145,147,279,289-291,327] **Becelli and colleagues** completed a retrospective analysis of complications after SSRO and found a 0.6% rate of lingual nerve dysfunction among 482 osteotomy patients during the first postoperative week. The authors documented complete resolution of this injury after 12 months of follow up in the study group. Interestingly, lingual nerve injury at the time of the removal of mandibular wisdom teeth as an isolated procedure is also estimated to be in the same range (i.e., <1%).[260] When a lingual nerve injury does occur with SSRO, it likely occurs during subperiosteal dissection along the medial ramus; when using a rotary drill (e.g., with a Lindeman bur); or when using the reciprocating saw (e.g., during a medial ramus cortical cut). It may also occur with use of the electrocautery to control bleeding, during screw fixation, or when closing the wound if the lingual nerve is caught within the suture tie (Fig. 16-14).

Pogrel and Hung Le completed a cadaver study that demonstrated that different modalities (e.g., sharp scalpel laceration, fissure bur injury, retraction stretch injury) produced different types of nerve injury, both clinically and histologically.[261] The clinical recovery of the injured nerve is believed to be primarily related to the type of injury sustained. For this reason, the treatment recommended should also vary in accordance with the type of injury sustained. Data reveal that 57% of lingual nerve injuries were unknown to the surgeon at the time; therefore, the type of injury could not be accurately ascertained.[86]

Unfortunately, many of the published lingual nerve injury studies are retrospective case series or questionnaire surveys.[260] The studies also frequently combine both lingual and IAN injuries. The literature recommends that observed injuries and those involving a foreign object causing compression or laceration (e.g., endodontic filling material, bone fragment, broken instrument) should be treated immediately. Caution is warranted when interpreting two-dimensional radiographic views when making decisions about impingement on the nerve by a foreign object (e.g., a fixation screw). Three-dimensional computed tomography scans may be useful to help make the determination.



• **Figure 16-14** A completed right sagittal split ramus osteotomy. The separation of the segments demonstrates both the inferior alveolar and lingual nerves. The lingual nerve is only occasionally visualized in the surgical field.

Patients with severe hypoesthesia who are demonstrating no sensory improvement at approximately 3 months after treatment may be surgical candidates. Those with at least some documented continuous sensory improvement are best managed conservatively.

Susarla and colleagues used a retrospective cohort study design to address whether the early repair of a lingual nerve injury improves functional sensory recovery.[324] The study sample was composed of 64 subjects with lingual nerve injuries who underwent repair with a mean time between injury and repair of 153 days (31 to 1606 days). Twenty-two percent of the subjects underwent early repair (i.e., <90 days after injury). The nerves were repaired either through direct suture (77%) or surgical exploration with decompressive neurolysis (23%). Ninety-three percent of the study subjects in the early repair group achieved functional sensory recovery within 1 year as compared with only 63% in the late repair group. Although early repair shows statistical benefits for the achievement of functional sensory recovery, it must also be assumed that a percentage of those individuals who submitted to early repair would have gone on to achieve adequate recovery through the natural course of events. A randomized clinical trial would be required to clarify any clear advantage of early repair.

A study by **Bagheri and colleagues** suggests that, for some individuals, secondary repair of a lingual nerve or IAN injury will result in the regaining of acceptable sensory function as classified by the Medical Research Council Scale.[17] However, at least some of those who are undergoing secondary repair will show little improvement, and the prospect of deterioration is not insignificant. The evaluation and management of both lingual and IAN dysfunction after an SSRO remains a challenging clinical dilemma. The specific etiology and incidence of persistent sensory deficit and neuropathic pain after SSRO remains poorly understood. Nevertheless, prudent care suggests that, when a lingual nerve injury occurs during a ramus osteotomy, the surgeon is obliged to offer evaluation by a knowledgeable peripheral nerve surgeon within 3 to 6 months. This will ensure that the injured individual receives the information needed to make a personal decision about how to proceed.

Facial Nerve Injury

Reports of injury to other than sensory nerves are primarily restricted to the facial nerve, and these mainly occur during mandibular procedures.[60,68,77,79,226,272,287] A retrospective study conducted by de Vries and colleagues after SSRO (N = 1747) found an incidence of seventh nerve palsy of 0.26% (9 patients).[83] Choi and colleagues found an incidence of facial nerve palsy of 0.1% among 3105 patients who underwent sagittal split ramus osteotomies.[60]

Significant Bleeding

In current practice, significant bleeding is rarely encountered at the time of SSRO.[16,64,177,184,185,191,207,253,288,303,306,326,372] In 1972, a 38% incidence of major intraoperative hemorrhage was reported; in 2005, an incidence of only 1% was reported.[184,257] The vessels to consider include the *maxillary artery* and its branches; the *retromandibular vein;* and the *facial artery and vein.* Behrman reported about two patients with excessive bleeding in which he carried out ligation of the external carotid artery, although doing so was relatively ineffective for controlling the hemorrhage.[23] In 1985, Turvey reported the incidence of troublesome hemorrhage to be 1.2%; the inferior alveolar and facial arteries were the sources.[322] Acebal-Bianco and colleagues reported about the laceration of the facial artery during SSRO in two patients with successful management.[2] Van Merkesteyn and colleagues discussed difficult-to-control bleeding from the inferior alveolar and facial arteries in two patients.[257] Teltzrow and colleagues reported bleeding from the retromandibular vein to be the most common source of severe hemorrhage after SSRO.[335]

The occurrence of a *false aneurysm* (i.e., pseudoaneurysm or traumatic aneurysm) after an elective mandibular ramus osteotomy is uncommon. Precious and colleagues reported three cases of false aneurysm after SSRO and reviewed the pertinent literature.[269] A false aneurysm will present as a delayed phenomenon, usually 1 to 16 weeks after injury. These conditions are thought to result from a partial transaction of an artery (i.e., a facial artery) that leads to the extravasation of blood under pressure into the surrounding soft tissues. The resultant hematoma organizes, and this is followed by fibrous pseudocapsule and

then endothelialization. A pulsatile and expanding mass characterizes a false aneurysm. The difficulty of this situation is related to the making of an accurate and timely diagnosis. The treatment approaches include primary surgical resection, arterial embolization, or arterialization followed by surgical resection. Superselective arterial embolization alone is the current preferred approach, whenever feasible.

The surgeon's best protections against these unusual vascular events when carrying out a sagittal split ramus osteotomy remain the following: 1) careful patient selection 2) meticulous technique 3) good intraoperative assistance 4) protective instrument placement and 5) fine control of power tools. When severe hemorrhage does occur after SSRO, interventional radiology procedures versus the direct ligation of the involved vessels must be considered.

Risks and Complications Specific to Le Fort I Osteotomy

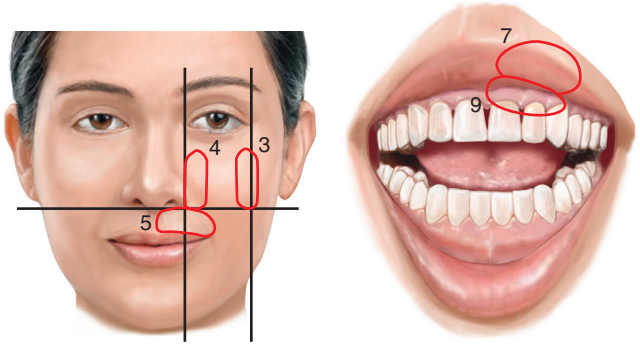
Infraorbital Nerve Injury

Sensory innervation of the area affected by the Le Fort I osteotomy is mainly through the maxillary division of the trigeminal nerve. During the Le Fort I osteotomy, the three superior alveolar nerves on each side are transected as part of the osteotomy, and the terminal labial branches of the infraorbital nerve are transected as part of the mucosal incision. After the infraorbital nerve emerges from the infraorbital foramen, it is also subject to trauma. A degree of injury to the infraorbital nerve at the time of Le Fort I osteotomy that results in temporary paresthesia is expected, whereas permanent dysesthesia of the cheek skin, the upper lip, the palate mucosa, and the gingiva is thought to be less common.* The nerve injury may result from compression or stretching, and direct injury may result from saw blades, burs, or surgical instruments (Fig. 16-15). The patient's age and gender will also be factor when considering nerve recovery and the occurrence of dysesthesia.

Posnick and colleagues objectively measured facial sensibility in adolescents (with and without cleft lip and palate) 1 year after Le Fort I osteotomy.[263] Fifty-nine individuals (118 infraorbital nerves) were included in the study and divided into three groups. The first group (N = 30) included subjects with unilateral cleft lip and palate (mean age, 18 years; with jaw deformity). The second group (N = 12) included subjects with bilateral cleft lip and palate (mean age, 19 years; with jaw deformity). All of these patients had undergone cleft lip and palate repair earlier during their lives; none had an associated craniofacial syndrome, developmental delay, or systemic neurologic impairment. The third group (N = 17) consisted of subjects without clefts or syndromes (mean age, 19 years) but with a developmental jaw deformity. Facial sensibility testing was conducted in the three groups 1 year after the Le Fort I osteotomy. Each patient was also asked about his or her subjective impressions of altered sensibility in the maxillary vestibular mucosa, the gingiva, the upper lip, or the cheek region at the 1-year examination. Previously defined fixed and reproducible coordinates were used for objective testing in the area that corresponds with the infraorbital nerve (Fig. 16-16).[267] Three sensory modalities were tested bilaterally at each coordinate. Static two-point discrimination was measured with a MacKinnon–Dellon Disk-Criminator. Vibratory thresholds were determined with the biothesiometer, and cutaneous pressure thresholds were measured with a set of Semmes–Weinstein monofilaments. The data was compared with the mean normal values that had been previously established for adolescents with and without cleft lip and palate before any orthognathic surgery.

The data confirmed that, at 1 year after Le Fort I osteotomy, none of the patients reported any subjective awareness of alterations in the sensibility of the maxillary vestibular mucosa, upper lip, or cheek regions. No significant differences were found in the mean postoperative values at each coordinate among the three groups in any of the sensory modalities tested. No significant differences were found

• **Figure 16-15** Intraoperative view after a circumvestibular incision for exposure to complete a Le Fort I osteotomy. The infraorbital nerve is visualized.



• **Figure 16-16** Illustration of the infraorbital nerve sensibility distribution. Coordinates 3, 4, 5, 7, and 9 are fixed sites that were used by Posnick and colleagues for sensibility testing.

*References 7, 52, 57, 71, 97-99, 120, 154, 160, 161, 261, 264, 265, 267, 273, 295, 323, 380, 381.

between the preoperative and postoperative pressure and vibratory threshold values in each patient group. The mean postoperative static two-point discrimination values were higher than the preoperative values in all areas tested and in all patient groups, but significant differences (i.e., $P < .05$) were found only in the anterior cheek skin area (coordinate 3) in the patients with unilateral cleft lip and palate on both the cleft and non-cleft sides. The study carried out by Posnick and colleagues confirmed the clinical impression that permanent functional sensory alteration of the soft tissue supplied by the infraorbital nerve is rare among teenagers and young adults after Le Fort I osteotomy.

Thygesen and colleagues recently looked at risk factors for paresthesia and dysesthesia of the infraorbital nerve after Le Fort I ostoeomy.[337] Their results are somewhat different from those of Posnick and colleagues. The authors found objective changes in somatosensory function 12 months after Le Fort I osteotomy in 7% to 60% of patients, depending on the site at which the sensory measurement was carried out (i.e., skin, gingiva, or palatal mucosa). The authors did not report instances of significant dysesthesia associated with the sensory loss in any patient. Despite a degree of subjective sensory loss at 12 months after surgery, all of the study patients were satisfied with the results of their operation, with 100% saying that they would do it again.

Visual Disturbances

Blindness

Decreased visual acuity (blindness) is a rare complication of Le Fort I osteotomy.* Although the exact mechanism of injury to the optic nerve has not been clarified, atypical fracture patterns that extend upward to the skull base from the pterygomaxillary (PM) separation at the time of downfracture have been implicated.[180,309,340,366] Girotto and colleagues reported on three cases of visual loss after elective standard Le Fort I osteotomy.[115] All three cases were presumed to be straightforward and were performed by experienced surgeons. The authors hypothesized that the unpredictable nature of the PM dysjunction may result in the extension of fracture to the skull base or generate deforming forces to the optic canal, with compression and pressure placed on the optic nerve. Lanigan reviewed the literature up through 1993 and reported two cases of blindness after Le Fort I osteotomy.[188] Bendor-Samuel and colleagues reported one additional case of blindness.[28] Lo and colleagues reported two cases of monocular blindness in 94 consecutive cleft palate patients who underwent mixed dentition maxillary (Le Fort I) osteotomy with osteodistraction with the use of the RED II Distraction System (KLS Martin, Mühlheim, Germany) during a 4-year period.[154] Although the exact etiology could not be clarified by Lo and colleagues, they stated that the PM disjunctions were performed at a higher level to avoid tooth bud injury in their patients with cleft palates during the mixed dentition. Therefore, the osteotomies were closer to the skull base and the orbit. The authors also speculated that the hard palate in the tuberosity region and the PM junction may have been more sclerotic as a result of the repaired cleft palate, thereby making precise PM disjunction less predictable.

Of the nine reported cases of blindness after Le Fort I osteotomy, five were of unknown cause, one was due to arterial aneurysm, two were attributed to propagation of the PM disjunction fracture through the skull base, and one was the result of the hypoperfusion of the optic nerve. There was no recovery in three cases, light perception returned in three, hand movements were visible in one, fingers could not be counted in one, and visual acuity showed much improvement in one. Interestingly, a majority of the reported cases of monocular blindness were in individuals born with cleft lip and palate. These patients had all undergone primary lip and palate repair, and several had previously undergone unsuccessful Le Fort I osteotomies. This likely represents a higher than average risk group for the rare complication of monocular blindness.[194]

Extraocular Cranial Nerve Dysfunction

Newlands and colleagues identified five cases of abducens palsy, three cases of oculomotor palsy, and one case in which both palsies were present together.[129,135,199,237,307,356] Most reported cases of extraocular nerve dysfunction after Le Fort I osteotomy occur immediately after surgery, although it has been reported up to 5 days postoperatively.

Nasolacrimal Apparatus Dysfunction

Tearing dysfunction has been reported after Le Fort I osteotomy by a number of authors; this involves either a lack of or excessive tearing.[202,210,211,300,338] A lack of tearing is thought to have occurred as a result of damage to the greater petrosal or Vidian nerve, which could interrupt parasympathetic supply to the lacrimal gland. Excessive tearing is thought to be related to nasolacrimal duct damage.

Significant Bleeding

Vascular complications (i.e., excessive bleeding)—either immediate or delayed—after Le Fort I osteotomy are rare.* The reported cause of the delayed bleeding is generally a partial laceration of a vessel with an *aneurysm* or an *arteriovenous fistula formation* (i.e., false aneurysm). The confirmation of a traumatic vascular abnormality is best made by angiography. This condition most frequently occurs in the internal maxillary artery or in the internal carotid artery region. The presentation of bleeding from the nose and occasionally from the oral cavity early after surgery is the typical clinical event. When massive epistaxis occurs soon

*References 28, 31, 43, 54, 58, 59, 67, 70, 115, 148, 188, 198, 199, 274, 278, 286, 339.

*References 4, 15, 40, 41, 64, 74, 116, 123, 126, 134, 136, 140, 168, 181, 183, 185, 187, 189, 190, 204, 205, 219, 236, 240, 241, 280, 302, 306, 308, 310, 316.

after Le Fort I osteotomy, the differential diagnosis may include coagulopathy, bleeding from a partially resected inferior turbinate, or bleeding from a large artery (i.e., the internal maxillary artery). Angiography with the possible need for embolization should be considered. A frequent palliative (but temporary) treatment approach is the packing of the nose. This is likely to immediately control bleeding; however, over time, a false aneurysm may form, and rebleeding (i.e., recurrent epistaxis) is typical.

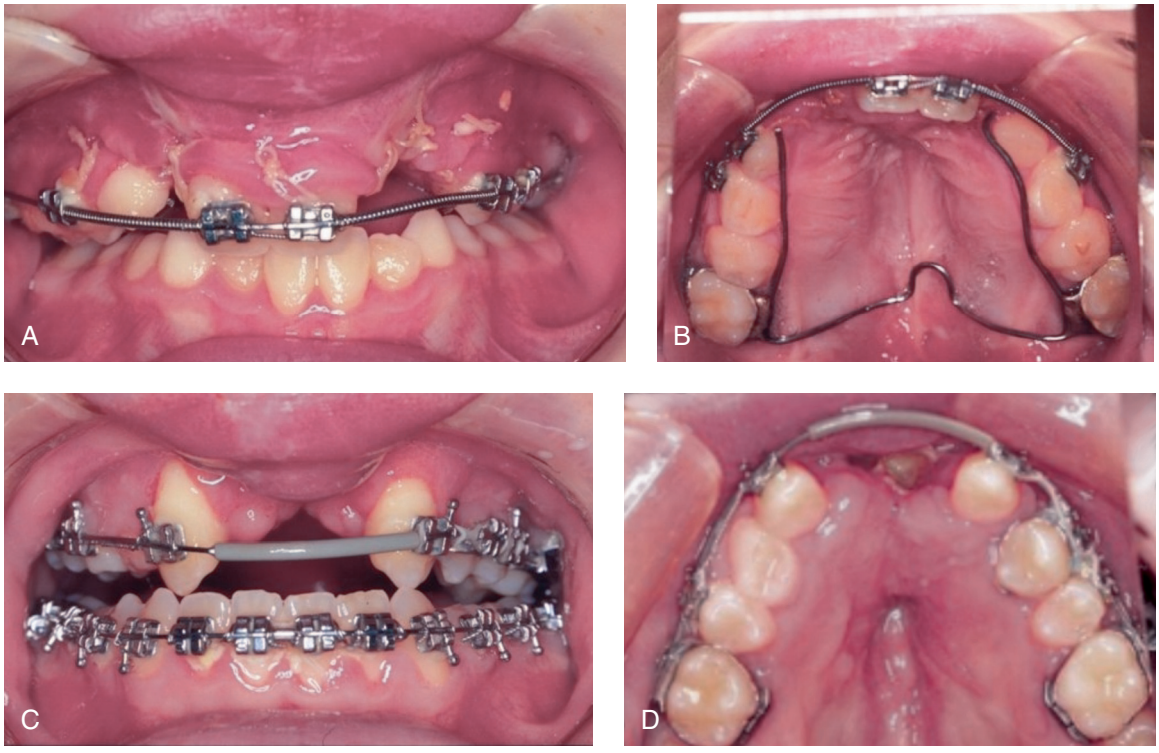
Procopio and colleagues described one patient (a 37-year-old woman who had undergone Le Fort I down-fracture) whose epistaxis occurred 2 hours after surgery and who was treated with anterior nasal packing.[270] Seven days later, she was taken back to surgery to reposition her maxilla for the treatment of malocclusion. No source of excessive bleeding was found. Further epistaxis occurred on days 21, 53, 63, 85, and 115. These episodes were treated with nasal packing, which resulted in resolution for short periods of time followed by rebleeding. On day 115, angiography and a computed tomography scan were completed. A false aneurysm of the sphenopalatine artery was identified and successfully embolized.

The standard *deliberate hypotensive anesthesia* techniques used during Le Fort I down-fracture can mask a partial laceration of the sphenopalatine or descending palatine artery. It is recommended that ligation or cauterization of the descending palatine artery be carried out when partial laceration occurs. It is also suggested that the orthognathic patient be allowed to emerge from hypotensive anesthesia before wound closure in an attempt to identify any major bleeding before the patient leaves the operating room.

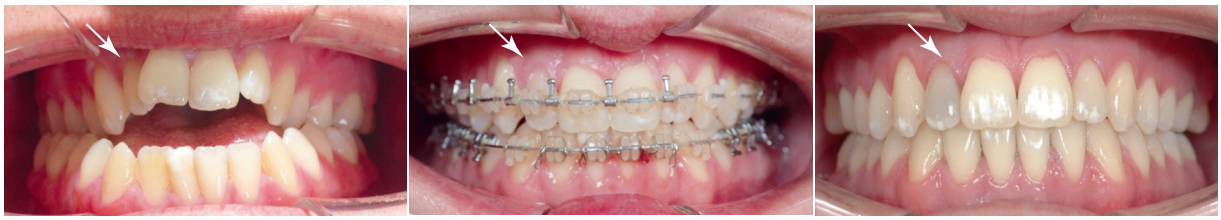
Aseptic Necrosis of Maxilla

A rare but serious complication of Le Fort I osteotomy is ischemic necrosis.[25,27,38,44,86,87,124,128,179,182,235,251,271,298,373] The severity of this postoperative complication is related to the degree of vascular compromise of the down-fractured maxillary segments. Reported sequelae of vascular compromise include infection, mucosa sloughing, periodontal defects, pulpal changes, malunion or nonunion, and the partial or complete loss of the maxilla and dentition (Figs. 16-17, 16-18, and 16-19).

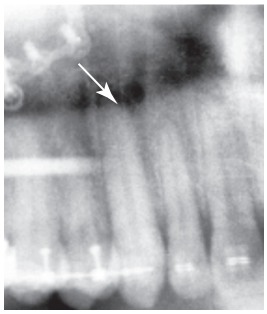
The biologic basis for maxillary (i.e., isolated segmental, down-fracture Le Fort I, and down-fracture Le Fort I with segmentation) osteotomies used in orthognathic surgery was extensively investigated in experimental animals by Bell and colleagues with the use of microangiographic and



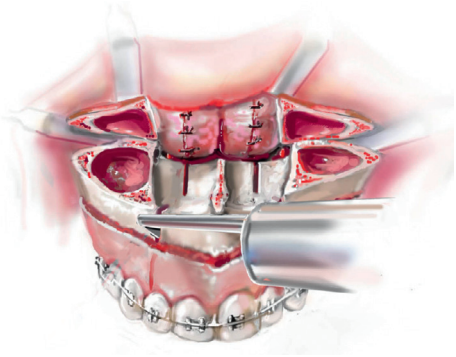
• **Figure 16-17** An individual who was born with a bilateral cleft lip and palate was treated at another institution. During the mixed dentition, he underwent unsuccessful iliac (hip) bone grafting and fistula closure. The premaxilla remained mobile, with residual alveolar clefts. When he was 17 years old, he underwent maxillary Le Fort I osteotomy, also at another institution. Unfortunately, given the residual alveolar clefts, the circumvestibular incision that was used resulted in circulation injury to the premaxilla (i.e., aseptic necrosis). Unless the bilateral cleft is successfully grafted through at least one of the alveolar sites, a circumvestibular incision that cuts through the labial vestibule of the premaxilla will predictably result in aseptic necrosis. **A** and **B,** Occlusal and palatal views are shown during the mixed dentition after unsuccessful grafting. **C** and **D,** The same views are shown during the teenage years after unsuccessful Le Fort I osteotomy with aseptic necrosis of the premaxilla and the resulting oronasal fistula.



Prior to treatment | 5 weeks after surgery | 6 months after surgery

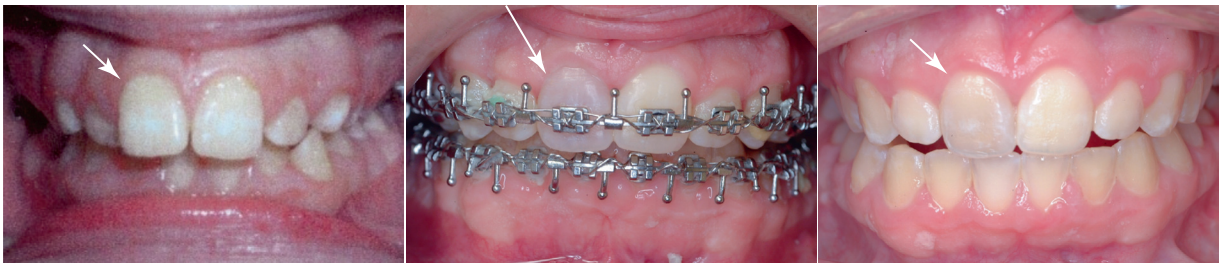


5 weeks after surgery



• **Figure 16-18** A man in his 20s presented with a maxillary deficiency and a relative mandibular excess growth pattern. He underwent orthodontics and reconstruction, including a Le Fort I osteotomy in three segments (i.e., interdental cuts between the lateral incisor and the canine on each side). Approximately 3 months after surgery, the right lateral incisor crown was noted to be dark in color. No other symptoms occurred. A postoperative radiograph shows the interdental osteotomy site. There was no direct injury to the dental root on either side of the osteotomy. The pulp of the tooth was treated with root canal therapy. Bleaching is planned to whiten the crown. Occlusal views are shown before orthodontics, at 5 weeks after surgery, and after successful reconstruction. An illustration and a maxillary model show the location of segmental osteotomies.

Pulpal necrosis



Pre treatment | 5 weeks after Le Fort I | 1 year after Le Fort I

• **Figure 16-19** Occlusal views are shown of a teenage girl with a long face growth pattern. She underwent an orthodontic and surgical approach. The surgery included maxillary Le Fort I osteotomy without segmentation. By 5 weeks after surgery, the right maxillary central incisor was noted to be dark in color. No other symptoms occurred. A postoperative radiograph showed no signs of root injury or periapical pathology. The pulp of the tooth was treated with root canal therapy followed by bleaching.

histologic techniques to study revascularization and wound healing (see Chapter 2).[25] Meyer and colleagues also completed animal studies involving radioactive microsphere methods to quantify preoperative and postoperative blood flow after classic osteotomies.[219] The results of Bell's microangiographic and histologic studies showed minimal transient vascular ischemia, minimal osteonecrosis, and early osseous union, with the maxilla pedicled essentially to the palatal mucosa. *The preservation of the integrity of the descending palatine arteries was not found to be essential to maintain circulation to the down-fractured maxilla.* The periosteal vascular bed provides adequate reserve blood supply after down-fracture, even with ligation of the named vessels. Alteration of the hemodynamics within the intramedullary cavity after total maxillary down-fracture was felt to produce only transient and clinically insignificant intraosseous ischemia (see Chapter 2).

In clinical practice, controversy continues to exist regarding the management of the descending palatine artery (DPA) during Le Fort I osteotomy. Some surgeons advocate the preservation of the DPA, whereas others ligate the vessel routinely. Dodson and colleagues completed a prospective randomized clinical study of patients (N = 34) who underwent Le Fort I osteotomy.[86,87] The individuals were randomly assigned to either Study Group 1 (N = 26), in which the DPA was ligated, or Study Group 2 (N = 18), in

which the DPA was preserved. The researchers measured maxillary gingival blood flow during the operation with the use of a laser Doppler flow meter, and they found "no statistically significant differences in mean maxillary gingival blood flow between patients having the DPA ligated and those having the DPA preserved."[87] The maintenance of adequate flap circulation through the palatal mucosa attached to the hard palate is the key to the avoidance of this problem.

Documented cases of aseptic necrosis after maxillary osteotomies primarily come from the questionnaire survey conducted by **Lanigan and colleagues.**[182] The authors sent out 5000 questionnaires to oral and maxillofacial Surgeons, and 800 replies were received. Fifty-one cases of aseptic necrosis after maxillary surgery were reported from 44 surgeons who had encountered this specific complication. The number of cases reported was assumed to be less than the magnitude of the clinical problem in practice. The results of the questionnaire suggested that, at the time of the survey (i.e., in 1986), a Le Fort I osteotomy performed in multiple segments in conjunction with superior repositioning and transverse expansion (especially when significant palatal perforations occurred) was more likely to result in a tenuous blood supply to the anterior maxilla, with resulting aseptic necrosis. More recently, **Singh and colleagues** described a single patient from the United Kingdom who underwent maxillary Le Fort I osteotomy with down-fracture (without segmentation) and who sustained aseptic necrosis of approximately two thirds of the total maxilla.[305] The patient's risk factors included nicotine abuse, the use of deliberate hypotensive anesthesia in the presence of baseline hypertension, and uncontrolled intraoperative bleeding. In 2010, Pereira and colleagues reported a case of maxillary aseptic necrosis after Le Fort I osteotomy.[255] The patient was 52 years old, with baseline hypertension, an ongoing smoking history of two packs per day, and chronic generalized periodontal bone loss and gingival recession. He underwent Le Fort I down-fracture osteotomy with horizontal advancement. Uncommon bleeding was noticed in the posterior region intraoperatively, and this was treated with gauze compression. On the seventh postoperative day, the researchers reported that the "[m]ucosa overlying the maxilla was ischemic and covered with a pseudomembrane."[255] The patient was treated with 20 sessions of hyperbaric oxygenation. The maxilla and the associated dentition were preserved, but further alveolar bone loss occurred.

Palatal Fistula

The tearing of the palatal mucosa can occur at the time of either surgically assisted rapid palatal expansion (SARPE) or standard parasagittal segmental osteotomies carried out through the Le Fort I down-fracture. A tear is more likely to become clinically apparent when significant transverse expansion is undertaken. Intraoperative palatal mucosa tears at the time of segmental osteotomy will occur and are best managed by meticulous nasal mucosa closure and by interposing a middle layer to limit epithelialization with fistula tract formation during the healing of the palatal mucosa. This can be accomplished with a biocompatible material (e.g., Surgicel, fibrin glue, acellular collagen matrix) that is placed through the down-fractured maxilla directly onto the bony palate (i.e., the floor of nose side) after stabilization of the segments into the prefabricated splint and before the placement of maxillary plate and screw fixation. By instituting precautions to limit pressure gradients across the opening and by avoiding hard food boluses on the roof of the mouth during initial healing, spontaneous closure is typically seen. Under these circumstances, long-term persistent fistulization of a palatal mucosal tear after segmental osteotomies is uncommon. When a palatal (i.e., oronasal) fistula remains and is of clinical concern as a result of a loss of fluid and air, it can be closed with the use of local palatal flaps (Fig. 16-20). Surgical closure should be postponed until 6 to 12 months after Le Fort I osteotomy to ensure adequate maxillary revascularization. During the interim, a custom acrylic palatal plate can be used to both obturate the palatal–nasal opening and to assist with the maintenance of the new arch form.

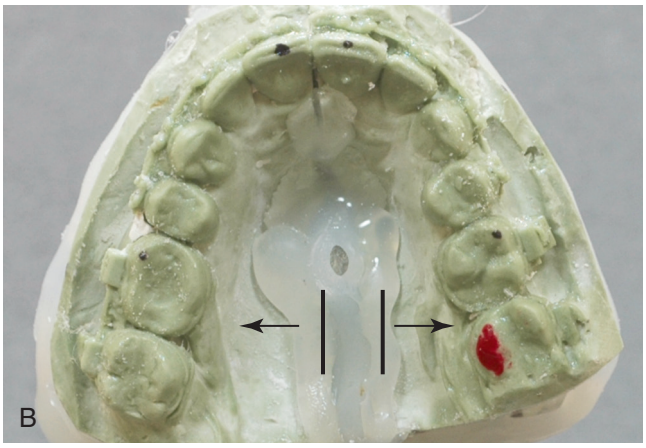
Fibrous Union (Painless Mobile Maxilla)

The fibrous union of the maxilla after Le Fort I osteotomy is an infrequent complication.[78,162,200,342,350] Risk factors should be recognized, and surgical techniques and postoperative management may then be adjusted to limit this complication. The combination of extensive horizontal advancement and vertical lengthening results in large osteotomy site defects with limited bone contact. Failure to form adequate bony bridges at the healing sites is likely to result in delayed healing, fibrous union, or late relapse. Influences on bone healing at the Le Fort I osteotomy site may include the following: 1) injury to flap circulation 2) thin osteoporotic bone at the buttress and the pyriform rims with inadequate stabilization (fixation) 3) the decision being made to not place interpositional grafts when the bone gaps are of critical size (as described previously) 4) infection or wound dehiscence after surgery 5) the presence of a heavy occlusion (e.g., grinding, clenching) 6) too rapid of a return to normal chewing forces 7) excessive mouth opening against intermaxillary elastics and 8) stress fracture of the fixation plates. Systemic risk factors for poor bone healing include 1) diabetes; 2) collagen disease; 3) vascular disease; 4) osteoporosis and the use of bisphosphonates; 5) nicotine use; 6) poor nutritional status; and 7) prior radiation.

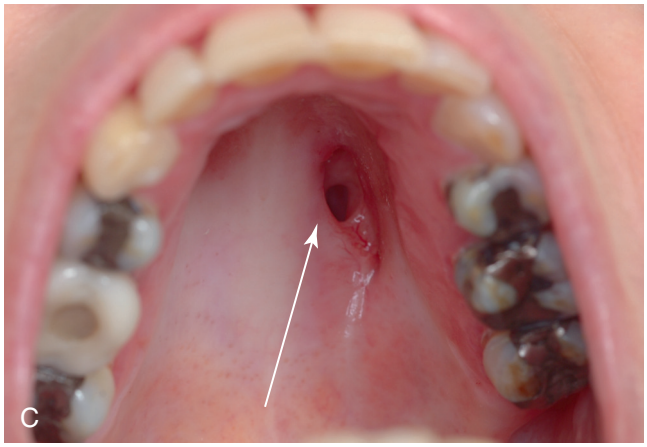
This author has experience with four of his own patients who underwent Le Fort I osteotomy and then developed fibrous union of the maxilla (Fig. 16-21). This represents a small percentage (i.e., <0.5%) of the total group of Le Fort I osteotomies performed. In each case, initial healing seemed unremarkable, with no infection or wound dehiscence. There were no signs of vascular compromise at the time of

Text continued on p. 516

Palatal view prior to surgery



Segmental osteotomies with widening



Palatal fistula early after surgery

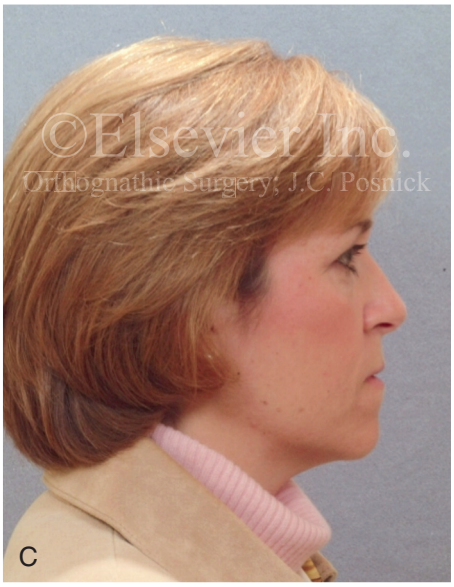
Successful surgical closure of fistula

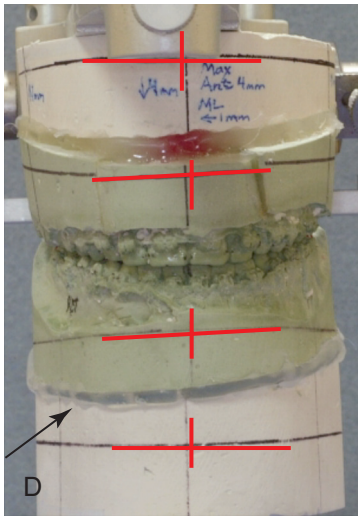
• **Figure 16-20** A 48-year-old woman with a long face growth pattern and Angle Class II anterior open-bite malocclusion is shown. The patient underwent four bicuspid extractions and orthodontic treatment during childhood as a compensatory approach. She was referred by a restorative dentist to an orthodontist and then to this surgeon for evaluation. She was found to have obstructive sleep apnea, chronic obstructive nasal breathing, and a long face growth pattern with a Class II tendency. She underwent a comprehensive surgical, orthodontic, and dental rehabilitative approach. The procedures included maxillary Le Fort I osteotomy in two segments (interdental osteotomy between the central incisors and then parasagitally down the hard palate). The maxillary segmentation was to widen the posterior arch by several millimeters. When placing the maxillary segments into the intermediate splints, a tear in the palatal mucosa at the parasagittal osteotomy location was appreciated. This was managed with Gelfoam and tissue glue through the down-fracture along the floor of the nose and with meticulous nasal mucosa suturing. A fistula persisted at 1 year after surgery, with air loss affecting her speech (i.e., hypernasal speech) and fluid loss occurring into the nose when she was drinking liquids. She underwent outpatient surgery to elevate the palatal flaps, to separate the nasal and palatal mucosa, to suture closed the nasal side, and then to suture closed the palatal side. She was placed on a mechanical soft diet and sinus precautions for 4 weeks. The fistula was successfully closed. **A,** Palatal view before surgery. **B,** Model planning indicating two-segment maxillary osteotomy with widening at the molar region. **C,** Palatal view 1 year after surgery indicating a persistent oronasal fistula. **D,** Palatal view 3 months after the successful surgical closure of the fistula.

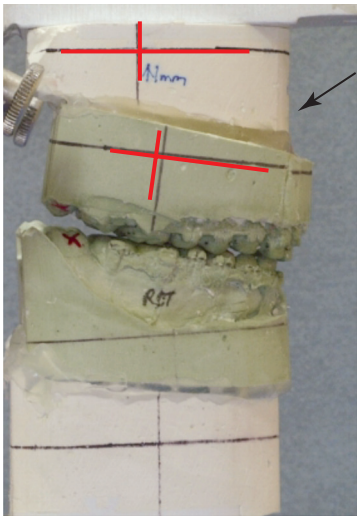
• **Figure 16-21** A woman in her mid 40s with an asymmetric mandibular excess growth pattern arrived for surgical evaluation. The longstanding asymmetric Angle Class III negative overjet anterior open-bite malocclusion had been detrimental to her dentition. She required crown restorations and had a need for root canal therapy in several of the posterior teeth. She desired the correction of the malocclusion (i.e., improved chewing and speech articulation) and improved lip closure (i.e., relief of mentalis strain). She had a lifelong history of obstructed nasal breathing and was found to have septal deviation and inferior turbinate enlargement. She agreed to an orthodontic and surgical approach. Orthodontic (dental) decompensation was followed by surgery that included maxillary Le Fort I osteotomy (horizontal advancement, clockwise rotation, and vertical lengthening); bilateral sagittal split ramus osteotomies (asymmetry correction); osseous genioplasty (horizontal advancement); and septoplasty and inferior turbinate reduction. The quality of the maxillary buttress bone was noted to be thin, and it did not hold the fixation plates and screws firmly in all locations as compared with bone of average thickness; however, it was felt to be adequate. No interpositional graft was used, and initial healing was on track. Five weeks after surgery, the patient progressed to a normal diet and her regular physical activities. She also returned to the care of her orthodontist. Postoperative radiographs (lateral cephalometric and Panorex) did not look out of the ordinary. Three months after surgery, during a routine orthodontic evaluation, she was found to have mobility of the maxilla but without pain and with a good occlusion. The diagnosis of fibrous union at the Le Fort I osteotomy site was made. The patient was placed on a liquid diet, told to avoid clenching and heavy biting forces, and asked to stop all use of interarch elastics. Despite a waiting period of an additional 2 months, a painless mobile maxilla (i.e., fibrous union) remained. The patient was returned to the operating room, where she underwent the following:

- Standard circumvestibular incision and subperiosteal anterior maxillary exposure
- Removal of all maxillary fixation plates and screws
- Down-fracture and complete disimpaction
- Removal and cleaning out of all fibrous tissue along the osteotomy sites
- Placement of the maxilla into the preferred occlusion through a prefabricated splint
- Adjustment of the maxilla to the preferred anterior vertical height
- Placement of new titanium bone plates and screws at each zygomatic buttress and pyriform aperture
- Harvesting and crafting of the iliac (hip) corticocancellous graft
- Placement of a bloc graft in each bony gap between the zygomatic buttress and the pyriform plates
- Securing of each graft with an additional titanium microplate and screws
- Packing of additional cancellous bone along the osteotomy sites
- Wound closure

At 5 weeks after surgery with indications of good healing, the patient's diet and physical activities were advanced, and she was returned to her orthodontist's care for maintenance. The appliances were removed 3 months later. **A,** Facial and occlusal views before treatment.







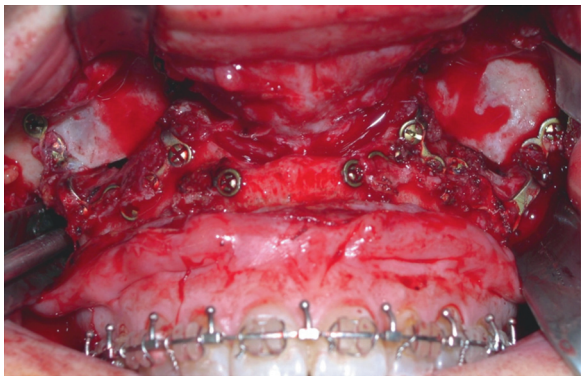
• **Figure 16-21, cont'd** **B,** Facial and occlusal views with orthodontic decompensation in progress. **C,** Facial profile and cephalometric views before surgery. **D,** Articulated dental casts that indicate analytic model planning.

Continued

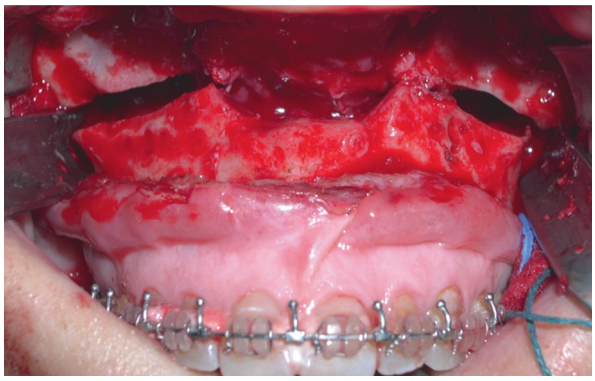
Fibrous union–mobile maxilla!
Good occlusion–no pain!

Fibrous union–mobile maxilla!
Good occlusion–no pain!

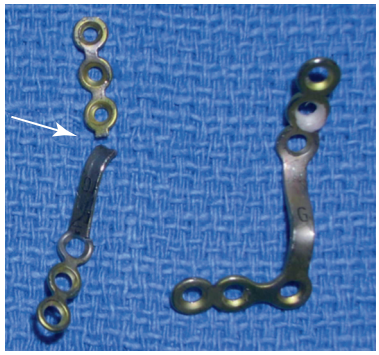
• **Figure 16-21, cont'd E,** Facial and occlusal views 3 months after surgery, when the fibrous union was recognized. **F,** Facial and lateral cephalometric views 3 months after surgery, when the fibrous union of the maxilla was recognized.

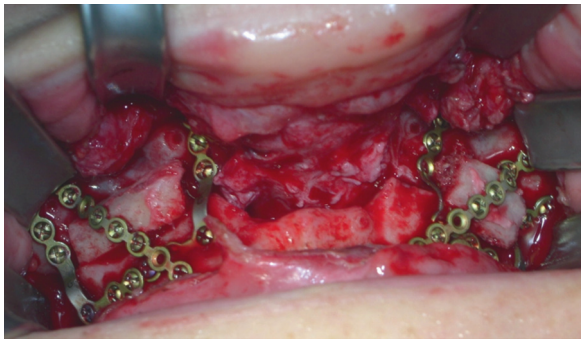


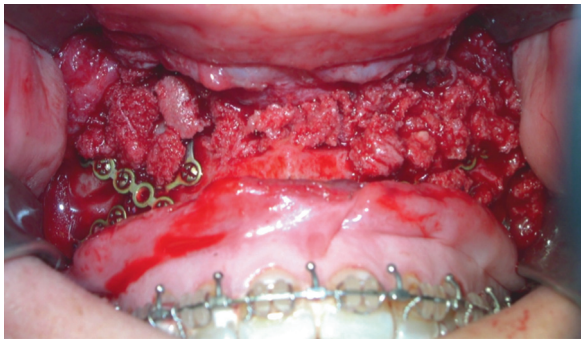
Prior to hardware removal

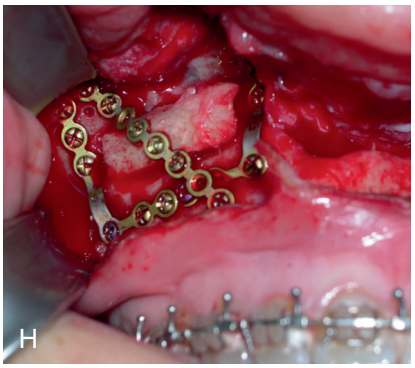


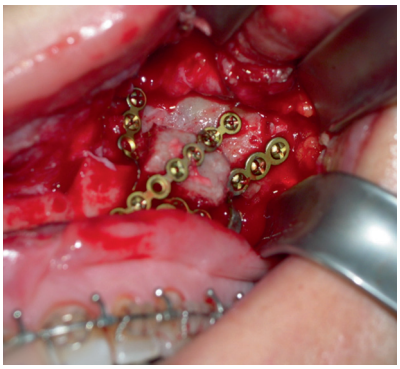
After redo osteotomy and downfracture











• **Figure 16-21, cont'd G,** Intraoperative views showing the fibrous union and the stress-fractured plates before removal and down-fracture. The removed plates are also shown. Three of the four plates were "fractured." The maxilla is then shown after the removal of the plates and screws, the down-fracture, and the cleaning out of fibrous tissue. **H,** Intraoperative views showing the maxilla secured with new plates (one at each zygomatic buttress and the pyriform rim) and screws. Bloc corticocancellous (iliac) grafts are in place, with additional plates and screws. Additional cancellous graft is packed into all defects before wound closure.

Continued

• **Figure 16-21, cont'd I,** Frontal views in repose before and after successful reconstruction. **J,** Frontal views with smile before and after successful reconstruction. **K,** Oblique facial views before and after successful treatment.

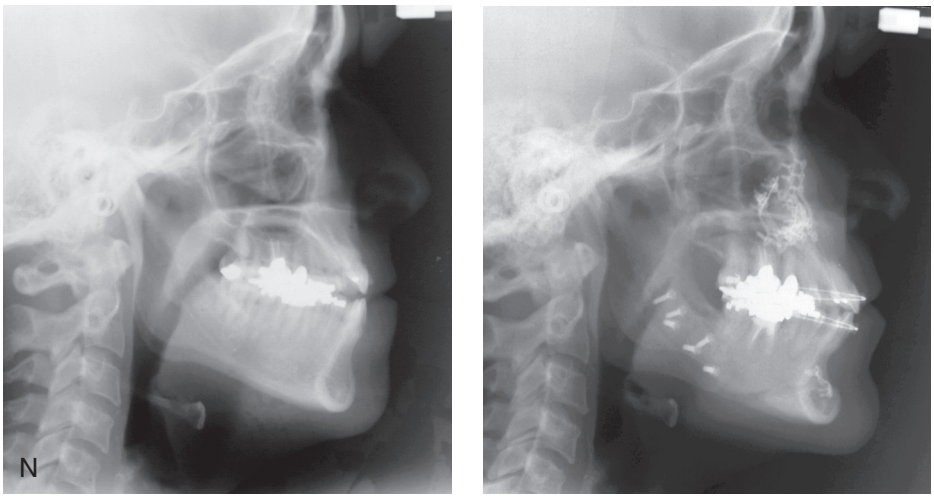
Prior to treatment

Pre surgery

After treatment

• **Figure 16-21, cont'd L,** Profile views before and after successful treatment. **M,** Occlusal views before treatment, before surgery, and after successful treatment.

Continued



• **Figure 16-21, cont'd N,** Preoperative and late postoperative lateral cephalometric radiographs.

osteotomy or after surgery. None of the patients were felt to have more than average residual mobility at the 5-week postoperative interval upon return to their orthodontist's care. They were then encouraged to gradually return to a regular diet and the usual physical activities as well as to work toward normal mouth opening. The four affected individuals were as follows:

1. *A 15-year-old Caucasian individual with Down syndrome who underwent Le Fort I osteotomy, SSRO of the mandible, and oblique osteotomy of the chin.* The maxilla underwent horizontal advancement and vertical lengthening without interpositional grafting. Maxillary stabilization was performed with a titanium plate placed at each pyriform aperture, and the zygomatic buttress region was secured with screws. The quality of the bone was thin and did not hold the fixation screws as firmly in all locations as is typical. Initial healing was felt to be normal. The patient returned to the orthodontist's care at 5 weeks without special concerns.
2. *A 25-year-old Asian graduate student who presented with congenital facial palsy that resulted in maxillofacial skeletal asymmetry.* This patient underwent Le Fort I osteotomy, SSRO of the mandible, and oblique osteotomy of the chin. The maxilla underwent cant correction and intrusion with minimal horizontal advancement. Stabilization was obtained with a titanium plate placed at each zygomatic buttress, and the pyriform aperture was secured with screws. The quality of the bone was thin and did not hold the fixation screws as firmly at all locations as is typical. Initial healing was felt to be normal. The patient returned to her orthodontist's care at 5 weeks without special concern.
3. *A 45-year-old woman with a history of osteoporosis that had previously been treated with oral bisphosphonates.* This patient's jaw deformity was characterized by maxillary deficiency with relative mandibular excess. She underwent Le Fort I osteotomy, SSRO, and oblique osteotomy of the chin. The maxilla was horizontally advanced and vertically lengthened. Stabilization was performed with a titanium bone plate placed at each zygomatic buttress, and the pyriform aperture was secured with titanium screws. The quality of the bone was thin and did not hold the fixation screws as firmly as is usual at all locations, but it was felt to be adequate. No interpositional graft was placed, and initial healing was felt to be normal. The patient returned to her orthodontist's care at 5 weeks without special concerns.
4. *A 45-year-old woman with an asymmetric mandibular excess growth pattern (see Fig. 18-20).* This patient underwent Le Fort I osteotomy, SSRO, and oblique osteotomy of the chin. The maxilla was horizontally advanced and vertically lengthened. Stabilization was with a titanium plate placed at each pyriform aperture, and the zygomatic buttress was secured with screws. The quality of the bone was thin; it did not hold the fixation screws firmly at all locations as is average, but it was felt to be adequate. No interpositional graft was placed, and initial healing was felt to be normal. The patient returned to her orthodontist's care at 5 weeks without special concerns.

Each of the four patients presented back to this surgeon between 4 and 6 months after surgery complaining of painless but visible motion of the maxilla when clenching and chewing. Each individual presented at that time with a good occlusion and favorable facial aesthetics but with a pain-free mobile maxilla with movement that was observed during compressive occlusal forces. In each case, the patient was placed on a liquid diet and told to avoid clenching and heavy biting forces, and the use of elastics was stopped. Despite a waiting period of an additional 2 months, a painless mobile maxilla (i.e., fibrous union) remained in each case. One of the four individuals elected not to proceed with further surgery and continues to have a painless mobile

maxilla during mastication. The other three patients underwent further maxillary surgery. The procedure included the following steps:

1. Return to the operating room with general anesthesia through standard nasotracheal intubation
2. Maxillary circumvestibular incision and standard subperiosteal dissection for Le Fort I exposure
3. Removal of all maxillary fixation plates and screws
4. Down-fracture and complete disimpaction
5. Removal of all fibrous tissue at the osteotomy sites
6. Placement of the maxilla into the preferred occlusion with the mandibular dentition using a prefabricated splint; adjustment of the maxilla to the preferred vertical height
7. Placement of new titanium plates and screws at each zygomatic buttress and pyriform aperture
8. Harvesting of iliac (hip) corticocancellous graft.
9. Crafting of a corticocancellous (iliac) bloc graft and placement in the bony gaps between the zygomatic buttress and the pyriform plates on each side
10. Securing of each corticocancellous graft with an additional titanium plate and screws
11. Packing of additional cancellous bone along the osteotomy sites
12. Closure of the circumvestibular wound

The three patients who underwent further treatment were maintained on a blenderized diet and placed on limited physical activities for 5 weeks. No intermaxillary fixation and only a limited use of elastics and compression occlusal forces were instituted during this time.

Interestingly, in the three patients who received additional treatment, the fracture of most of the titanium plates was recognized at the time of the redo Le Fort I osteotomy (see Fig. 16-21D). One explanation is that the titanium plate failure occurred soon after surgery. If so, then the fractured plates would be responsible for inadequate immobilization at the osteotomy site, thereby resulting in fibrous union. Another explanation is that bony union did not occur at the usual time (i.e., approximately 4 to 5 weeks after surgery). The initially successful plate and screw fixation masked the fibrous union for several months; this was followed by the eventual stress fracture of the titanium plates in response to the continuous masticatory compressive occlusal forces. Painless mobility of the maxilla eventually became noticeable to the patient (i.e., 4 to 6 months after surgery), and this was then brought to the surgeon's attention.

Despite this author's extensive experience with cleft orthognathic surgery that often requires maxillary segmentation with significant horizontal advancement and vertical lengthening, no cases of fibrous union have occurred in this patient group. The author believes that the routine use of interpositional iliac (hip) grafts placed at the Le Fort I osteotomy site in patients with clefts who are undergoing significant advancement and vertical lengthening is helpful. (This is in addition to the use of titanium plates and screws and a prefabricated splint; the maintenance of a non-chew, liquid diet for 5 weeks; and the absence of postoperative infection or wound dehiscence.)

Acute and Chronic Nasal Obstruction

Buckling and deviation of the nasal septum can occur during the superior repositioning of the maxilla after Le Fort I osteotomy (see Fig. 16-22). When vertically intruding the maxilla, adequate resection of the inferior aspect of the cartilaginous and bony septum is required to avoid this complication.[208] If the septum is properly managed at operation, the occurrence of a "crooked nose" after the removal of the nasotracheal tube should be uncommon. Visual inspection of the nose is necessary after maxillary repositioning just before and again after extubation to confirm that the septum remains straight. If septal displacement is observed after extubation and is simply due to nasotracheal tube pressure, then limited manual manipulation in the recovery room will suffice. If the surgeon did not adequately resect (i.e., manage) the inferior aspect of the septum when completing a Le Fort I intrusion, then a return to the operating room to do so will be required.

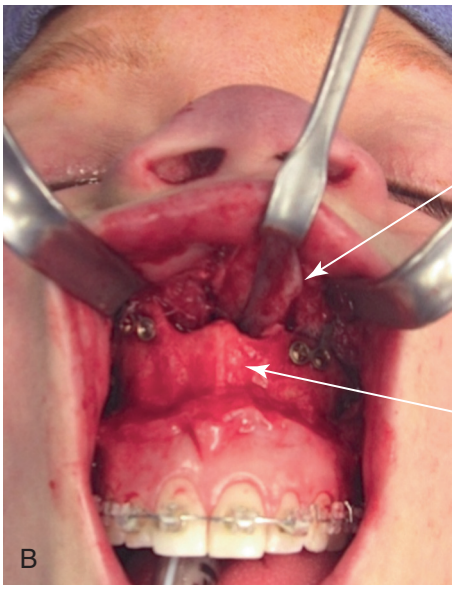
The most common reason for persistent postoperative nasal obstruction is preoperative chronic nasal obstruction that was not recognized and addressed (see Chapter 10). The second likely cause of persistent nasal obstruction is inadequate recontouring of the nasal floor, the pyriform rims, and the anterior nasal spine in conjunction with maxillary intrusion.

NOTE: A worsening of any preoperative chronic nasal obstruction in the patient with a long face growth pattern is more likely to occur if the baseline excess anterior nasal floor height is not simultaneously addressed (Fig. 16-22).

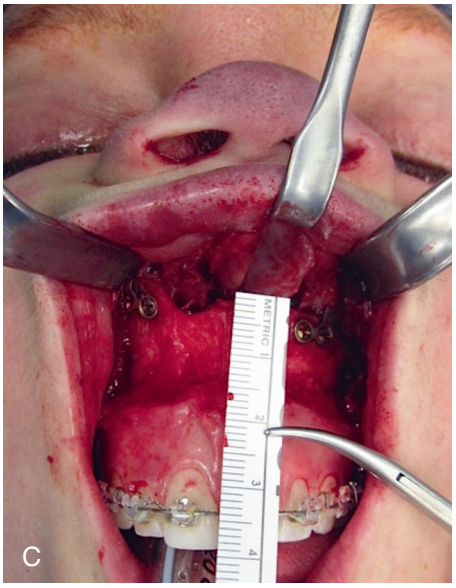
Acute and Chronic Maxillary Sinusitis

The occurrence of persistent maxillary sinusitis that requires treatment after Le Fort I osteotomy is not thought to be common, but the true incidence has not been fully studied.[256,374] When maxillary sinusitis after Le Fort I osteotomy does occur, it may be due to the following: 1) changes in the clearance mechanism of sinus mucous; 2) the retention of a blood clot in the sinus cavity; 3) dental infection; 4) the presence of foreign bodies (e.g., grafts, loose fixation devices) in the sinus cavity; or 5) the anatomic blockage of the osteomeatal opening (Fig. 16-23). A blocked ostium can generally be assessed with fiber-optic nasoendoscopy in the office setting. The occurrence of a loose displaced graft that cannot be expressed through the ostium and that then becomes chronically contaminated with bacteria should also be ruled out. A sinus computed tomography scan is helpful to clarify these issues. Standard titanium fixation screws are expected to perforate through the anterior wall into the

Text continued on p. 525



5 weeks after Le Fort I impaction



Incisal edge to ANS
Patient is 40 mm

Incisal edge to ANS
Should be 30 mm

• **Figure 16-22** A woman in her mid 30s was diagnosed with a long face growth pattern and mild obstructive sleep apnea. She underwent perioperative orthodontics and orthognathic procedures, including maxillary Le Fort I osteotomy (horizontal advancement and vertical intrusion); bilateral sagittal split osteotomies of the mandible (advancement); and osseous genioplasty (horizontal advancement). The surgery was carried out by another surgeon. Immediately after surgery, in the recovery room, significant asymmetry and distortion of the base of the nose and nasal tip were recognized. Although the patient generally convalesced well, there was persistent obstructed nasal breathing and noticeable nasal base and tip asymmetry and distortion. At 5 weeks after surgery, consultation with this surgeon occurred. Clinical and radiographic examination confirmed uncorrected excess anterior alveolar maxillary height as well as a buckled and deviated quadrangular cartilage. Visually, there was marked distortion of the base of the nose and nasal tip as well as blocked nasal airflow. The patient was taken to the operating room and underwent general anesthesia through orotracheal intubation. A limited intraoral circumvestibular incision was made with the dissection of the anterior maxilla, the floor of the nose, and the anterior aspect of the septum. The maxillary incisal edge to the anterior nasal spine measured 40 mm (elongated by three standard deviations) and confirmed vertical excess. The quadrangular cartilage was buckled and deviated, which further contributed to an obstructed left nasal passage. Using a rotary drill with a watermelon bur and a rongeur, the nasal rims, the floor of the nose, and the anterior nasal spine were recontoured and lowered toward normal morphology. The incisal edge to the anterior nasal spine now measured 30 mm. The inferior aspect of the quadrangular cartilage was resected and repositioned to the midline. **A,** Frontal facial view at 5 weeks after initial surgery indicating distortion and asymmetry of the nasal base and tip. **B,** Intraoperative view of anterior vertically maxillary excess and buckled quadrangular cartilage at redo surgery. **C,** Measurement of the incisal edge to the anterior nasal spine (40 mm) before recontouring confirms the baseline elevation of the nasal floor.

Planned recontouring

Rotary drill to recontour

Nasal floor recontoured

Reduction of ANS

Resection of inferior septum

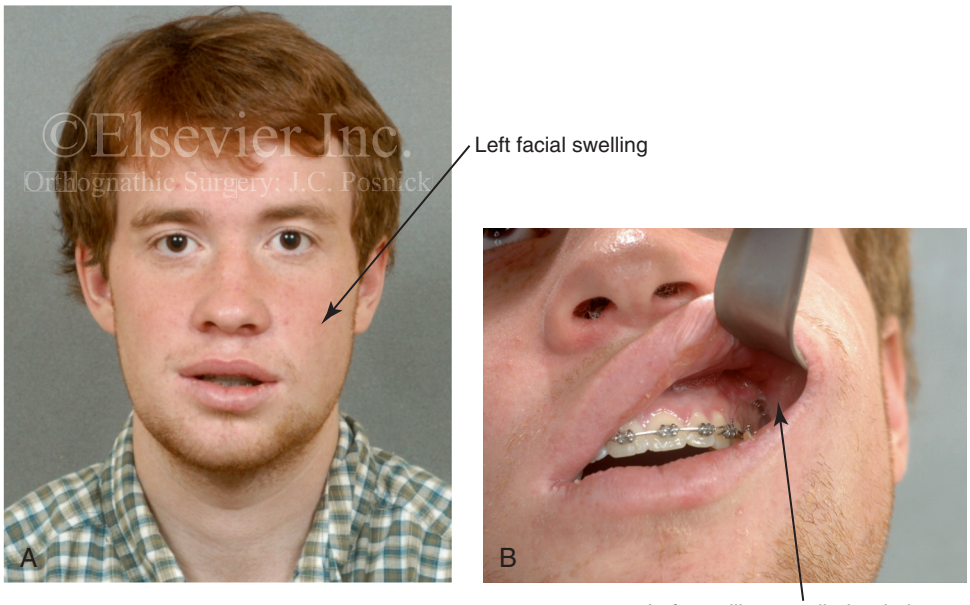
• **Figure 16-22, cont'd** **D,** Pencil marks indicate the planned lowering of the floor of the nose. **E,** The floor of the nose is recontoured (lowered) with a rotary drill. **F,** This is followed by rongeur resection and refinement of the nasal spine. **G,** A rongeur was then used to resect the inferior aspect of the quadrangular cartilage.

Continued

5 weeks after Le Fort I impaction with crooked nose

8 weeks after Le Fort I- 3 weeks after recountouring/septoplasty

• **Figure 16-22, cont'd H,** Facial views of the patient 5 weeks after the initial orthognathic procedure and then 3 weeks after secondary revision (i.e., recontouring of the floor of the nose and septoplasty). The patient's nose is no longer crooked, and the nasal airway is now patent.

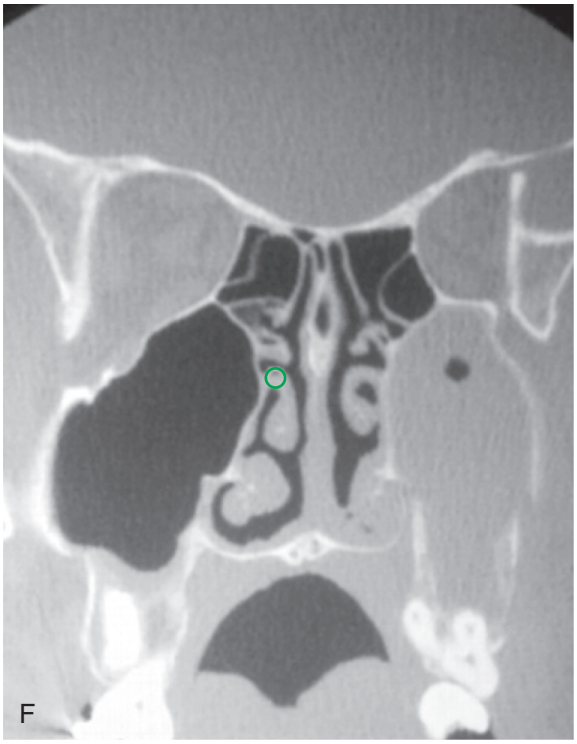


• **Figure 16-23** A 19-year-old man with a maxillary deficiency in combination with asymmetric mandibular excess presented for treatment. He also had chronic obstructed nasal breathing. He was referred for orthodontic and surgical correction. His surgery included maxillary Le Fort I osteotomy (horizontal advancement and clockwise rotation); bilateral sagittal split osteotomies of the mandible (correction of asymmetry); osteotomy of the chin (horizontal advancement); septoplasty; and inferior turbinate reduction. In the presence of a penicillin allergy, prophylactic clindamycin antibiotic was initiated just before surgery and continued for 5 days. Approximately 2 weeks after surgery, left cheek region swelling and intraoral purulent drainage were noted. A computed tomography scan confirmed a fluid-filled left maxillary sinus. The patient was restarted on clindamycin (600 mg by mouth four times daily), and a Penrose drain was inserted into the left maxillary vestibular wound. The cultures confirmed beta-hemolytic streptococci (Group C). Sensitivities indicated that ciprofloxacin was the antibiotic of choice, so the patient was started on a 10-day course. He was also placed on nasal decongestants. Within days, the drainage subsided, and the swelling diminished. The Penrose drain was removed 1 week after insertion. At 5 weeks after surgery, the healing of the osteotomies remained on track, and the patient resumed a regular diet. At 6 weeks, left cheek swelling recurred. A computed tomography scan confirmed a fluid-filled maxillary sinus without adequate drainage into the nose. Nasoendoscopy confirmed a blocked ostium. The endoscope was used to create an ostium to drain the sinus. No further sequelae occurred. **A,** Frontal facial view 2 weeks after surgery showing left cheek swelling. **B,** Left maxillary vestibular wound indicating the site of drainage.

Post operative CT scan
demonstrates osteotomy locations

©Elsevier Inc.
Orthognathic Surgery: J.C. Posnick

6 weeks after orthognathic surgery



Fluid filled left maxillary siunus

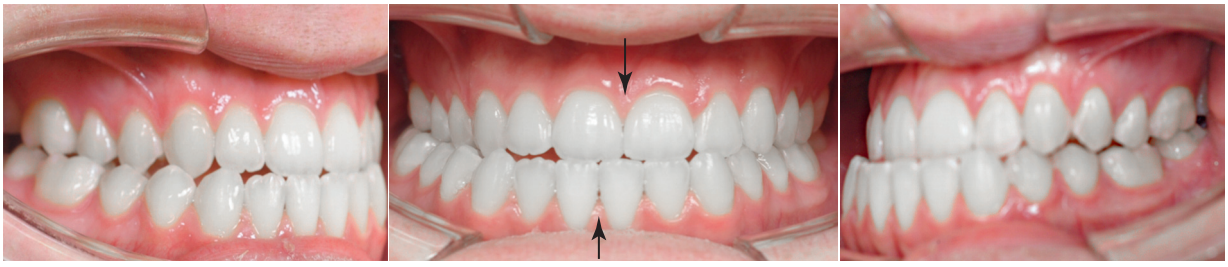
• **Figure 16-23, cont'd C,** A computed tomography scan taken 2 weeks postoperatively confirmed a fluid-filled left maxillary sinus. **D,** A computed tomography scan taken 2 weeks after surgery indicated the osteotomy locations and the repositioning of the jaws. **E** and **F,** Frontal facial and sinus computed tomography scan views at 6 weeks indicating recurrent left maxillary sinus blockage and mucocele.

Continued

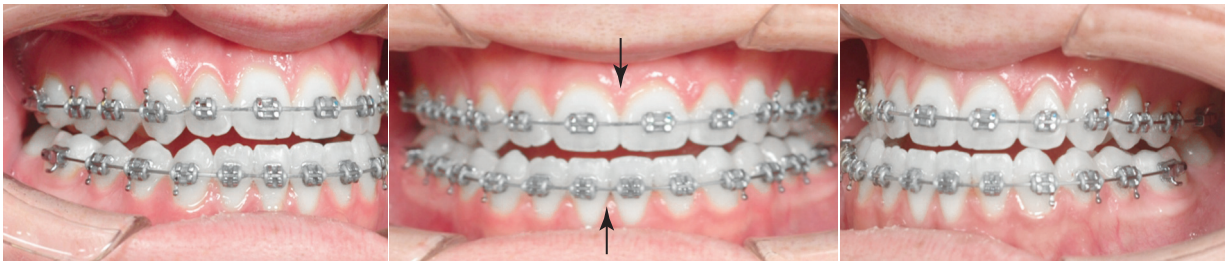
• **Figure 16-23, cont'd G,** Frontal views in repose before and 6 months after treatment. **H,** Frontal views with smile before and 6 months after reconstruction.

• **Figure 16-23, cont'd I,** Oblique facial views before and 6 months after treatment. **J,** Profile views before and 6 months after treatment.

Continued



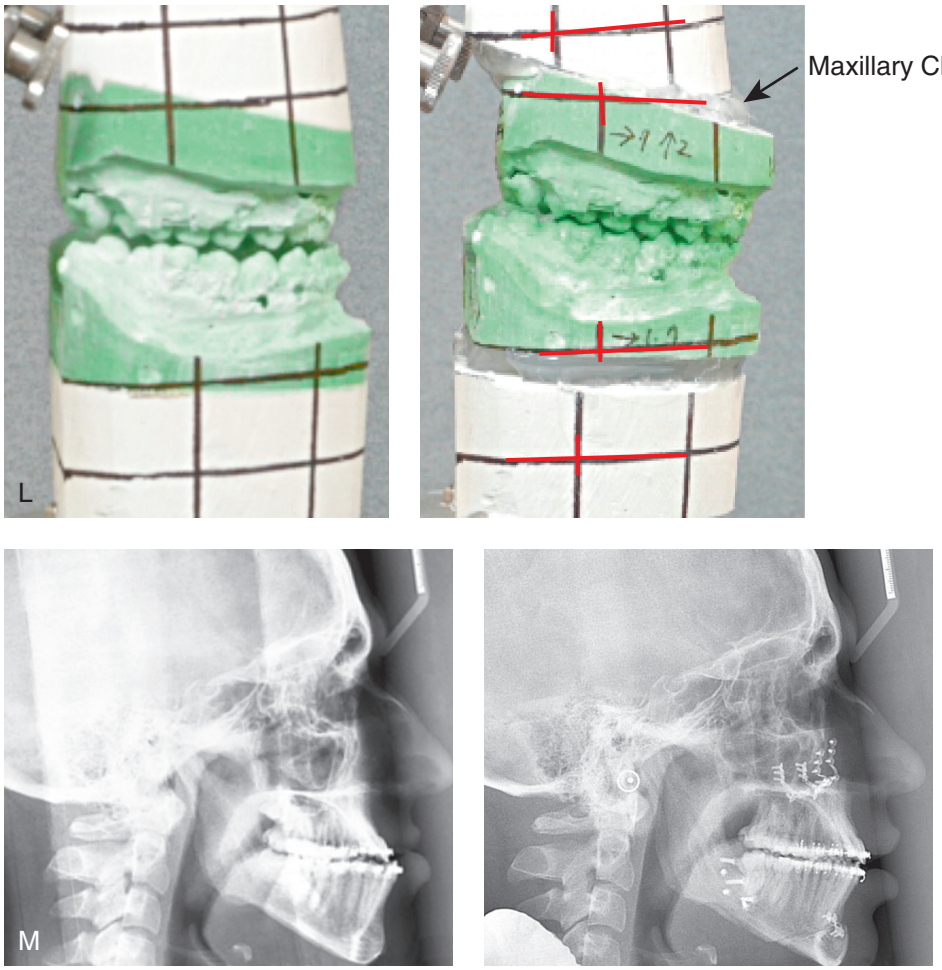
Prior to retreatment



Pre surgery

After surgery

• **Figure 16-23, cont'd K,** Occlusal views before redo orthodontics, with orthodontics in progress, and after treatment.



• **Figure 16-23, cont'd L,** Articulated dental casts indicate analytic model planning. **M,** Lateral cephalometric radiographs before and after reconstruction.

sinus. They are often presumed to be the source of sinusitis; however, this author's experience, that is rarely the case. Interestingly, the most common reason for postoperative chronic sinusitis is preoperative chronic sinusitis that was not recognized and addressed.

The entity of a surgical *ciliated cyst* has been reported after elective orthognathic procedures. These cysts are typically lined with respiratory epithelium. The pathogenesis is hypothesized to involve antral mucosa that is exposed at the time of Le Fort I osteotomy. If the cyst is located in the soft tissues, the displaced mucosa will have been "trapped" in the surgical wound. When the cyst is located in the antrum, then the etiology is thought to be disrupted mucosa trapped in the sinus behind a closed ostium.

Risks and Complications Specific to the Oblique Osteotomy of the Chin (see Chapter 38)

Infection

Infection occurs in less than 1% of patients who are undergoing osseous genioplasty. The routine use of prophylactic antibiotics to cover both oral flora and skin organisms is useful for the limiting of this complication. If an implant is placed to augment the chin (rather than a chin osteotomy being performed) a higher rate of infection is expected.

Mental Nerve Injury

The mental nerve is at risk for injury in association with an osseous genioplasty.[114,238,349] When the soft-tissue dissection and retraction carried out for the exposure of the chin are carefully completed, the mental nerves will be stretched but rarely lacerated or avulsed. If this is the case, the observed immediate loss of sensibility in the lip and chin region should be temporary. The mental nerve can be cut during the soft-tissue incision. Care is taken with the lateral aspect of the incision to avoid this problem. The nerve can be stretched excessively or avulsed from the mental foramen during retraction at the time of osteotomy (see Chapter 15). It is important to remember that the nerve dips below the mental foramen posteriorly. The nerve also extends anteriorly (i.e., the incisal nerve) and may be injured in this location. After genioplasty, approximately 10% of patients will experience a degree of permanent loss of sensibility in the distribution of the mental nerve on one or both sides.

Wound Complications

The osseous genioplasty is completed through an intraoral vestibular incision.[63] To limit wound-healing difficulties, the incision should be made in the depth of the vestibule. The maintenance of a thick cuff of mucosa and muscle tissue adjacent to the cervical margins of the teeth for a relaxed wound closure in two layers (i.e., muscle and mucosa) limits wound dehiscence, muscle dysfunction, chin ptosis, and long-term mucogingival problems. Placing the soft-tissue incision too high in the vestibule can negatively affect wound closure, thereby resulting in dehiscence or scar bands with periodontal sequelae (i.e., mucogingival pull) (Videos 11 and 12).

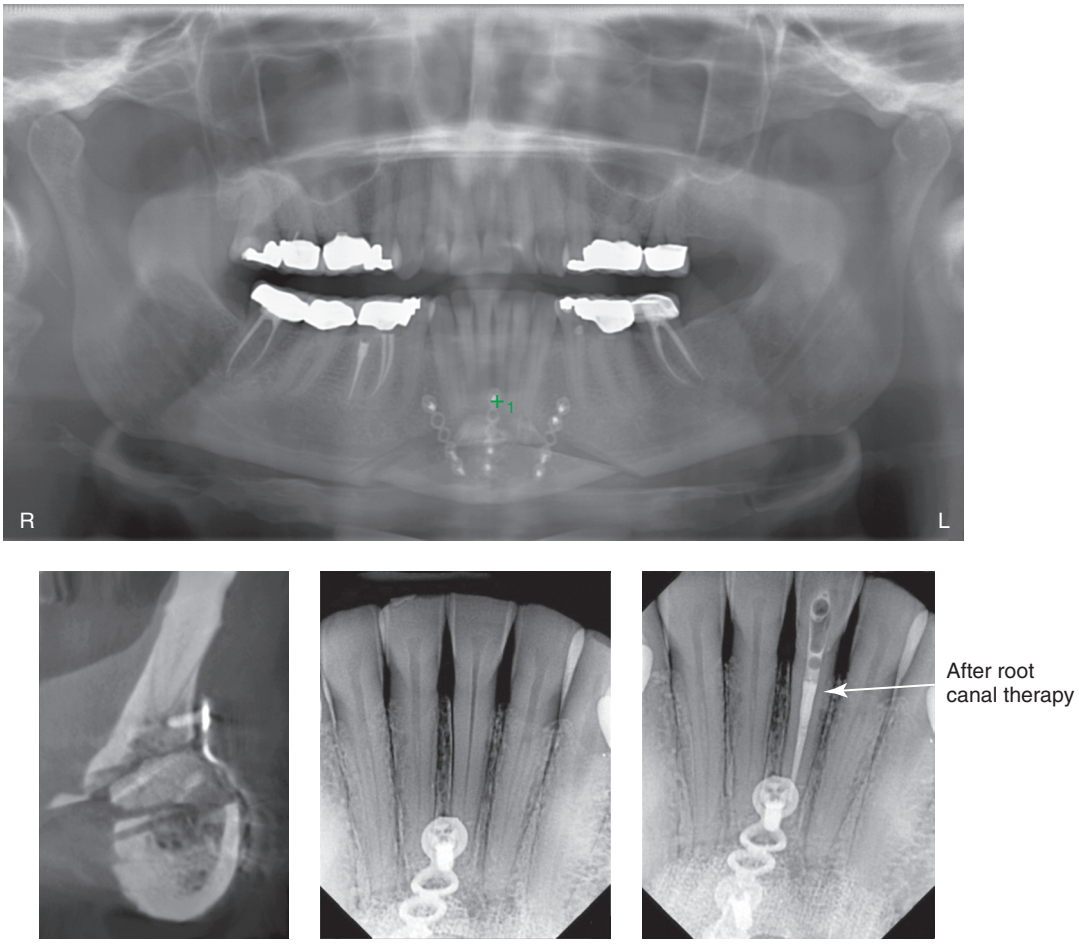
Injury to the Teeth

The location of the chin osteotomy should be based on the patient's chin dysmorphology and a review of the radiographs of the anterior teeth. Injury to the teeth at the time of osteotomy should be an uncommon occurrence. Dental injury may also occur from the fixation screws. If a dental injury is sustained, evaluation by the patient's dentist, referral to an endodontist, and a discussion between the surgeon and the patient are essential, because root canal therapy and other forms of treatment may be indicated (Fig. 16-24).

Suboptimal Facial Aesthetics

Patient dissatisfaction after an osseous genioplasty may relate to facial aesthetics. This is best avoided by doing the following:

1. Having a detailed discussion with the patient concerning his or her objectives and reviewing the patient's overall pretreatment facial and chin morphology and aesthetics (i.e., the recognition of any associated dentofacial deformity) as well as any presenting head and neck dysfunction (e.g., lip incompetence, mentalis strain, obstructive sleep apnea)



• **Figure 16-24** An osseous genioplasty was carried out for the vertical lengthening (interpositional graft) and horizontal advancement of the chin. Postoperative radiographs indicate the closeness of screw fixation (1.7 mm wide and 4 mm in length) to the lateral incisor but without root resorption. The darkening of the crown indicated pulpal injury. Root canal therapy and internal bleaching were carried out.

2. Making sure that there is a clear understanding between the surgeon and the patient with regard to the operative objectives and any compromises in treatment
3. Having the surgeon meticulously execute the procedures with the patient under controlled anesthesia and with competent assistance and proper instrumentation
4. Having the surgeon perform an objective assessment and then discussing with the patient the results achieved and any shortcomings perceived

Conclusions

No matter how accurate the diagnosis, how comprehensive the approach, and how meticulous the surgical technique, complications will occur in a small percentage of patients after orthognathic surgery. Through the discussion of the spectrum of procedure-specific complications and their biologic basis, informed decisions can be jointly agreed to every step of the way by the surgeon, the patient, and the patient's family.

Patient Education Materials

Coordination of Orthodontics and Orthognathic Surgery

To achieve optimal occlusion and facial aesthetics, individuals with jaw discrepancies benefit from a comprehensive coordinated orthodontic and surgical approach.

The goals of orthodontics in preparation for jaw surgery will be distinctly different from those of orthodontic "camouflage" treatment of a skeletal malocclusion. Orthodontic camouflage occurs when your orthodontist attempts to pull the upper and lower teeth together even though the jaws are far apart. Over the long term, this may cause periodontal problems, including the loss of the bone and gingiva surrounding the teeth. In any case, orthodontic camouflage does not correct the jaw deformity or its associated dysfunctions and facial aesthetic concerns. To fully evaluate your needs, your surgeon will likely suggest consultation visits with a speech pathologist and an ear, nose, and throat surgeon. He or she may also recommend evaluation by other medical or dental specialists.

For these reasons, it is important for you to make an educated decision (i.e., orthodontics and jaw-straightening surgery versus orthodontic camouflage treatment alone) before you begin orthodontic treatment. Attempting to correct a jaw problem orthodontically with surgery held out as a contingency plan may result in extended orthodontic treatment time, and it is likely to compromise long-term dental health and facial appearance.

The coordination of orthodontic treatment and jaw surgery from the beginning provides the best opportunity to help you to achieve long-term dental (periodontal) health, efficiency of treatment time, and preferred facial aesthetics. A benchmark time frame for the achievement of the presurgical goals can be set in advance. After you make the decision to proceed, you may wish to contact your medical insurance carrier to determine its commitment for the expected procedures. Our office will work with you toward that end.

For most individuals, good interdigitation of the upper and lower teeth (i.e., occlusion) at the time of surgery requires that specific orthodontic objectives be met. The presurgery orthodontic goals may include the following:

- The correction of dental rotations, anteroposterior misalignments, spacing, and crowding
- The proper torquing of the anterior teeth to achieve ideal dental inclination
- The closure of extraction spaces (unless this is to be achieved by "segmenting" the jaw during surgery)
- The coordination of the dental arch width (unless this is to be achieved by "segmenting" the jaw during surgery)
- The leveling of the occlusal plane or curve of Spee (unless this is to be achieved by "segmenting" the jaw during surgery)

Orthodontic appliances (i.e., braces) are necessary for both presurgical and postsurgical tooth movement as well as to stabilize the jaws during and after surgery. The orthodontic wire sequence is accomplished by the orthodontist at interval visits before surgery. This will preferably end with full-dimensional steel stabilization wires. These passive surgical wires in each arch (i.e., the upper and lower teeth) will prevent any further movement of the teeth just before and during surgery.

The stabilizing passive surgical wires will be placed by your orthodontist approximately 2 to 6 weeks before the date of surgery and only after sufficient presurgical orthodontic objectives are met. Closer to the date of surgery, your orthodontist will also place interdental hooks on the stabilizing wires to meet surgical and early postsurgical needs.

Continued

After the passive surgical wires are in place, your surgeon will obtain the immediate presurgical records, which include alginate impressions of the teeth, centric bite registration, face-bow registration, and facial measurements. The dental casts are generally mounted on a semi-adjustable articulator for the completion of surgical planning and splint construction. Information from the surgical planning will be used during the operation to achieve the most precise occlusion and preferred facial aesthetics.

Orthognathic surgery is performed in an operating room with the use of general anesthesia. Before surgery, you should undergo a physical examination by your general physician and obtain necessary laboratory work to ensure your safety. You will check into the hospital on the morning of your surgery, and the operation will generally take from 2 to 4 hours, depending on surgical details. The length of hospital stay is generally 1 to 2 nights.

During the initial recovery period, you can expect swelling (especially of the lips and cheeks), and this will be followed by bruising. The swelling is progressive during the initial 24 to 48 hours after surgery. Nasal congestion, a sore throat, and varying degrees of nausea should also be anticipated. Medications are prescribed to limit these issues. During surgery, the sectioned facial bones are secured back together in their new location with the use of small titanium plates and screws. The use of these plates and screws is a technical advance that limits the need for wiring the jaws together. Instead, orthodontic elastic bands will be used intermittently after surgery and for several weeks after to allow the jaws to comfortably rest together, as needed. The orthodontic elastic bands can be removed at any time, if need be.

Prescriptions for liquid pain medication and antibiotics will be given for use after you leave the hospital. We will advise you about maintaining a high-protein blenderized diet, the importance of adequate hydration, and necessary limitations of your physical activities.

In general, after you leave the hospital, you will be asked to basically remain at home for the initial 2 weeks. Ambulation (walking) is encouraged, and you may read and complete desk-type work as you wish. Returning to "basic" office work or school after 2 weeks is the general rule, but you must refrain from physical activities (e.g., exercise, sports) for a total of 5 weeks.

Although the initial healing phase will generally take approximately 5 weeks to occur, the completion of the healing process will require 6 to 12 months. In general, your orthodontist will begin the postsurgical phase of orthodontic treatment 5 weeks after surgery to maintain the result and to fine-tune your bite. Braces are generally removed within 6 months after surgery, but this will vary in accordance with your individual needs.

As with any operation, certain side effects and complications are possible. Complications specific to orthognathic surgery may be those related to anesthesia, infection, bleeding, alterations in facial sensibility, injury to adjacent teeth or bones, relapse of the new jaw position with malocclusion, limited range of lower jaw movement, temporomandibular (jaw) joint dysfunction, and suboptimal facial appearance. Fortunately, most of these complications are rare; however, the relative risks for your specific surgery should be discussed and understood before proceeding. A separate and detailed consent form will also be provided.

Orthognathic (jaw-straightening) surgery in combination with orthodontics will generally move your teeth and jaws into a new position that is more balanced, functional, and healthy. For a successful outcome, communication is essential. Your orthodontist and surgeon will work together and respond to your concerns. We look forward to providing the highest-quality medical, dental, and facial care to you.

Jeffrey C. Posnick, DMD, MD
Director, Posnick Center for Facial Plastic Surgery

Consent for Orthognathic Surgery

_____ **INSTRUCTIONS**

Initial here This is a document that has been prepared to help your surgeon to educate and inform you about your orthognathic (jaw) surgery and its risks as well as alternative treatment options. It is important that you read this document carefully and completely. Please initial each section on each page to indicate that you have read and understand it. In addition, please sign the consent for surgery on the last page as proposed by your surgeon.

_____ **INTRODUCTION**

Initial here Orthognathic surgery is carried out in conjunction with orthodontic treatment. In general, this is for the purpose of aligning the teeth, improving the occlusion (bite), and enhancing the facial appearance. For most patients, the improvement of speech articulation, breathing, swallowing, and ease of lip closure are additional benefits. Whether or not these would be expected benefits for you should be discussed with your surgeon.

From start to finish, the process generally requires 1 to 2 years, with the majority of orthodontic treatment being carried out before the surgical procedure. The braces remain on the teeth at the time of operation. After surgery, orthodontic treatment continues for the purposes of maintaining the result and detailing the bite.

As your presurgical orthodontic treatment nears completion, additional dental and facial records will be taken. Your surgeon will use articulated dental models to simulate surgery and to more precisely achieve the desired occlusion and facial aesthetics for you in the operating room. From these models, an acrylic bite wafer (splint) will be made to serve as a guide during surgery for the tooth and jaw alignment that we hope to achieve.

Orthognathic surgery is performed in a hospital operating room with the use of general anesthesia. The operation will generally take from 2 to 4 hours, depending on your individual surgical details. The length of the hospital stay is generally 1 or 2 nights.

Orthognathic surgery is an art and a science, but it is not magic. The procedures are individualized for each patient, and the results depend on the characteristics and quality of your tissues (i.e., bones, muscles, skin, teeth, temporomandibular joints, mucosa) and on complicated biologic factors (e.g., tissue deficiencies, effects of previous treatments). The best candidates for orthognathic surgery are those individuals who require and who are interested in making improvements to their occlusion their long-term dental health, their upper airway (breathing), and their facial aesthetics. These patients are looking for enhancements in one or more of these areas but do not expect perfection; they have realistic expectations, they understand the potential risks, and they are psychologically stable.

_____ **ALTERNATIVE TREATMENTS**

Initial here Alternative forms of management include not undergoing orthognathic surgery at all or possibly proceeding with other, more limited surgical options. These alternatives should be understood and discussed to your satisfaction before proceeding. In some circumstances, individuals may choose an orthodontic "camouflage" treatment option or restorative dental procedures to alter the occlusion, even though the skeletal (jaw) deformity will remain. Risks and potential complications are also associated with these alternative forms of treatment. These are all issues to consider in detail before you select the option that best suits your overall needs.

_____ **RISKS OF SURGERY**

Initial here Every surgical procedure involves a certain amount of risk. An individual's choice to undergo a procedure is based on the comparison of the risk to the potential benefit. Although the majority of patients do not experience the complications detailed below, you should discuss each of these items with your surgeon. Be sure that you understand the risks, potential complications, and consequences of the orthognathic and facial procedures that are planned for you.

Continued

_____ **SURGICAL ANESTHESIA**

Initial here Both local and general anesthesia involve risk. There is a possibility of complications, injury, and even death from all forms of surgery as well as from surgical anesthesia or sedation. At your request, a presurgical consultation visit with the anesthesiologist can be arranged to review these issues in detail.

_____ **AIRWAY COMPROMISE**

Initial here Compromise of the airway in association with orthognathic surgery may occur: at the time of the induction of anesthesia; during the surgical procedure; early after surgery before hospital discharge; or later during convalescence at home. With a skilled and experienced team (i.e., the surgeon, the anesthesiologist, and the hospital staff), close patient supervision (i.e., by family and friends), and cooperation after discharge, the risk of airway compromise can be minimized every step of the way.

_____ **INFECTION**

Initial here Infection is infrequent after orthognathic surgery. Prophylactic antibiotics that are started just before surgery and continued during hospitalization and after discharge will be given. If an infection does occur, additional treatment (e.g., a change in antibiotics, drainage and other procedures) may be necessary.

_____ **SCARRING**

Initial here Although good healing after the surgical procedure is expected, less than ideal healing including unattractive scars may occur within the skin, the deeper structures, and the mucous membranes. Additional treatment, including surgical procedures, may be helpful to manage any scarring.

_____ **BLEEDING**

Initial here The use of hypotensive anesthesia techniques has greatly diminished the need for blood transfusions during orthognathic surgery. The decision about when to transfuse will be dependent on a combination of your physiologic parameters. The need for blood replacement should not be considered a complication but rather a consequence of the orthognathic procedures in a small percentage of patients (approximately 5%). In rare instances, excessive hemorrhage may occur that requires either ligation of the involved blood vessel or selective embolization. Ask your surgeon for more information about the limited need for blood transfusions during orthognathic surgery.

_____ **ALTERATION OF FACIAL SENSIBILITY AS A RESULT OF UPPER JAW SURGERY**

Initial here With upper jaw surgery (i.e., Le Fort I osteotomy), the infraorbital nerve is at risk for injury. As a result, the skin of the cheek, the side of the nose, the upper lip, the gingiva (gums), and the mucosa of the roof of the mouth (palate) will have diminished feeling. Gradually over the course of 6 to 12 months, the feeling improves and generally returns towards normal. This should not be considered a complication but rather a consequence of the procedure. Ask your surgeon for more information about facial sensory loss associated with upper jaw surgery.

_____ **ALTERATION OF FACIAL SENSIBILITY AS A RESULT OF LOWER JAW SURGERY**

Initial here With lower jaw surgery (i.e., sagittal ramus osteotomies), the inferior alveolar nerve is at risk for injury; with reshaping of the chin (i.e., osseous genioplasty), the mental nerve is at risk. This will result in diminished sensation of the lower anterior teeth and gums, the lower lip, and the skin of the chin region. Gradually over the course of 6 to 12 months, the feeling improves, but it does not fully return to normal. The degree of permanent diminished sensation and how it is interpreted is unique to the individual and not entirely predictable. This should not be considered a complication but rather a consequence of the procedure. In rare cases, lower jaw surgery may result in altered sensation and taste ability of the tongue (i.e. lingual nerve). Ask your surgeon for more information about facial sensory loss associated with lower jaw surgery.

_____ **INJURY TO TEETH AND BONE**

Initial here During the orthognathic procedures, adequate circulation to each of the dental and bone segments must be maintained for satisfactory healing. Direct injury to the roots of the teeth or interruption of the circulation to the bone segments can occur. If so, loss of bone is possible. Injury to the teeth (i.e., devitalization) that requires treatment with a root canal is also possible. Additional potential complications include unplanned bone fractures (i.e., fragmentation), delayed healing, sinus obstruction, or openings from the roof of the mouth into either the sinus cavity or nose (i.e., fistulas). These are not common events, but they must be mentioned as possible occurrences.

_____ **FIXATION HARDWARE**

Initial here The use of titanium plate and screw fixation has greatly improved the results of orthognathic surgery and diminished complications such as infection, malocclusion, and relapse. After the jaw bones have healed in their new locations, the plates and screws are no longer needed. In general, however, they are not routinely removed. In approximately 5% of individuals, one or more of the plates or screws may eventually be of concern. If so, these can be removed after healing has occurred. In rare cases, a fixation screw may injure a tooth. If so, the injured tooth may require root canal therapy or even extraction.

_____ **RELAPSE OF THE NEW JAW POSITION**

Initial here With the use of plate and screw fixation across each osteotomy site, unmanageable degrees of relapse (i.e., the return of the jaws toward their preoperative locations) have become much less frequent. Nevertheless, remodeling at the osteotomy sites and of the condylar heads (i.e., the jaw joints) may occur, especially during the initial 6 to 12 months after surgery. When this happens, varying degrees of relapse can occur. Suboptimal occlusion that is appreciated early after the operation is also possible. In some cases, further jaw growth may occur after treatment, which will result in recurrent malocclusion. Despite the potential for these problems, the need for revision osteotomies (i.e., redo jaw surgery) is not frequent.

_____ **TEMPOROMANDIBULAR JOINT PAIN OR DEGENERATION**

Initial here An individual with a jaw deformity and malocclusion may be at greater than average risk for baseline temporomandibular (jaw) joint problems (i.e., pain, popping, clicking, locking, and limited range of motion) as compared with the general population. Orthodontic treatment, restorative dentistry, and orthognathic surgery carried out to improve occlusion should be considered somewhat unpredictable with regard to their overall effect on preexisting temporomandibular joint dysfunction. For some, temporomandibular joint complaints will diminish and function will improve, whereas for others these issues may worsen or not change at all as a result of treatment (e.g. orthodontics, restorative dentistry, orthognathic surgery). Progressive condylar (jaw joint) reabsorption (i.e., the melting away of the bone) is an infrequent cause of relapse after orthodontic treatment and orthognathic surgery. When it does occur, malocclusion results, and further treatment may be required. Ask your surgeon for more information about this rare condition (i.e., idiopathic condylar resorption).

_____ **FACIAL PAIN**

Initial here Individuals with jaw deformities and malocclusion may be at higher risk for baseline facial pain than the general population. The effects of jaw surgery should be considered somewhat unpredictable with regard to their overall effect on facial pain. The changes in occlusion (bite) and altered facial sensation as a result of the jaw surgery are interpreted differently by each individual. For a small percentage of patients (approximately 5%), facial pain or discomfort (i.e., dysesthesia) is persistent and bothersome. Additional treatment or surgery may be possible should facial pain or discomfort persist.

Continued

_____ **DISSATISFACTION WITH FACIAL APPEARANCE**

Initial here An individual may feel dissatisfied with the overall facial appearance achieved after orthodontics and orthognathic (facial) surgery. Despite your surgeon's best efforts, on occasion, this type of surgery can result in visible deformities, loss of function, or structural malposition. You may be disappointed if the results of treatment (e.g., restorative dentistry, orthodontics, orthognathic and facial surgery) do not meet your expectations. Additional treatments and procedures may be possible should the results be suboptimal for you.

_____ **FACIAL ASYMMETRY**

Initial here The human face is normally asymmetric. This asymmetry may be in the upper or lower aspects of the face. It may be in the soft tissues, the skeleton, or both. There can be variations from one side of the face to the other with regard to the results achieved with orthognathic and facial procedures. Additional surgery may be possible should the extent of asymmetry be of concern to you.

_____ **INTRANASAL SURGERY**

Initial here For some individuals, the shape, position and size of the septum of the nose (i.e., the dividing wall between the left and right nasal passages) or the inferior turbinates (i.e., specific structures inside the nose) can interfere with nasal breathing, sinus drainage, or both. When septoplasty (i.e., the removal of bone and cartilage from the septum) and inferior turbinate reduction (i.e., the removal of part of the turbinate) procedures are carried out with a goal of improving breathing and sinus drainage, it is possible that you may experience problems such as bleeding or continued difficulties with the airway and sinus function. There is also the possibility that the septal surgery will cause a perforation (i.e. opening) between the nasal passages to develop. Fortunately, occurrences of these complications are not common. Despite your surgeon's best efforts, a septoplasty or an inferior turbinate operation may not fully achieve the desired result. In some cases, additional treatment or surgery may be helpful to resolve these residual problems.

_____ **REMOVAL OF WISDOM AND OTHER TEETH**

Initial here The extraction of teeth is an irreversible process. As with any surgery, there are associated risks. For example, infection may occur, despite the use of prophylactic antibiotics. Possible damage to adjacent teeth is another concern, especially for those teeth with large fillings or caps. Numbness or altered sensations of the teeth, gums, lips, tongue, and chin may occur as a result of the closeness of the tooth roots to the nerves, which can be bruised or damaged. The incomplete removal of tooth fragments (i.e., small root tips) may be necessary to avoid injury to vital structures such as the nerves or the sinus. A sinus opening or perforation that requires further treatment may occur with the removal of an upper wisdom tooth. Most procedures to remove wisdom teeth are routine. and serious complications are not expected.

_____ **MEDICATIONS**

Initial here Medications, drugs, anesthetics, and prescriptions may cause drowsiness and a lack of awareness and coordination, which can be increased by the use of alcohol or other drugs. Your surgeon advises you not to operate any vehicles, automobiles, or other potentially hazardous devices for 4 weeks after orthognathic surgery and then only after approval is given.

CONSENT FOR ORTHOGNATHIC SURGERY

1. I hereby authorize my surgeon, _____, and such assistants as may be selected by my surgeon to perform the following procedure(s):

   _____

   _____

   _____

   _____

2. I have received, read, and understand the following information sheets:
   - Consent for Orthognathic Surgery
   - Coordination of Orthodontics and Orthognathic Surgery
   - Patient Instructions for Orthognathic Surgery
3. I recognize that, during the course of the operation, unforeseen conditions may necessitate different procedures than those listed above. I therefore authorize my surgeon, _____, and my surgeon's assistants or designees to perform such other procedures that are in the exercise of professional judgment necessary and desirable. The authority granted under this paragraph shall include all conditions that require treatment and that are not known to my surgeon at the time the operation is begun.
4. It has been explained to me that, during the initial healing phase after surgery, I will be using orthodontic rubber bands (elastics) that position my upper and lower teeth together. I agree to be familiar with how to remove the rubber bands. I understand that I may remove the rubber bands at any time if I feel the need to do so.
5. I acknowledge that no guarantee has been given by anyone with regard to the results that may be achieved by the procedures to be carried out.
6. I agree not to use alcoholic beverages and unprescribed drugs, and I have been advised to avoid all sports and water activities for at least 5 (five) weeks and then to resume them only after approval by my surgeon.
7. I agree not to use nicotine or nicotine products for at least 3 weeks before and 2 weeks after surgery.
8. I consent to the photographing of me (head and neck region) before, during, and after surgery. These photographs may be used by my surgeon for medical, scientific, or educational purposes now and in the future.
9. I have provided my surgeon, _____, with all of my past and current medical and dental information. I agree to cooperate completely with all recommendations while I am under my surgeon's care, and I realize that not doing so could result in a less than optimal result.
10. I consent to the disposal of any body tissues or medical devices that may be removed.
11. **THE FOLLOWING INFORMATION HAS BEEN EXPLAINED TO ME IN A WAY THAT I UNDERSTAND:**
    - **THE PREVIOUSLY DESCRIBED PROCEDURES TO BE UNDERTAKEN**
    - **ALTERNATIVE PROCEDURES AND METHODS OF TREATMENT THAT I HAVE DECIDED AGAINST**
    - **THE SPECIFIC RISKS AND POTENTIAL COMPLICATIONS OF THE PROCEDURES THAT I PLAN TO UNDERGO**

I CONSENT TO THE PROCEDURES AND THE ABOVE LISTED ITEMS (1 THROUGH 11). I AM SATISFIED WITH THE EXPLANATION GIVEN TO ME.

_____	_____
Patient (or Person Authorized to Sign for Patient)	Date
_____	_____
Witness	Date

References

1. Abu-Id MH, Warnke PH, Gottschalk J, et al: "Bis-phossy jaws" high and low risk factors for bisphosphonate-induced osteonecrosis of the jaw. *J Craniomaxillofac Surg* 36:95–103, 2008.
2. Acebal-Bianco F, Vuylsteke PLPJ, Mommaerts MY, De Clerq CAS: Perioperative complications in corrective facial orthognathic surgery: A 5-year retrospective study. *J Oral Maxillofac Surg* 58:754–760, 2000.
3. Ahn YS, Kim SG, Baik SM, et al: Comparative study between resorbable and nonresorbable plates in orthognathic surgery, *J Oral Maxillofac Surg* 68:287–292, 2010.
4. Albernaz VS, Tomsick TA: Embolisation of arteriovenous fistulae of the maxillary artery after Le Fort I osteotomy: Report of two cases. *J Oral Maxillofac Surg* 53:208, 1995.
5. Al-Bishri A, Barghash Z, Rosenquist J, et al: Neurosensory disturbance after sagittal split and intraoral vertical ramus osteotomy: As reported in questionnaires and patients' records. *Int J Oral Maxillofac Surg* 34:247, 2005.
6. Al-Bishri A, Rosenquist J, Sunzel B: On neurosensory disturbance after sagittal split osteotomy. *J Oral Maxillofac Surg* 62:1472–1476, 2004.
7. Al-Din OF, Coghlan KM, Magennis P: Sensory nerve disturbance following Le Fort I osteotomy. *Int J Oral Maxillofac Surg* 25:13, 1996.
8. Alpert B, Seligson D: Removal of asymptomatic bone plate used for orthognathic surgery and facial fractures. *J Oral Maxillofac Surg* 54:618, 1996.
9. Alpha C, O'Ryan F, Silva AC, et al: The incidence of postoperative wound healing problems following sagittal ramus osteotomies stabilized with miniplates and monocortical screws. *J Oral Maxillofac Surg* 64:659, 2006.
10. Appiah-Anane S: Amputation neuroma: A late complication following sagittal split osteotomy of the mandible. *J Oral Maxillofac Surg* 49:1218, 1991.
11. Aragon SB, Van Sickels JE, Dolwick MF, et al: The effect of orthognathic surgery on mandibular range of motion. *J Oral Maxillofac Surg* 43:938, 1985.
12. August M, Marchona J, Donady J, Kaban L: Neurosensory deficit and functional impairment after sagittal ramus osteotomy: A long-term follow-up study. *J Oral Maxillofac Surg* 56:1231–1235, 1998.
13. Ayoub AF, Lalani Z, Moos KF, et al: Complications following orthognathic surgery that required early surgical intervention: Fifteen years' experience. *Int J Adult Orthodon Orthognath Surg* 16:138, 2001.
14. Aziz SR, Agnihotri N, Ziccardi VB: Lobar collapse immediately after orthognathic surgery. *J Oral Maxillofac Surg* 68:2335, 2010.
15. Baddour HM, Watson J, Erwin BJ: Life-threatening hemorrhage from a Le Fort I osteotomy. *J Oral Maxillofac Surg* 40:117, 1982.
16. Baddour HM, Watson J, Erwin BJ: Injuries to the internal carotid artery following orthognathic surgery. *J Oral Maxillofac Surg* 3:215, 1988.
17. Bagheri S, Meyer RA, Khan HA, et al: Microsurgical repair of the peripheral trigeminal nerve after mandibular sagittal split ramus osteotomy. *J Oral Maxillofac Surg* 68:2770–2782, 2010.
18. Bailey PH, Bays RA: Evaluation of long-term sensory changes following mandibular augmentation procedures. *J Oral Maxillofac Surg* 42:722, 1984.
19. Baker SB, Weinzweig J, Bartlett SP, et al: Brain abscess as a complication of orthognathic surgery: Diagnosis, management and pathophysiology. *Plast Reconstr Surg* 104:480, 1999.
20. Baqain ZH, Hyde N, Patrikidou A, et al: Antibiotic prophylaxis for orthognathic surgery: A prospective, randomized clinical trial. *Br J Oral Maxillofac Surg* 42:506, 2004.
21. Becelli R, Renzi G, Carbomni A, et al: Inferior alveolar nerve impairment after mandibular sagittal split osteotomy: An analysis of spontaneous recovery patterns observed in 60 patients. *J Craniofac Surg* 13:315–320, 2002.
22. Becelli RB, Fini G, Renzi G, et al: Complications of bicortical screw fixation observed in 482 mandibular sagittal osteotomies. *J Craniofac Surg* 15:64–68, 2004.
23. Behrman SJ: Complications of sagittal osteotomy of the mandibular ramus. *J Oral Surg* 30:554, 1972.
24. Bell RB, Phillips C, Manente SJ: Patients' expectations following video imaging prior to orthognathic surgery. *J Oral Maxillofac Surg* 55 (supplement 3):88–89, 1997.
25. Bell WH: Biologic basis for maxillary osteotomies. *Am J Phys Anthropol* 38:279, 1973.
26. Bell WH, Schendel SA: Biologic basis for modification of the sagittal ramus split operation. *J Oral Surg* 35:362–369, 1977.
27. Bell WH, You ZH, Finn RA, et al: Wound healing after multisegmental Le Fort I osteotomy and transection of the descending palatine vessels. *J Oral Maxillofac Surg* 53:1425, 1995.
28. Bendor-Samuel R, Chen YR, Chen PK: Unusual complications of the Le Fort I osteotomy. *Plast Reconstr Surg* 96:1289, 1995.
29. Bentley KC, Head TW, Aiello GA: Antibiotic prophylaxis in orthognathic surgery: A 1 day versus 5-day regimen. *J Oral Maxillofac Surg* 57:226, 1999.
30. Bercault N, Boulain T, Kuteifan K, et al: Obesity-related excess mortality rate in an adult intensive care unit: A risk-adjusted matched cohort study. *Crit Care Med* 32:998–1003, 2004.
31. Berlin R: *Injuries to the optic nerve and ophthalmic artery from fracture of the optic canal*, London, 1881, Klockman, p 154.
32. Bhaskaran AA, Courtney DJ, Anand P, et al: A complication of Le Fort I osteotomy. *Int J Oral Maxillofac Surg* 39:292, 2010.

32A. Bhatt V, Langford RJ: Removal of miniplates in maxillofacial surgery: University Hospital Birmingham experience. *J Oral Maxillofac Surg* 61:553–556, 2003.

33. Bigoli SJ, Dumont L, Mattys M, et al: A serious anesthetic complication of a Le Fort I osteotomy. *Eur J Anaesthesiol* 16:201, 1999.
34. Blackburn TK, Pritchard K, Richardson D: Symptomatic venous thromboembolism after orthognathic operations: An audit. *Br J Oral Maxillofac Surg* 44:389, 2006.
35. Blakey GH, 3rd, Zuniga JR: Lingual nerve injury associated with superior border wire fixation. *Int J Adult Orthodon Orthognath Surg* 7:115, 1992.
36. Blinder D, Rotenberg L, Taicher S: Conversion disorder after maxillofacial trauma and surgery. *Int J Oral Maxillofac Surg* 25:116, 1996.
37. Blomqvist JE, Alberius P, Isaksson S: Sensibility following sagittal split osteotomy in the mandible: A prospective clinical study. *Plast Reconstr Surg* 102:325–333, 1998.
38. Bloomquist DS: Intraoperative assessment of maxillary perfusion during Le Fort I osteotomy (discussion). *J Oral Maxillofac Surg* 52:831, 1994.
39. Bouwman JPB, Husak A, Putman GD, et al: Screw fixation following bilateral sagittal ramus osteotomy for mandibular advancement—complications in 700 consecutive cases. *Br J Oral Maxillofac Surg* 33:231–234, 1995.
40. Brady S, Courtemanche A, Steinbok P: Carotid artery thrombosis after elective mandibular and maxillary osteotomies. *Ann Plast Surg* 6:121, 1981.
41. Braun TW, Sotereanos GC: Vascular changes in the pterygopalatine fossa after craniofacial dysjunction surgery. *J Oral Surg* 37:88, 1979.
42. Broadbend TR, Woolf RM: Our experience with sagittal split osteotomy for retrognathia. *Plast Reconstr Surg* 60:860, 1977.
43. Brown RH, Schauble JF, Miller NR: Anemia and hypotension as contributors to perioperative loss of vision. *Anesthesiology* 80:222, 1994.
44. Browne RM, Brady CL, Frame JW: Tooth pulp changes following Le Fort I maxillary osteotomy in a primate model. *Br J Oral Maxillofac Surg* 28:1, 1990.
45. Brusati R, Fiamminghi L, Sesenna E, et al: Functional disturbances of the inferior

alveolar nerve in sagittal osteotomy of mandibular ramus: Operating technique for prevention. *J Maxillofac Surg* 9:123, 1981.
46. Buijs GJ, Van Der Houwen EB, Stegenga B, et al: Mechanical strength and stiffness of biodegradable and titanium osteofixation systems. *J Oral Maxillofac Surg* 65:2148, 2007.
47. Buijs GJ, van der Houwen EB, Stegenga B, et al: Torsion strength of biodegradable and titanium screws: A comparison. *J Oral Maxillofac Surg* 65:2142, 2007.
48. Burden DW: Principles of antimicrobial prophylaxis. *World J Surg* 6:262, 1981.
49. Burns SJ, Dippe SE: Postoperative wound infections detected during hospital stay and after discharge in a community hospital. *Am J Infect Control* l:60, 1982.
50. Calle EE, Thun MJ, Petrelli JM, et al: Body-mass index and mortality in a prospective cohort of US adults. *N Eng J Med* 341:1097–1105, 1999.
51. Campbell RL, Shamaskin RG, Harkins SW: Assessment of recovery from injury to inferior alveolar and mental nerves. *Oral Surg Oral Med Oral Pathol* 64:519, 1987.
52. Canavan D, Graff-Radford S, Gratt BM: Traumatic dysesthesia of the trigeminal nerve. *J Orofac Pain* 8:391, 1994.
53. Caplan RA, Posner KL, Ward RJ, et al: Adverse respiratory events in anesthesia: A closed analysis. *Anesthesiology* 72:828, 1990.
54. Carr RJ, Gilbert P: Isolated partial third nerve palsy following Le Fort I maxillary osteotomy in a patient with cleft lip and palate. *Br J Oral Maxillofac Surg* 24:206, 1986.
55. Chau MNY, Jönsen E, Lee KM: Traumatic neuroma following sagittal mandibular osteotomy. *Int J Oral Maxillofac Surg* 18:95, 1989.
56. Chebel NA, Ziade D, Achkouty R: Bilateral pneumothorax and pneumomediastinum after treatment with continuous positive airway pressure after orthognathic surgery. *Br J Oral Maxillofac Surg* 48:e14, 2010.
57. Chen N, Neal CE, Lingenbrink P, et al: Neurosensory changes following orthognathic surgery. *Int J Adult Orthodon Orthognath Surg* 14:259, 1999.
58. Chen YR, Fisher DM: Blindness as a complication of Le Fort I osteotomies: Role of atypical fracture patterns and distortion of the optic canal (discussion). *Plast Reconstr Surg* 102:1422, 1998.
59. Cheng HC, Chi LH, Wu JY, et al: Blindness and basal ganglia hypoxia as a complication of Le Fort I osteotomy attributable to hypoplasia of the internal carotid artery: A case report. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 104:e27, 2007.
60. Choi BK, Goh RCW, Chen PKT, et al: Facial nerve palsy after sagittal split ramus osteotomy of the mandible: Mechanism and outcomes. *J Oral Maxillofac Surg* 68:1615–1621, 2010.
61. Chow LK, Singh B, Chiu WK, et al: Prevalence of postoperative complications after orthognathic surgery: A 15 year review. *J Oral Maxillofac Surg* 65:984, 2007.
62. Chua W, Chidambaram A, Doyle PT, et al: Voicing concern: An unusual sequelae of orthognathic surgery. *Prog Orthod* 7:220, 2006.
63. Clark CL, Baur DA: Management of mentalis muscle dysfunction after advancement genioplasty: a case report. *J Oral Maxillofac Surg* 62:611, 2004.
64. Clark R, Lew D, Giyanani VL, et al: False aneurysm complicating orthognathic surgery. *J Oral Maxillofac Surg* 45:57, 1987.
65. Coghlan KM, Irvine GH: Neurological damage after sagittal split osteotomy. *Int J Oral Maxillofac Surg* 15:369–371, 1986.
66. Colella G, Cannavale R, Vicidomini A, et al: Neurosensory disturbance of the inferior alveolar nerve after bilateral sagittal split osteotomy: A systematic review. *J Oral Maxillofac Surg* 65:1707, 2007.
67. Connolly SE, Gordon KB, Horton JC: Salvage of vision after hypotension-induced ischemic optic neuropathy. *Am J Ophthalmol* 117:235, 1994.
68. Consolo U, Salgarelli A: Transient facial nerve palsy following orthognathic surgery: A case report. *J Oral Maxillofac Surg* 50:77, 1992.
69. Costas PD, Heatley G, Seckel BR: Normal sensation of the human face and neck. *Plast Reconstr Surg* 93:1141, 1994.
70. Cruz AA, dos Santos AC: Blindness after Le Fort I osteotomy: A possible complication associated with pterygomaxillary separation. *J Craniomaxillofac Surg* 34:210, 2006.
71. Cunningham LL, Jr, Tiner BD, Clark GM, et al: A comparison of questionnaire versus monofilament assessment of neurosensory deficit. *J Oral Maxillofac Surg* 54:454–459, 1996.
72. Cunningham SJ, Crean SJ, Hunt NP, Harris M: Preparation, perception and problem: A long term follow up of orthognathic surgery. *Int J Adult Orthodon Orthognath Surg* 11:41, 1996.
73. Cunningham SJ, Feinmann C, Horrocks EN: Psychological problems following orthognathic surgery. *J Clin Orthod* 29:755, 1995.
74. D'Orta JA, Shatney CH: Post-traumatic pseudoaneurysm of the internal maxillary artery. *J Trauma* 22:161, 1982.
75. Daftari TK, Whitesides TE, Heller JG, et al: Nicotine on the revascularization of bone graft: An experimental study in rabbits. *Spine* 19:904, 1994.
76. Dan AE, Thygesen TH, Pinholt EM: Corticosteroid administration in oral and orthognathic surgery: A systematic review of the literature and meta-analysis. *J Oral Maxillofac Surg* 68:2207–2220, 2010.
77. Danda AK, Ravi P: Effectiveness of postoperative antibiotics in orthognathic surgery: A meta-analysis. *J Oral Maxillofac Surg* 69:2650–2656, 2011.
78. Danda AK, Wahab A, Narayanan V, Siddareddi A: Single-dose versus single-day antibiotic prophylaxis for orthognathic surgery: A prospective, randomized, double-blind clinical study. *J Oral Maxillofac Surg* 68:344–346, 2010.
79. Dendy RA: Facial nerve paralysis following sagittal split mandibular osteotomy: A case report. *Br J Oral Surg* 11:101, 1973.
80. De Mop van Otterloo JJ, Tuinzing DB, Greebe RB, et al: Intra and early postoperative complications of the Le Fort I osteotomy: A retrospective study on 410 cases. *J Craniomaxillofac Surg* 19:217, 1991.
81. De Sousa A: Psychological issues in oral and maxillofacial reconstructive surgery. *J Oral Maxillofac Surg* 46:661, 1998.
82. Dervis E, Tuncer E: Long-term evaluations of temporomandibular disorders in patients undergoing orthognathic surgery compared with a control group. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 94:554, 2002.
83. De Vries K, Devriese PP, Hovinga J, et al: Facial palsy after sagittal split osteotomies: A survey of 1747 sagittal split osteotomies. *J Craniomaxillofac Surg* 21:50, 1993.
84. Dimitroulis G: Complications of orthognathic surgery. *Aust Orthod J* 14:158, 1996.
85. Dimopoulos MA, Kastritis E, Anagnostopoulos A, et al: Osteonecrosis of the jaw in patients with multiple myeloma treated with bisphosphonates: Evidence of increased risk after treatment with zoledronic acid. *Haematologica* 91:968–971, 2006.
86. Dodson TB, Kaban LB: Recommendations for management of trigeminal nerve defects based on a critical appraisal of the literature. *J Oral Maxillofac Surg* 55:1380, 1997.
87. Dodson TB, Neuenschwander MC: Maxillary perfusion during Le Fort I osteotomy after ligation of the descending palatine artery. *J Oral Maxillofac Surg* 55:51, 1997.
88. Donoff RB: Surgical management of inferior alveolar nerve injuries (Part I): The case for early repair. *J Oral Maxillofac Surg* 53:1327, 1995.
89. Doucet JC, Morrison AD, Davis BR, et al: The presence of mandibular third molars during sagittal split osteotomies does not increase the risk of complications. *J Oral Maxillofac Surg* 70:1935–1943, 2012.
90. Doucet JC, Morrison AD, Davis BR, et al: Concomitant removal of mandibular third molars during sagittal split osteotomy minimizes neurosensory dysfunction. *J Oral Maxillofac Surg* 70:2153–2163, 2012.
91. Edwards DB, Scheffler RB, Jackler I: Postoperative pneumomediastinum and pneumothorax following orthognathic surgery. *J Oral Maxillofac Surg* 44:137, 1986.
92. Edwards RC, Kiely KD, Eppley BL: The fate of resorbable poly-L-lactic/polyglycolic acid (LactoSorb) bone fixation devices in orthognathic surgery. *J Oral Maxillofac Surg* 59:19, 2001.
93. El Deeb M, Wolford L, Bevis R: Complications of orthognathic surgery. *Clin Plast Surg* 16:825, 1989.

94. Ellis E, III, Dechow PC, Carlson DS: A comparison of stimulated bite force after mandibular advancement using rigid and non rigid fixation. *J Oral Maxillofac Surg* 46:26, 1988.
95. El-Zawawy HB, Gill CS, Wright RW, et al: Smoking delays chondrogenesis in a mouse model of closed tibial fracture healing. *J Orthop Res* 24:2150, 2006.
96. Eppley BL: Bioabsorbable plate and screw fixation in orthognathic surgery. *J Craniofac Surg* 18:818, 2007.
96A. Espeland L, Hogevold HA, Stenvik A: A 3-year patient-centered follow-up of 516 consecutively treated orthognathic surgery patients. *Eur J Orthod* 30:24–30, 2008.
97. Essick GK: Comprehensive clinical evaluation of perioral sensory function. *Oral Maxillofac Surg Clin North Am* 4:503, 1992.
98. Essick GK, Austin S, Phillips C, et al: Short-term sensory impairment after orthognathic surgery. *Oral Maxillofac Surg Clin North Am* 13:295, 2001.
99. Essick GK, Phillips C, Turvey TA, et al: Facial altered sensation and sensory impairment after orthognathic surgery. *Int J Oral Maxillofac Surg* 36:577, 2007.
100. Evans GRD, Crawley W, Dellon AL: Inferior alveolar nerve grafting: An approach without intermaxillary fixation. *Ann Plast Surg* 33:221, 1994.
101. Ferdousi AM, MacGregor AJ: The response of the peripheral branches of the trigeminal nerve to trauma. *Int J Oral Surg* 14:41, 1985.
102. Fiamminghi L, Aversa C: Lesions of the inferior alveolar nerve in sagittal osteotomy of the ramus: Experimental study. *J Maxillofac Surg* 7:125, 1979.
103. Flynn NM, Lawrence RM: Antimicrobial prophylaxis. *Med Clin North Am* 63:1225, 1979.
104. Francel TJ, Birely BC, Ringelman PR, Manson PN: The fate of plates and screws after facial fracture reconstruction. *Plast Reconstr Surg* 90:568, 1992.
105. Fridrich KL: Discussion of Zijderveld, et al: Preoperative antibiotic prophylaxis: A randomised, double-blind and placebo-controlled clinical study. *J Oral Maxillofac Surg* 57:1406–1407, 1999.
106. Fridrich KL, Holton TJ, Pansegrau KJ, Berckley MJ: Neurosensory recovery following the mandibular bilateral sagittal split osteotomy. *J Oral Maxillofac Surg* 53:1300–1306, 1995.
107. Fridrich KL, Partnoy BE, Williamson LW: Closed intraoral wound suction: A prospective analysis of 65 cases and a review of the literature. *Int J Adult Orthodon Orthognath Surg* 5:183–187, 1990.
108. Fridrich KL, Partnoy BE, Zeitler D: Prospective analysis of antibiotic prophylaxis for orthognathic surgery. *Int J Adult Orthodon Orthognath Surg* 9:129–131, 1994.
109. Fujioka M, Hirano A, Fujii T: Comparative study of inferior alveolar disturbance restoration after sagittal split osteotomy by means of bicortical versus monocortical osteosynthesis. *Plast Reconstr Surg* 102:37–41, 1998.
110. Gallagher DM, Epker BN: Infection following intraoral surgical correction of dentofacial deformities. *J Oral Surg* 38:117–120, 1980.
111. Garner JS, Jarvis WR, Emori TG, et al: CDC definitions for nosocomial infections 1988. *Am J Infect Control* 16:128, 1988.
112. Geha HJ, Gleizal AM, Nimeskern NJ, et al: Sensitivity of the inferior lip and chin following mandibular bilateral sagittal split osteotomy using piezosurgery. *Plast Reconstr Surg* 118:1598, 2006.
113. Gent JF, Shafer DM, Frank ME: The effect of orthognathic surgery on taste function on the palate and tongue. *J Oral Maxillofac Surg* 61:766, 2003.
114. Gianni AB, D'Orto O, Biglioli F, et al: Neurosensory alterations of the inferior alveolar and mental nerve after genioplasty alone or associated with sagittal osteotomy of the mandibular ramus. *J Craniomaxillofac Surg* 30:295, 2002.
115. Girotto JA, Davidson J, Wheatly M, et al: Blindness as a complication of Le Fort osteotomies: Role of atypical fracture patterns and distortion of the optic canal. *Plast Reconstr Surg* 102:1409, 1998.
116. Goffinet L, Laure B, Tayeb T, et al: An arteriovenous fistula of the maxillary artery as a complication of Le Fort I osteotomy. *J Craniomaxillofac Surg* 38:251, 2010.
117. Goodson ML, Manemi R, Paterson AW: Pneumothorax after orthognathic surgery. *Br J Oral Maxillofac Surg* 48:180, 2010.
118. Grammer FC, Meyer MW, Ricater KC: A radioisotope study of the vascular response to sagittal split osteotomy of the mandibular ramus. *J Oral Surg* 32:578, 1974.
119. Greenberg RN, James RN, Marier RL, et al: Microbiologic and antibiotic aspects of infections in the oral and maxillofacial region. *J Oral Surg* 37:873, 1979.
120. Gregg JM: Studies of traumatic neuralgias in the maxillofacial region: Surgical pathology and neural mechanisms. *J Oral Maxillofac Surg* 48:228, 1990.
121. Gregg JM: Surgical management of inferior alveolar nerve injuries (Part II): The case for delayed management. *J Oral Maxillofac Surg* 53:1330, 1995.
122. Grime PD, Tyler C: An obstructed airway: Cuff herniation during nasotracheal anesthesia for a bimaxillary osteotomy. *Br J Oral Maxillofac Surg* 29:14, 1991.
123. Gruber EA, Bhaskaran A, Anand P, et al: A complication of Le Fort I osteotomy—a case report and review of the literature. *Br J Oral Maxillofac Surg* 46:e25, 2008.
124. Gunaseelan R, Anantanarayanan P, Veerabahu M, et al: Intraoperative and perioperative complications in anterior maxillary osteotomy: A retrospective evaluation of 103 patients. *J Oral Maxillofac Surg* 67:1269, 2009.
125. Gutta R, Louis PJ, Birmingham AL: Bisphosphonates and osteonecrosis of the jaws: Science and rationale. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 104:186–189, 2007.
126. Habal M: A carotid cavernous sinus fistula after maxillary osteotomy. *Plast Reconstr Surg* 77:981, 1986.
127. Haers PE, Sailer HF: Biodegradable self-reinforced poly-L/DL-lactide plate and screws in bimaxillary orthognathic surgery: short term skeletal stability and material related failures. *J Craniomaxillofac Surg* 26:363, 1998.
128. Hall HD: *Apparent ischemic necrosis after maxillary osteotomy: Report of three cases. Case reports and outlines of selected scientific session.* Presented at the 60th Annual Meeting of the American Association of Oral and Maxillofacial Surgeons, Chicago, Ill, 1978, pp 43.
129. Hanu-Cernat LM, Hall T: Late onset of abducens palsy after Le Fort I maxillary osteotomy. *Br J Oral Maxillofac Surg* 47:416, 2009.
130. Harada K, Enomoto S: Stability after surgical correction of mandibular prognathism using the sagittal split ramus osteotomy and fixation with poly-L-lactic acid screws. *J Oral Maxillofac Surg* 55:464, 1997.
131. Haug RH: Clinical controversies in oral and maxillofacial surgery: I. Retention of asymptomatic bone plates used for orthognathic surgery and facial fractures. *J Oral Maxillofac Surg* 54:611, 1996.
132. Hegtvedt AK, Zuniga JR: Lingual nerve injury as a complication of rigid fixation of the sagittal ramus osteotomy: Report of a case. *J Oral Maxillofac Surg* 48:647, 1990.
133. Heit JM, Farhood VW, Edwards RC: Survey of antibiotic prophylaxis for intraoral orthognathic surgery. *J Oral Maxillofac Surg* 49:340–342, 1991.
134. Hemmig S, Johnson RS, Ferraro N: Management of a ruptured pseudoaneurysm of the sphenopalatine artery following a Le Fort I osteotomy. *J Oral Maxillofac Surg* 45:533, 1987.
135. Herold J, Falworth M: Sub-total unilateral oculomotor nerve palsy in Le Fort I osteotomy. *Br J Oral Maxillofac Surg* 34:104, 1996.
136. Hes J, de Man K: Carotid-cavernous sinus fistula following maxillofacial trauma and orthognathic surgery. *Int J Oral Maxillofac Surg* 17:295, 1988.
137. Hollinger JO, Schmitt JM, Hwang K, et al: Impact of nicotine on bone healing. *J Biomed Mater Res* 45:294, 1999. Erratum in J Biomed Mat Res 46:438, 1999.
138. Hori M, Okaue M, Hasegawa M, et al: Worsening of pre-existing TMJ dysfunction following sagittal split osteotomy: A study of three cases. *J Oral Sci* 41:133, 1999.
139. Hovinga J, Kraal ER, Roorda LAM: A follow-up of osteotomies for dysgnathia. *J Maxillofac Surg* 7:271, 1979.

140. Humber CC, Lanigan DT, Hohn FI: Retrograde hemorrhage (hemolacria) from the lacrimal puncta after a Le Fort I osteotomy: A report of 2 cases and a review of the literature. *J Oral Maxillofac Surg* 69:520, 2011.
141. Jaaskelainen SK, Peltola JK, Forssell K, et al: Evaluating function of the inferior alveolar nerve with repeated nerve conduction tests during mandibular sagittal split osteotomy. *J Oral Maxillofac Surg* 53:269, 1995.
142. Jaaskelainen SK, Peltola JK, Lehtinen R: The mental nerve blink reflex in the diagnosis of lesions of the inferior alveolar nerve following orthognathic surgery of the mandible. *Br J Oral Maxillofac Surg* 34:87, 1996.
143. Jaaskelainen SK, Teerijoki-Oksa T, Forssell K, et al: Intraoperative monitoring of the inferior alveolar nerve during mandibular sagittal-split osteotomy. *Muscle Nerve* 23:368, 2000.
144. Jaaskelainen SK, Teerijoki-Oksa T, Virtanen A, et al: Sensory regeneration following intraoperatively verified trigeminal nerve injury. *Neurology* 62:1951, 2004.
145. Jacks SC, Zuniga JR, Turvey TA, et al: A retrospective analysis of lingual nerve sensory changes after mandibular bilateral sagittal split osteotomy. *J Oral Maxillofac Surg* 56:700, 1998.
146. Jackson L: Outpatient orthognathic surgery: Review of 205 cases (discussion). *J Oral Maxillofac Surg* 55:563, 1997.
147. Jerjes W, Swinson B, Moles DR, et al: Permanent sensory nerve impairment following third molar surgery: A prospective study. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 102:e1, 2006.
148. Johnson MW, Kincaid MC, Trobe JD: Bilateral retrobulbar optic nerve infarctions after blood loss and hypotension: A clinicopathologic case study. *Ophthalmology* 94:1577, 1987.
149. Jones JK, Triplett RG: The relationship of cigarette smoking to impaired intraoral wound healing: A review of evidence and implications for patient care. *J Oral Maxillofac Surg* 50:237, 1992.
150. Jones JK, Van Sickels JE: Facial nerve injuries associated with orthognathic surgery: A review of incidence and management. *J Oral Maxillofac Surg* 49:740, 1991.
151. Jorgenson DS, Mayer MH, Ellenbogen RG, et al: Detection of titanium in human tissues after craniofacial surgery. *Plast Reconstr Surg* 99:976, 1997.
152. Junior SML, Granato R, Marin C, et al: Analysis of 40 cases of intraoral verticosagittal ramus osteotomies to treat dentofacial deformities. *J Oral Maxillofac Surg* 67:1840, 2009.
153. Kademani D, Voiner JL, Quinn PD: Acute hypertensive crisis resulting in pulmonary edema and myocardial ischemia during orthognathic surgery. *J Oral Maxillofac Surg* 62:240, 2004.
154. Kahnberg KE, Engstrom H: Recovery of maxillary sinus and tooth sensibility after Le Fort I osteotomy. *Br J Oral Maxillofac Surg* 25:68, 1987.
155. Kallela I, Laine P, Suuronen R, et al: Assessment of material- and technique-related complications following sagittal split osteotomies stabilized by biodegradable polylactide screws. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 99:4, 2005.
156. Karabouta J, Martis C: The TMJ dysfunction syndrome before and after sagittal split osteotomy of the rami. *J Maxillofac Surg* 13:185, 1985.
157. Karas ND, Boyd SB, Sinn DP: Recovery of neurosensory function following orthognathic surgery. *J Oral Maxillofac Surg* 48:124, 1990.
158. Keighley MRB: A rational approach to antimicrobial prophylaxis in surgery. *World J Surg* 6:327, 1982.
159. Keiser GJ, Bozentka NE, Gold BD: Laryngeal granuloma: A complication of prolonged endotracheal intubation. *Anesth Prog* 38:232, 1991.
160. Kersarwani A, Antonyshyn O, Mackinnon SE, et al: Facial sensibility testing in the normal and post traumatic population. *Ann Plast Surg* 22:416, 1989.
161. Kim H-S, Kho H-K, Kim S-W, et al: Reliability and characteristics of current perception thresholds in the territory of the infraorbital and inferior alveolar nerves. *J Orofac Pain* 14:286, 2000.
162. Kim SG, Park SS: Incidence of complications and problems related to orthognathic surgery. *J Oral Maxillofac Surg* 65:2438, 2007.
163. Kim YG, Oh SH: Effect of mandibular setback surgery on occlusal force. *J Oral Maxillofac Surg* 55:121, 1997.
164. Kim YK, Yeo HH, Lim SC: Tissue response to titanium plate: A transmitted electron microscopic study. *J Oral Maxillofac Surg* 55:322, 1997.
165. Kin BC, Padwa BL, Park HS, Jung YS: Stability of maxillary position after Le Fort I osteotomy using self-reinforced biodegradable poly-70L/30DL-lactide miniplates and screws. *J Oral Maxillofac Surg* 69:1442–1446, 2011.
166. Kipp DP, Goldstein BH, Weiss WW: Dysesthesia after mandibular third molar surgery in retrospective study and analysis of 1,377 surgical procedures. *J Am Dent Assoc* 100:185, 1980.
167. Kiyak HA, McNeill RW, West RA: The emotional impact of orthognathic surgery and conventional orthodontics. *Am J Orthod* 88:224, 1985.
168. Klemm E, Stösslein F, Mürbe B: Arteriovenous fistula of the maxillary artery, eustachian tube dysfunction and tinnitus after Le Fort I osteotomy [article in German]. *HNO* 49:216, 2001.
169. Kobayashi A, Yoshimasu H, Kobayashi J, et al: Neurosensory alteration in the lower lip and chin area after orthognathic surgery: Bilateral sagittal split osteotomy versus inverted L ramus osteotomy. *J Oral Maxillofac Surg* 64:778, 2006.
170. Kohno M, Nakajima T, Someya G: Effects of maxillomandibular fixation on respiration. *J Oral Maxillofac Surg* 51:992, 1993.
171. Kooke R, Egyedi P: Postoperative contamination of mandibular osteotomy sites with saliva. *Int J Oral Maxillofac Surg* 16:554, 1987.
172. Kopelman PG: Obesity as a medical problem. *Nature* 404:635–643, 2000.
173. Kramer FJ, Baethge C, Swennen G, et al: Intra-and perioperative complications of the Le Fort I osteotomy: A prospective evaluation of 1000 patients. *J Craniofac Surg* 15:971, 2004.
174. Kriwalsky MS, Maurer P, Veras RB, et al: Risk factors for a bad split during sagittal split osteotomy. *Br J Oral Maxillofac Surg* 46:177–179, 2008.
175. Krobbuaban B, Diregpoke S, Kumkeaw S, et al: The predictive value of the height ratio and thyromental distance: Four predictive tests for difficulty laryngoscopy. *Anesth Analg* 101:1542, 2005.
176. Krueger JK, Rohrich RJ: Clearing the smoke: The scientific rationale for tobacco abstention with plastic surgery. *Plast Reconstr Surg* 108:1063, 2001.
176A. Kuhlefelt M, Laine P, Suominen AL, et al: Smoking as a significant risk factor for infections after orthognathic surgery. *J Oral Maxillofac Surg* 70:1643–1647, 2012.
177. Lai JP, Hseih CH, Chen YR, et al: Unusual late vascular complications of sagittal split osteotomy of the mandibular ramus. *J Craniofac Surg* 16:664, 2005.
178. Landes CA, Ballon A: Skeletal stability in bimaxillary orthognathic surgery: P(L/DL) LA-resorbable versus titanium osteofixation. *Plast Reconstr Surg* 118:703, 2006.
179. Lanigan DT: Wound healing after multisegmental Le Fort I osteotomy and transection of the descending palatine vessels (discussion). *J Oral Maxillofac Surg* 53:1433, 1995.
180. Lanigan DT: The inherent risks of the pterygomandibular dysjunction and maxillary downfracture. *Jpn J Jaw Deform* 10:158, 2000.
181. Lanigan DT, Guest P: Alternative approaches to pterygomaxillary separation. *Int J Oral Maxillofac Surg* 22:131, 1993.
182. Lanigan DT, Hey JH, West RA: Aseptic necrosis following maxillary osteotomies: Report of 36 cases. *J Oral Maxillofac Surg* 48:142, 1990.
183. Lanigan DT, Hey JH, West RA: Major vascular complications of orthognathic surgery: Hemorrhage associated with Le Fort I osteotomies. *J Oral Maxillofac Surg* 48:561, 1990.
184. Lanigan DT, Hey JH, West RA: Hemorrhage following mandibular osteotomies: A report of 21 cases. *J Oral Maxillofac Surg* 49:713, 1991.

185. Lanigan DT, Hey JH, West RA: Major vascular complications of orthognathic surgery: False aneurysms and arteriovenous fistulas following orthognathic surgery. *J Oral Maxillofac Surg* 49:571, 1991.
186. Lanigan DT, Hohn FI: Facial nerve injuries after sagittal split mandibular ramus osteotomies for advancement: A report of 2 cases and review of the literature. *J Oral Maxillofac Surg* 62:503, 2004.
187. Lanigan DT, Loewy J: Postoperative computed tomography scan study of the pterygomaxillary separation during the Le Fort I osteotomy using a micro-oscillating saw. *J Oral Maxillofac Surg* 53:1161, 1995.
188. Lanigan DT, Romanchuk K, Olson CK: Ophthalmic complications associated with orthognathic surgery. *J Oral Maxillofac Surg* 51:480, 1993.
189. Lanigan DT, Tubman DE: Carotid-cavernous sinus fistula following Le Fort I osteotomy. *J Oral Maxillofac Surg* 45:969, 1987.
190. Lanigan DT, West RA: Management of postoperative hemorrhage following the Le Fort I maxillary osteotomy. *J Oral Maxillofac Surg* 42:367, 1984.
191. Lanigan DT, West RA: Aseptic necrosis of the mandible: Report of two cases. *J Oral Maxillofac Surg* 48:296, 1990.
192. Laureano-Filho JR, de Oliveiro Neto PJ, Duarte N, et al: Successful management of malignant hyperthermia during orthognathic surgery: A case report. *J Oral Maxillofac Surg* 66:1485, 2008.
193. Lee JG, Kim SG, Lim KJ, et al: Thermographic assessment of inferior alveolar nerve injury in patients with dentofacial deformity. *J Oral Maxillofac Surg* 65:74, 2007.
194. Lee SH, Lee SH, Mori Y, et al: Evaluation of pterygomaxillary anatomy using computed tomography: Are there any structural variations in cleft patients? *J Oral Maxillofac Surg* 69:2644–2649, 2011.
195. Leira JI, Gillhuus-Moe OT: Sensory impairment following sagittal split osteotomy for correction of mandibular retrognathism. *Int J Adult Orthodon Orthognath Surg* 6:161–167, 1991.
196. Lemke RR, Clark GM, Bays RA, et al: Effects of hypesthesia on oral behaviors of the orthognathic surgery patient. *J Oral Maxillofac Surg* 56:153, 1998.
197. Lemke RR, Rugh JD, Van Sickels J, et al: Neurosensory differences after wire and rigid fixation in patients with mandibular advancement. *J Oral Maxillofac Surg* 59:1354–1359, 2000.
198. Lenz JH, Steiner-Krammer B, Schmidt W, et al: Does avascular necrosis of the jaws in cancer patients only occur following treatment with bisphosphonates? *J Craniomaxillofac Surg* 33:395–403, 2005.
199. Li KK, Meara JG, Rubin PAD: Orbital compartment syndrome following orthognathic surgery. *J Oral Maxillofac Surg* 53:964, 1995.
200. Li KK, Stephens W: Fractures of the atrophic, edentulous maxilla during Le Fort I osteotomy. *Int J Oral Maxillofac Surg* 25:430, 1996.
201. Lindeboom JA, Baas EM, Kroon FH: Prophylactic single-dose administration of 600 mg clindamycin versus 4-time administration of 600 mg clindamycin in orthognathic surgery: A prospective randomised study in bilateral mandibular sagittal ramus osteotomies. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 95:145–149, 2003.
202. Little C, Mintz S, Ettinger AC: The distal lacrimal ductal system and traumatic epiphora. *Int J Oral Maxillofac Surg* 20:31, 1991.
203. Lo LJ, Hung KF, Chen YR: Blindness as a complication of Le Fort I osteotomy for maxillary distraction. *Plast Reconstr Surg* 109:688, 2002.
204. Lowry JC: Thromboembolic disease and thromboprophylaxis in oral and maxillofacial surgery: Experience and practice. *Br J Oral Maxillofac Surg* 33:101, 1995.
205. Lustbader DP, Schwartz MH, Zito J, et al: The use of percutaneous transcatheter embolisation to control postoperative bleeding following Le Fort I osteotomy: Report of three cases. *J Oral Maxillofac Surg* 49:426, 1991.
206. Macintosh RB: Experience with the sagittal osteotomy of the mandibular ramus: A 13 year review. *J Oral Maxillofac Surg* 9:151, 1981.
207. Madani M, Veznedaroglu E, Pazoki A, et al: Pseudoaneurysm of the facial artery as a late complication of bilateral sagittal split osteotomy and facial trauma. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 110:579, 2010.
208. Mainous EG, Crowell NT: Nasal septum perforation following total maxillary osteotomy: Report of a case. *J Oral Surg* 31:869, 1973.
209. Malmgren B, Aström E, Soderhall S: No osteonecrosis in jaws of young patients with osteogenesis imperfecta treated with bisphosphonates. *J Oral Pathol Med* 37:196–200, 2008.
210. Manikandhan R, Anantanarayanan P, Mathew PC, et al: Incidence and consequences of bur breakage in orthognathic surgery: A retrospective study with discussion of 2 interesting clinical situations. *J Oral Maxillofac Surg* 69:2442–2447, 2011.
211. Marais J, Brookes GB: Secretomotor rhinopathy after Le Fort I maxillary osteotomy. Case report. *Int J Oral Maxillofac Surg* 22:17, 1993.
212. Martis CS: Complications after mandibular sagittal split osteotomy. *J Oral Maxillofac Surg* 42:101–107, 1984.
213. Martis CS, Karabouta I: Infection after orthognathic surgery with and without preventive antibiotics. *Int J Oral Surg* 13:490, 1984.
214. Marx RE: Pamidronate (Aredia) and zoledronate (Zometa) induced avascular necrosis of the jaws: A growing epidemic. *J Oral Maxillofac Surg* 61:1115–1117, 2003.
215. Marx RE, Cillo JE, Jr, Ulloa JJ: Oral bisphosphonate-induced osteonecrosis: Risk factors, prediction of risk using serum CTX testing, prevention, and treatment. *J Oral Maxillofac Surg* 65:2397–2410, 2007.
216. Maurer P, Otto C, Eckert AW, et al: Actinomycosis as a rare complication of orthognathic surgery. *Int J Adult Orthodon Orthognath Surg* 17:230, 2002.
217. McKenzie WS, Rosenberg M: Iatrogenic subcutaneous emphysema of dental and surgical origin: A literature review. *J Oral Maxillofac Surg* 67:1265, 2009.
218. Mehra P, Castro V, Freitas RZ, Wolford LM: Complications of the mandibular sagittal split ramus osteotomy associated with the presence or absence of third molars. *J Oral Maxillofac Surg* 59:854–858, 2001.
219. Meyer MW, Cavanaugh GD: Blood flow changes after orthognathic surgery: Maxillary and mandibular subapical osteotomy. *J Oral Surg* 34:495, 1976.
220. Migliorati CA, Schubert MM, Peterson DE, Seneda LM: Bisphosphonate-associated osteonecrosis of mandibular and maxillary bone: An emerging oral complication of supportive cancer therapy. *Cancer* 104:83–93, 2005.
221. Milles M, Desjardins J: Reduction of postoperative facial swelling by low-dose methylprednisolone: An experimental study. *J Oral Maxillofac Surg* 51:987, 1993.
222. Mirzai R, Chang C, Greenspan A, Gershwin ME: The pathogenesis of osteonecrosis and the relationships to corticosteroids. *J Asthma* 36:77–95, 1999.
223. Monaghan A, Hindle I: Malignant hyperpyrexia in oral surgery: Case report and literature review. *Br J Oral Maxillofac Surg* 32:190, 1994.
224. Morris DE, Lo LJ, Margulis A: Pitfalls in orthognathic surgery: Avoidance and management of complications. *Clin Plast Surg* 34:17, 2007.
225. Mosbah MR, Oloyede D, Koppel DA, et al: Miniplate removal in trauma and orthognathic surgery—a retrospective study. *Int J Oral Maxillofac Surg* 32:148–151, 2003.
226. Motamedi MH: Transient temporal nerve paresis after intraoral subcondylar ramus osteotomy. *J Oral Maxillofac Surg* 55:527, 1997.
227. Mucci SJ, Dellon AL: Restoration of lower-lip sensation: Neurotization of the mental nerve with the supraclavicular nerve. *J Reconstr Microsurg* 13:151, 1997.
228. Nagler RM, Peled M, Laufer D: Prolonged dysphagia after orthognathic surgery: Report of a case and review of the literature. *J Oral Maxillofac Surg* 54:523, 1996.
229. Nakagawa K, Ueki K, Matsumoto N, et al: The assessment of trigeminal sensory nerve paraesthesia after bilateral sagittal split

osteotomy: Modified somatosensory evoked potentials recording method. *J Oral Maxillofac Surg* 25:97, 1997.
230. Nakagawa K, Ueki K, Takatsuka S, et al: Somatosensory-evoked potential to evaluate the trigeminal nerve after sagittal split osteotomy. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 91:146, 2001.
231. Nakagawa K, Ueki K, Takatsuka S, et al: Trigeminal nerve hypesthesia after sagittal split osteotomy in setback cases: Correlation of postoperative computed tomography and long-term trigeminal somatosensory evoked potentials. *J Oral Maxillofac Surg* 61:898, 2003.
232. Nanda R, Sugawara J: Mandibular adaptations following total maxillary osteotomy in adolescent monkeys. *Am J Orthod* 83:485, 1983.
233. Nanni V, Sachs S: Mediastinal emphysema following Le Fort I osteotomy: Report of a case. *Oral Surg Oral Med Oral Pathol* 62:508, 1986.
234. Naples RJ, Van Sickels JE, Jones DL: Long-term neurosensory deficits associated with bilateral sagittal split osteotomy versus inverted "L" osteotomy. *Oral Surg Oral Med Oral Pathol* 77:318, 1994.
235. Nelson RL, Path MG, Ogle RG, et al: Quantitation of blood flow after Le Fort I osteotomy. *J Oral Surg* 35:10, 1977.
236. Newhouse RF, Schow SR, Kraut RA, et al: Life-threatening hemorrhage from a Le Fort I osteotomy. *J Oral Maxillofac Surg* 40:117, 1982.
237. Newlands C, Dixon A, Altman K: Ocular palsy following Le Fort I osteotomy: A case report. *Int J Oral Maxillofac Surg* 33:101, 2004.
238. Nishioka GJ, Mason M, Van Sickels JE: Neurosensory disturbance associated with the anterior mandibular horizontal osteotomy. *J Oral Maxillofac Surg* 46:107, 1988.
239. Nishioka GJ, Zysset MK, Van Sickels JE: Neurosensory disturbance with rigid fixation of the bilateral sagittal split osteotomy. *J Oral Maxillofac Surg* 45:20, 1987.
240. Nout E, Mathijssen IM, van der Meulen JJ, et al: Internal carotid dissection after Le Fort III distraction in Apert syndrome: A case report. *J Craniomaxillofac Surg* 38:529–533, 2010.
241. O'Regan B, Bharadwaj G: Prospective study of the incidence of serious posterior maxillary hemorrhage during a tuberosity osteotomy in low level Le Fort I operations. *Br J Oral Maxillofac Surg* 45:538, 2007.
242. O'Ryan F: Complications of orthognathic surgery. Part II. Maxillary and two-jaw surgery. *Sel Readings Oral Maxillofac Surg* 1:21–30, 1989.
243. O'Ryan F: Complications of orthognathic surgery. *Oral Maxillofac Surg Clin North Am* 2:602, 1990.
244. O'Ryan, F: Complications with orthognathic surgery. In Fonseca R, Turvey T, editors: Oral and maxillofacial surgery. Ch 30, Vol III, St Louis, 2009, Missouri: Saunders Elsevier Co.
245. O'Ryan F, Ebker BN: Prolonged apnea after orthognathic surgery due to atypical cholinesterase. *Int J Oral Surg* 10:338, 1981.
246. O'Ryan F, Poor DB, Hattori M: Intraoperative angioedema induced by angiotensin-converting enzyme inhibitors: Overview and case report. *J Oral Maxillofac Surg* 63:551, 2005.
247. Olson RE, Laskin DM: Expectations of patients from orthognathic surgery. *J Oral Surg* 38:283, 1980.
248. Ortega C, Montemurro F, Faggiuolo R, et al: Osteonecrosis of the jaw in prostate cancer patients with bone metastases treated with zoledronate: A retrospective analysis. *Acta Oncol* 46:664–668, 2007.
249. Ozaki W, Abubaker AO, Sotereanos GC, Patterson GT: Cervicofacial actinomycosis following sagittal split ramus osteotomy: A case report. *J Oral Maxillofac Surg* 50:649, 1992.
250. Pagar DM, Kupperman AW, Stern M: Cutting of nasoendotracheal tube: An unusual complication of maxillary osteotomies. *J Oral Surg* 36:314, 1978.
251. Pames El, Becker ML: Necrosis of the anterior maxilla following osteotomy: Report of a case. *Oral Surg* 33:326, 1972.
252. Panula K, Finne K, Oikarinen K: Incidence of complications and problems related to orthognathic surgery: A review of 655 patients. *J Oral Maxillofac Surg* 59:1128–1136, 2001.
253. Pappa H, Richardson D, Niven S: False aneurysm of the facial artery as a complication of sagittal split osteotomy. *J Craniomaxillofac Surg* 36:180, 2008.
254. Patel PK, Morris DE, Gassmann A: Complications of orthognathic surgery. *J Craniofac Surg* 18:975, 2007.
255. Pereira FL, Yaedu RY, Sant'Ana AP, Sant'Ana E: Maxillary aseptic necrosis after Le Fort I osteotomy: A case report and literature review. *J Oral Maxillofac Surg* 68:1402–1407, 2010.
256. Pereira-Filho VA, Gabrielli MF, Gabrielli MA, et al: Incidence of maxillary sinusitis following Le Fort I osteotomy: Clinical, radiographic, and endoscopic study. *J Oral Maxillofac Surg* 69:346–351, 2011.
257. Peterson LJ, Booth DF: Efficacy of antibiotics prophylaxis in intraoral orthognathic surgery. *J Oral Surg* 34:1088, 1976.
258. Phillips C, Essick G, Blakey G, III, et al: Relationship between patients' perceptions of postsurgical sequelae and altered sensations after bilateral sagittal split osteotomy. *J Oral Maxillofac Surg* 65:597, 2007.
259. Piecuch JF, West RA: Spontaneous pneumomediastinum associated with orthognathic surgery: A case report. *Oral Surg Oral Med Oral Pathol* 48:506, 1979.
260. Pogrel MA, Jergensen R et al: Long-term outcome of trigeminal nerve injuries related to dental treatment. *J Oral Maxillofac Surg* 69:2284–2288, 2011.
261. Pogrel MA, Le H: Etiology of lingual nerve injuries in the third molar region: A cadaver and histologic study. *J Oral Maxillofac Surg* 64:1790–1794, 2006.
261A. Poort LJ, van Neck JW, van der Wal KG: Sensory testing of inferior alveolar nerve injuries: A review of methods used in prospective studies. *J Oral Maxillofac Surg* 67:292–300, 2009.
262. Posnick JC: Orthognathic Surgery: General considerations; complications; and patient educational material. In Posnick JC, editor: Craniofacial and maxillofacial surgery in children and young adults, 42, Philadelphia, 2000, WB Saunders Co, pp 1103–1112.
263. Posnick JC, Al-Qattan MM, Pron G: Facial sensibility in adolescents with and without clefts 1 year after undergoing Le Fort I osteotomy. *Plast Reconstr Surg* 94:431, 1994.
264. Posnick JC, Al-Qattan MM, Pron GE, Grossman JA: Facial sensibility in adolescents born with cleft lip after undergoing repair in infancy. *Plast Reconstr Surg* 93:682, 1994.
265. Posnick JC, Al-Qattan MM, Stepner NM: Alteration in facial sensibility in adolescents following sagittal split and chin osteotomies of the mandible. *Plast Reconstr Surg* 97(5):920–927, 1996.
266. Posnick JC, Grossman JAI: Facial sensibility testing: A clinical update. *Plast Reconstr Surg* 106(4):892–894, 2000.
267. Posnick JC, Zimbler AG, Grossman JA: I. Normal cutaneous sensibility of the face. *Plast Reconstr Surg* 86:429, 1990.
268. Precious DS, Lung KE, Pynn BR, Goodday RH: Presence of impacted teeth as a determining factor of unfavourable splits in 1256 sagittal-split osteotomies. *Oral Surg* 85:362–365, 1998.
269. Precious DS, Powell JE, Tuzuner A, et al: False aneurysms after sagittal split ramus osteotomies. *J Oral Maxillofac Surg* 70: e58–e65, 2012.
270. Procopio O, Fusetti S, Liessi G, Ferronato G: False aneurysm of the sphenopalatine artery after a Le Fort I osteotomy: Report of 2 cases. *J Oral Maxillofac Surg* 61:520–524, 2003.
271. Quejada JG, Kawamura H, Finn RA, et al: Wound healing associated with segmental total maxillary osteotomy. *J Oral Maxillofac Surg* 44:366, 1986.
272. Rai KK, Shivakumar HR, Sonar MD: Transient facial nerve palsy following bilateral sagittal split ramus osteotomy for setback of the mandible: A review of incidence and management. *J Oral Maxillofac Surg* 66:373, 2008.
273. Rasmussen OC: Painful traumatic neuromas in the oral cavity. *Oral Surg Oral Med Oral Pathol* 49:191, 1980.
274. Reiner S, Willoughby JH: Transient abducens nerve palsy following a Le Fort I maxillary osteotomy: Report of a case. *J Oral Maxillofac Surg* 46:699, 1988.

275. Renton T, Hankins M, Sproate C, et al: A randomised controlled clinical trial to compare the incidence of injury to the inferior alveolar nerve as a result of coronectomy and removal of mandibular third molars. *Br J Oral Maxillofac Surg* 43:7, 2005.
276. Reyneke JP, Tsakiris P, Becker P: Age as a factor in the complication rate after removal of unerupted/impacted third molars at the time of mandibular sagittal split osteotomy. *J Oral Maxillofac Surg* 60:654–659, 2002.
277. Robinson PP: Observations on the recovery of sensation following inferior alveolar nerve injuries. *Br J Oral Maxillofac Surg* 26:177, 1988.
278. Robinson PP, Hendy CW: Pterygoid plate fractures caused by the Le Fort I osteotomy. *Br J Oral Maxillofac Surg* 24:198, 1986.
279. Robinson RC, Williams CW: Documentation method for inferior alveolar and lingual nerve paresthesias. *Oral Surg Oral Med Oral Pathol* 62:128, 1986.
280. Rogers SN, Patel M, Beirne JC, Nixon TE: Traumatic aneurysm of the maxillary artery: The role of interventional radiology. A report of two cases. *Int J Oral Maxillofac Surg* 24:336, 1995.
281. Rosenberg A, Gratz KW, Sailer HF: Should titanium miniplates be removed after bone healing is complete? *Int J Oral Maxillofac Surg* 22:185, 1993.
282. Rosendorf LL, Octavia J, Estes JP: Effect of methods of postdischarge wound infection surveillance on reported infection rates. *Am J Infect Control* 11:226, 1983.
283. Ruggiero SL, Mehrotra B, Rosenberg TJ, Engroff SL: Osteonecrosis of the jaws associated with the use of bisphosphonates: A review of 63 cases. *J Oral Maxillofac Surg* 62:527–534, 2004.
284. Ruggles JE, Hann JR: Antibiotic prophylaxis in intraoral orthognathic surgery. *J Oral Maxillofac Surg* 42:797–801, 1984.
285. Russell RG, Rogers MJ: Bisphosphonates: From the laboratory to the clinic and back again. *Bone* 25:97–106, 1999.
286. Sakai Y, Kobayashi S, Sekiguchi J, Ohmori K: New method of endoscopic pterygomaxillary disjunction for a Le Fort type I osteotomy. *J Craniofac Surg* 7:111, 1996.
287. Sammartino G, Califano L, Grassi R, et al: Transient facial nerve paralysis after mandibular sagittal osteotomy. *J Craniofac Surg* 16:1110, 2005.
288. Sanni K, Campbell R, Rosner M, Goyne WB: Internal carotid arterial occlusion following mandibular osteotomy. *J Oral Maxillofac Surg* 42:394, 1984.
289. Schow SR, Triplett RG, Solomon JM: Lingual nerve injury associated with overpenetration of bicortical screws used for fixation of a bilateral mandibular sagittal split ramus osteotomy. *J Oral Maxillofac Surg* 54:1451, 1996.
290. Schultze-Mosgau S, Krems H, Ott R, Neukam FW: A prospective electromyographic and computer-aided thermal sensitivity assessment of nerve lesions after sagittal split osteotomy and le Fort I osteotomy. *J Oral Maxillofac Surg* 59:128–139, 2001.
291. Schultze-Mosgau S, Reich RH: Assessment of inferior alveolar and lingual nerve disturbances after dentoalveolar surgery, and of recovery of sensitivity. *Int J Oral Maxillofac Surg* 22:214, 1993.
292. Schwartz HC: Osteonecrosis and bisphosphonates: Correlation versus causation. *J Oral Maxillofac Surg* 62:763–764, 2004.
293. Schwartz HC, Wilson MC: Cervicofacial actinomycosis following orthognathic surgery: Report of 2 cases. *J Oral Maxillofac Surg* 59:447, 2001.
294. Schwartz S, Joseph C, Iera D, Vu DD: Bisphosphonates, osteonecrosis, osteogenesis imperfecta and dental extractions: A case series. *J Can Dent Assoc* 74:537–542, 2008.
295. Seo K, Tanaka Y, Terumitsu M, et al: Efficacy of steroid treatment for sensory impairment after orthognathic surgery. *J Oral Maxillofac Surg* 62:1193, 2004.
296. Seo K, Tanaka Y, Terumitsu M, et al: Characterization of different paresthesias following orthognathic surgery of the mandible. *J Oral Maxillofac Surg* 63:298, 2005.
297. Shand JM, Heggie AA: Use of a resorbable fixation system in orthognathic surgery. *Br J Oral Maxillofac Surg* 38:335, 2000.
298. Sher MR: A survey of complications in segmental orthognathic surgical procedures. *Oral Surg Oral Med Oral Pathol* 58:537, 1984.
299. Sheridan SM: Traumatic neuroma following sagittal split mandibular osteotomy. *Br J Oral Surg* 21:198, 1983.
300. Shoshani Y, Samet N, Ardekian L, et al: Nasolacrimal duct injury after Le Fort I osteotomy. *J Oral Maxillofac Surg* 52:406, 1994.
301. Sickels JE: Neurosensory recovery following the mandibular bilateral sagittal split osteotomy (discussion). *J Oral Maxillofac Surg* 53:1306, 1995.
302. Siebert JW, Angrigiani C, McCarthy JG, et al: Blood supply of the Le Fort I maxillary segment: An anatomic study. *Plast Reconstr Surg* 100:843, 1997.
303. Silva AC, O'Ryan F, Beckley ML, et al: Pseudoaneurysm of a branch of the maxillary artery following mandibular sagittal split ramus osteotomy: Case report and review of the literature. *J Oral Maxillofac Surg* 65:1807, 2007.
304. Sinclair P, Kilpelanien P, Phillips C, et al: The accuracy of video imaging in orthognathic surgery. *Am J Dentofac Orthop Orthod* 107:177–185, 1995.
305. Singh J, Doddrige M, Broughton A, Goss A: Reconstruction of post-orthognathic aseptic necrosis of the maxilla. *Br J Oral Maxillofac Surg* 46:408–410, 2008.
306. Singhal A, Golomb M, Mochida G, et al: Another case of internal carotid artery dissection after mandibular osteotomy. *J Oral Maxillofac Surg* 56:115, 1998.
307. Sirikumara M, Sugar AW: Adie's pupil following Le Fort I maxillary osteotomy. A complication of coincidence? *Br J Oral Maxillofac Surg* 28:306, 1990.
308. Smith IM, Anderson PJ, Wilks MJ, et al: Traumatic arteriovenous malformation following maxillary Le Fort I osteotomy. *Cleft Palate Craniofac J* 45:329, 2008.
309. Smith KS, Heggie AAC: Vomero-sphenoidal disarticulation during the Le Fort I maxillary osteotomy; report of a case. *J Oral Maxillofac Surg* 53:465, 1995.
310. Solomons NB, Blumgart R: Severe late-onset epistaxis following Le Fort I osteotomy: Angiographic localization and embolization. *J Laryngol Otol* 102:260, 1988.
311. Song HC, Throckmorton GS, Ellis E, III, et al: Functional and morphologic alterations after anterior or inferior repositioning of the maxilla. *J Oral Maxillofac Surg* 55:41, 1997.
312. Spaey YJ, Bettens RM, Mommaerts MY, et al: A prospective study on infectious complications in orthognathic surgery. *J Craniomaxillofac Surg* 33:24, 2005.
313. Stacy GC: Recovery of oral opening following sagittal ramus ostectomy for mandibular prognathism. *J Oral Maxillofac Surg* 45:487, 1987.
314. Stajcic Z, Roncevic R: Facial nerve palsy following combined maxillary and mandibular osteotomy. *J Craniomaxillofac Surg* 18:192, 1990.
315. Steel BJ, Cope MR: Unusual and rare complications of orthognathic surgery: A literature review. *J Oral Maxillofac Surg* 70:1678–1691, 2012.
316. Stern NS, Shensa DR, Trop RC: Cavernous sinus thrombosis: A complication of maxillary surgery. *J Oral Surg* 39:436, 1981.
317. Stewart TD, Sexton J: Depression: A possible complication of orthognathic surgery. *J Oral Maxillofac Surg* 45:847, 1987.
318. St-Hilaire H, Montazem AH, Diamond J: Pneumomediastinum after orthognathic surgery. *J Oral Maxillofac Surg* 62:892, 2004.
319. Stone HH, Haney BB, Kolb LD, et al: Prophylactic and preventive antibiotic therapy: Timing, duration and economics. *Ann Surg* 189:691–699, 1979.
320. Storum K, Bell WH: Hypomobility after maxillary and mandibular osteotomies. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 57:7, 1984.
321. Stringer DE, Dolwick MF, Steed DL: Subcutaneous emphysema after Le Fort I osteotomy: Report of two cases. *J Oral Surg* 37:115, 1979.
322. Sun JR, Kim SG: Facial nerve injuries following bilateral sagittal osteotomies. *Hosp Dent (Tokyo)* 19:13, 2007.
323. Sunderland S: A classification of peripheral nerve injuries producing loss of function. *Brain* 74:491, 1951.

324. Susarla SM, Kaban LB, Donoff RB, Dodson TB: Does early repair of lingual nerve injuries improve functional sensory recovery? *J Oral Maxillofac Surg* 65:1070–1076, 2007.
325. Suuronen R, Ashammakhi N, Peltoniemi H, et al: Developments in craniomaxillofacial surgery: Use of self-reinforced bioabsorbable osteofixation devices. *Plast Reconstr Surg* 108:167, 2001.
326. Suzuki H, Saito E, Hashimoto K: Dissecting aneurysm of the internal carotid artery after a mandibular osteotomy. *J Oral Maxillofac Surg* 55:747, 1997.
327. Svartz K, Ahlborg G, Finne K, Nethander G: Nerve disturbances after sagittal split osteotomy. *Int J Oral Surg* 12:279, 1983.
328. Taher AAY: Facial palsy: A complication of sagittal ramus osteotomy (Obwegeser-Dal Pont technique): Report of a case. *Quintessence Int* 19:229, 1988.
329. Takasaki Y, Noma H, Masaki H, et al: A clinical analysis of the recovery from sensory disturbance after sagittal splitting ramus osteotomy using a Semmes-Weinstein pressure aesthesiometer. *Bull Tokyo Dent Coll* 39:189, 1998.
330. Takeuchi T, Furusawa K, Hirose I: Mechanism of transient mental nerve paraesthesia in sagittal split mandibular ramus osteotomy. *Br J Oral Maxillofac Surg* 32:105–108, 1994.
331. Tay AB, Go WS: Effect of exposed inferior alveolar neurovascular bundle during surgical removal of impacted lower third molars. *J Oral Maxillofac Surg* 62:592, 2004.
332. Tay AB, Poon CY, Teh LY: Immediate repair of transected inferior alveolar nerves in sagittal split osteotomies. *J Oral Maxillofac Surg* 66:2476–2481, 2008.
333. Teerijoki-Oksa T, Jääskeläinen S, Forssell H, et al: Risk factors of nerve injury during mandibular sagittal split osteotomy. *Int J Oral Maxillofac Surg* 31:33–39, 2002.
334. Teerijoki-Oksa T, Jääskeläinen S, Forssell K, et al: An evaluation of clinical and electrophysiological tests in nerve injury diagnosis after mandibular sagittal split osteotomy. *Int J Oral Maxillofac Surg* 32:15–23, 2003.
335. Teltzrow T, Kramer FJ, Schulze A, et al: Perioperative complications following sagittal split osteotomy of the mandible. *J Craniomaxillofac Surg* 33:307, 2005.
336. Theodossy T, Jackson O, Petrie A, et al: Risk factors contributing to symptomatic plate removal following sagittal split osteotomy. *Int J Oral Maxillofac Surg* 35:598, 2006.
337. Thygesen TH, Bardow A, Norholt SE, et al: Surgical risk factors and maxillary nerve function after Le Fort I osteotomy. *J Oral Maxillofac Surg* 67:528, 2009.
338 Tomasetti BJ, Broutas M, Gormley M, et al: Lack of tearing after Le Fort I osteotomy. *J Oral Surg* 34:1095, 1976.
339. Tomazzoli-Gerosa L, Marchini G, Monaco A: Amaurosis and atrophy of the optic nerve: An unusual complication of mandibular-nerve anesthesia. *Ann Ophthalmol* 20:170, 1988.
340. Trimble LD, Tideman H, Stoelinga PJ: A modification of the pterygoid separation in low-level maxillary osteotomies. *J Oral Maxillofac Surg* 41:544, 1983.
341. Tuinzing DB, Greebe RB: Complications related to the intraoral vertical ramus osteotomy. *Int J Oral Surg* 14:319, 1985.
342. Tung TC, Chen YR, Bendor-Samuel R: Surgical complications of the Le Fort I osteotomy: A retrospective review of 146 cases. *Changgeng Yi Xue Za Zhi* 18:102, 1995.
343. Turvey TA: Intraoperative complications of sagittal osteotomy of the mandibular ramus: Incidence and management *J Oral Maxillofac Surg* 43:594, 1985.
344. Turvey TA, Bell BB, Tejera TJ, Proffit WR: The use of self-reinforced biodegradable bone plates and screws in orthognathic surgery. *J Oral Maxillofac Surg* 60:59–65, 2002.
345. Turvey TA, Bell RB, Phillips C, Proffit WR: Self-reinforced biodegradable screw fixation compared with titanium screw fixation in mandibular advancement. *J Oral Maxillofac Surg* 64(1):40–46, 2006.
346. Van de Perre JP, Stoelinga PJ, Blijdorp PA, et al: Perioperative morbidity in maxillofacial orthopedic surgery: A retrospective study. *J Craniomaxillofac Surg* 24:263, 1996.
347. Van Merkesteyn JPR, Groot RH, van Leeuwaarden R, et al: Intra-operative complications in sagittal and vertical ramus osteotomies. *Int J Oral Maxillofac Surg* 16:665, 1987.
348. Van Sickels JE: Concomitant removal of mandibular third molars during sagittal split osteotomies minimizes neurosensory dysfunction: Comment. *J Oral Maxillofac Surg* 70:2164, 2012.
349. Van Sickels JE, Hatch JP, Dolce C, et al: Effect of age, amount of advancement and genioplasty on neurosensory disturbance after a bilateral sagittal split osteotomy. *J Oral Maxillofac Surg* 60:1012, 2002.
350. Van Sickels JE, Tucker MR: Management of delayed union and nonunion of maxillary osteotomies. *J Oral Maxillofac Surg* 48:1039, 1990.
351. Vaughan TC, Cronin A: Neurosurgery required following a sagittal split osteotomy. *Br J Oral Maxillofac* 47:e23, 2009.
352. Veterans Administration Ad Hoc Interdisciplinary Advisory Committee on Antimicrobial Drug Usage: Prophylaxis in surgery. *J Am Med Assoc* 237:1003–1008, 1977.
353. Waack D: Perioperative complications associated with Le Fort I osteotomies. *J Oral Maxillofac Surg* 52 (suppl 2):92, 1994.
354. Walter JM, Jr, Gregg JM: Analysis of postsurgical neurologic alteration in the trigeminal nerve. *J Oral Surg* 37:410, 1979.
355. Wang JH, Waite DE: Evaluation of the surgical procedure of sagittal split osteotomy of the mandibular ramus. *Oral Surg Oral Med Oral Pathol* 38:167, 1974.
355A. Ward Booth P: Discussion: Risk factors contributing to symptomatic plate removal in orthognathic surgery patients. *J Oral Maxillofac Surg* 57:682, 1999.
356. Watts PG: Unilateral abducent nerve palsy: A rare complication following a Le Fort I maxillary osteotomy. *Br J Oral Maxillofac Surg* 22:212, 1984.
357. Weber CR, Griffin JM: Evaluation of dexamethasone for reducing postoperative edema and inflammatory response after orthognathic surgery. *J Oral Maxillofac Surg* 52:35, 1994.
358. Westermark A, Bystedt H, von Konow L: Inferior alveolar nerve function after mandibular osteotomies. *Br J Oral Maxillofac Surg* 36:425–428, 1998.
359. Westermark A, Bystedt H, von Konow L: Inferior alveolar nerve function after sagittal split osteotomy of the mandible: Correlation with degree of intraoperative nerve encounter and other variables in 496 operations. *Br J Oral Maxillofac Surg* 36:429–433, 1998.
360. Westermark A, Bystedt H, von Konow L: Patient's evaluation of the final result of sagittal split osteotomy: Is it influenced by impaired sensitivity of the lower lip and chin? *Int J Adult Orthodon Orthognath Surg* 14:135, 1999.
361. Westermark A, Englesson L, Bongenhielm U: Neurosensory function after sagittal split osteotomy of the mandible: A comparison between subjective evaluation and objective assessment. *Int J Adult Orthodon Orthognath Surg* 14:268–275, 1999.
362. Westwood RM, Tilson HB: Complications associated with maxillary osteotomies. *J Oral Surg* 33:104, 1975.
363. Willmar K: On Le Fort I osteotomy. *Scand J Plast Reconstr Surg Suppl* 12, 1974.
364. Willmar K, Hogeman KE, Thiseus S: Sagittal split osteotomy in our experience: A follow-up study of 100 operated patients. *Scand J Plast Reconstr Surg* 13:445, 1979.
365. Wilson ME, Ellis FR: Predicting malignant hyperpyrexia. *Br J Anaesth* 51:66, 1979.
366. Wilson MW, Wheatley MJ, Shults WT: Secondary fractures of Le Fort I osteotomy. *Ophthal Plast Reconstr Surg* 16:258, 2000.
367. Woo SB, Hellstein JW, Kalmar JR: Systematic review: Bisphosphonates and osteonecrosis of the jaws. *Ann Intern Med* 144:753–761, 2006.
368. Ylikontiola L, Kinnunen J, Laukkanen P, et al: Prediction of recovery from neurosensory deficit after bilateral sagittal split osteotomy. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 90:275, 2000.
369. Ylikontiola L, Kinnunen J, Oikarinen K: Comparison of different tests assessing neurosensory disturbances after bilateral sagittal split osteotomy. *Int J Oral Maxillofac Surg* 27:417, 1998.
370. Ylikonitiola L, Kinnunen J, Oikarinen K: Factors affecting neurosensory disturbance after mandibular bilateral sagittal split osteotomy. *J Oral Maxillofac Surg* 58:1234, 2000.

371. Yoshida T, Nagamine T, Kobayashi T, et al: Impairment of the inferior alveolar nerve after sagittal split osteotomy. *J Craniomaxillofac Surg* 17:271, 1989.
372. You ZH: A study of maxillary and mandibular vasculature in relation to orthognathic surgery [article in Chinese]. *Zhonghua Kou Qiang Yi Xue Za Zhi* 26:263, 1991.
373. You ZH, Zhane ZK, Zhane XE: Le Fort I osteotomy with descending palatal artery intact and ligated: A study of blood flow and quantitative histology. *Contemp Stomatol* 5:71, 1991.
374. Young RA, Epker BN: The anterior maxillary osteotomy: A retrospective evaluation of sinus health, patient acceptance, and relapse. *J Oral Surg* 30:69, 1972.
375. Yrastorza J: Indications for antibiotics in orthognathic surgery. *J Oral Surg* 34:514, 1976.
376. Yun KI, Lee JA, Park JU: Intubation granuloma: Report of a case. *J Oral Maxillofac Surg* 66:1263, 2008.
377. Zarrinkelk HM, Throckmorton GS, Ellis E, III, et al: A longitudinal study of changes in masticatory performance of patients undergoing orthognathic surgery. *J Oral Maxillofac Surg* 53:777, 1995.
378. Zaytoun HS, Jr, Phillips C, Terry BC: Long-term neurosensory deficits following transoral vertical ramus and sagittal split osteotomies for mandibular prognathism. *J Oral Maxillofac Surg* 44:193, 1986.
379. Zijderveld SA, Smeele LE, Kostense PJ, Tuinzing DB: Preoperative antibiotic prophylaxis: A randomized, double-blind and placebo-controlled clinical study. *J Oral Maxillofac Surg* 57:1403–1406, 1999.
380. Zuniga JR, Essick GK: A contemporary approach to the clinical evaluation of trigeminal nerve injuries. *Oral Maxillofac Surg Clin North Am* 4:353, 1992.
381. Zuniga JR, Meyer RA, Gregg JM, et al: The accuracy of clinical neurosensory testing for nerve injury diagnosis. *J Oral Maxillofac Surg* 56:2, 1998.

17

Malocclusion after Orthodontics and Orthognathic Surgery: Prevention and Treatment Pitfalls

JEFFREY C. POSNICK, DMD, MD

- Sagittal Split Ramus Osteotomies of the Mandible: Skeletal Stability and Relapse
- Le Fort I Osteotomy: Skeletal Stability and Relapse
- Orthodontic Treatment: Dental Stability and Relapse
- Management of Transverse Maxillary Deficiency: Techniques and Relapse Patterns
- Conclusions

There are inherent limitations to the orthodontic movement of teeth and the surgical repositioning of the jaws and jaw segments; these limitations are imposed by the alveolar bone and the periodontium, the overlying soft-tissue envelope, the masticatory and facial musculature that surrounds the maxillomandibular skeleton, and the temporomandibular joints. If the biologic boundaries are not respected during the movement of teeth, and the surgical repositioning of the jaws, malocclusion is one potential adverse consequence that may follow. The vector changes of the jaws, the jaw segments, and the teeth that are more prone to relapse are known, and so are many of the reasons why. The intent of this chapter is to review the most prevalent causes of malocclusion after osteotomies used to reposition the jaws and orthodontic mechanics used for the movement of teeth in the correction of dentofacial deformities.

Sagittal Split Ramus Osteotomies of the Mandible: Skeletal Stability and Relapse

Immediate (Early) Malocclusion after Sagittal Split Ramus Osteotomies

An immediate (early) malocclusion that is recognized either intraoperatively or soon after surgery (i.e., during the first several days or weeks afterward) will occur after sagittal split ramus osteotomies of the mandible unless each of the following intraoperative conditions is met* (Figs. 17-1 through 17-4):

1. The proximal segment is successfully split from the distal mandible (i.e., no "bad" split) on each side (right and left).
2. The distal mandible is placed in the preferred occlusion and tightly held in intermaxillary fixation (IMF). Unless the natural occlusion between the upper and lower teeth is precise (i.e., without centric relation–centric occlusion slides), the IMF should be maintained with the use of an accurate prefabricated acrylic splint.
3. Each proximal segment is positioned with the condyle seated in the terminal hinge position. Each proximal segment is then secured to the distal segment with the use of either passively placed bicortical screws or monocortical screws and plates. This is accomplished without torquing or overseating the proximal segment. The following conditions should be present:

Text continued on p. 555

*References 1, 5, 8, 39-41, 43, 44, 52, 53, 58, 74, 75, 84, 86, 88, 90, 91, 98, 102, 106-108, 111-113, 116, 126-128, 131, 132, 136, 137, 140, 150, 153, 155, 156, 160-163, 165, 166, 173, 180, 181, 183, 184, 188, 189, 194, 198, 209, 230, 255, 262, 263, 265, 271, 279, 292-295, 299, 301, 304, 305, 307, 309-311, 313, 318-321, 326

• **Figure 17-1** A woman in her late 20s was referred by her orthodontist for surgical evaluation. She suffered with masticatory muscle discomfort while chewing and had a jaw deformity, malocclusion, and a lifelong history of obstructed nasal breathing. A long face Class II growth pattern was evident from her childhood onward. She had undergone attempted orthodontic growth modification and then a camouflage approach, without success. She then underwent orthodontics (including upper bicuspid extractions) and surgery at another institution; this included a Le Fort I osteotomy and bilateral ramus osteotomies. Analysis confirmed both inadequate positioning of the jaws at the time of operation and then dental relapse of the maxillary arch form over time. When she presented to this surgeon, her clinical problems included the following:

- TMD with masticatory muscle discomfort
- Maxillary deformity (transverse constriction, vertical excess, horizontal deficiency, and clockwise rotation)
- Mandibular deformity (horizontal retrusion and clockwise rotation)
- Chin deformity (excess vertical length and horizontal retrusion)
- Obstructed nasal airway (septal deviation, high nasal floor, constricted aperture, and inferior turbinate enlargement)

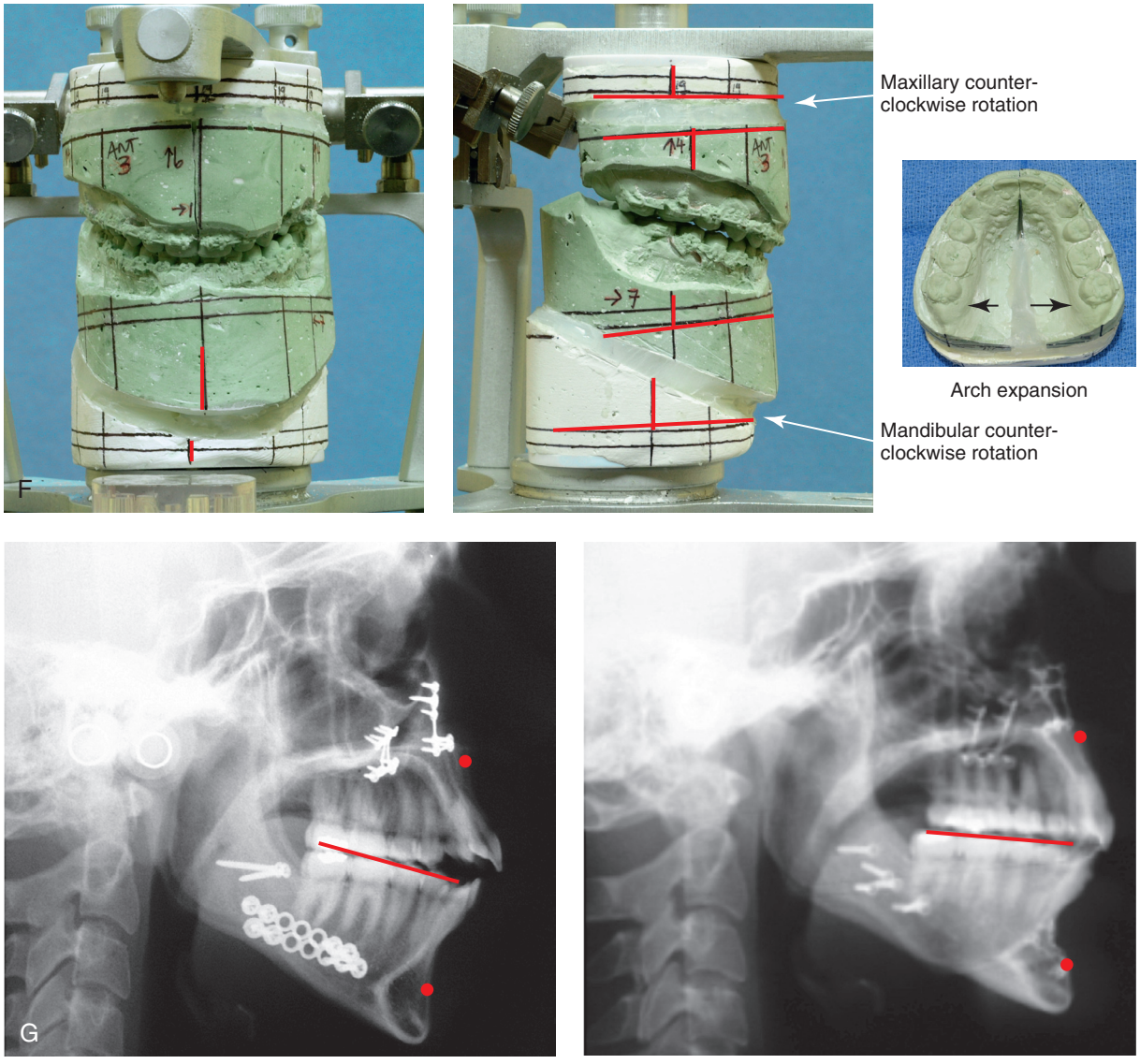
The patient was treated for temporomandibular disorder and then with a comprehensive orthodontic and surgical approach. Splint therapy was useful to relieve masticatory muscle comfort, and orthodontic dental decompensation was then carried out over approximately 12 months. Her surgery included maxillary Le Fort I osteotomy in segments (horizontal advancement, vertical intrusion, counterclockwise rotation, and transverse widening); bilateral sagittal split ramus osteotomies (horizontal advancement and counterclockwise rotation); osseous genioplasty (vertical reduction and horizontal advancement); and septoplasty, inferior turbinate reduction, and recontouring of the nasal floor. After initial healing and splint removal (i.e., 5 weeks after surgery), orthodontic maintenance and detailing continued for 9 months. **A,** Frontal and occlusal views before retreatment. **B,** Profile and cephalometric views before retreatment.

Prior to retreatment

After treatment

• **Figure 17-1, cont'd** **C,** Frontal facial views before and after retreatment. **D,** Profile views before and after retreatment. **E,** Occlusal views before and 2 years after retreatment.

Continued



• **Figure 17-1, cont'd** **F,** Articulated dental casts that indicate analytic model planning. **G,** Lateral cephalometric radiographs before and after retreatment.

• **Figure 17-2** A man with a developmental jaw deformity that was characterized as an asymmetric mandibular excess growth pattern (i.e., hemimandibular elongation) presented for treatment. His orthodontist originally attempted growth modification followed by orthodontic camouflage that included four bicuspid extractions, without success. When he was 19 years old, the patient underwent further orthodontics and a surgical approach at another institution. His jaw surgery at the time included maxillary Le Fort I osteotomy and bilateral vertical oblique ramus osteotomies. An initial satisfactory surgical result was not achieved, and further orthodontic camouflage was attempted. During ongoing orthodontic treatment, alveolar bone loss and gingival recession occurred along the buccal aspect of the posterior maxillary dentition and the labial aspect of the anterior mandibular dentition. At the time of referral to this surgeon, the patient was 35 years old; thus, it had been 16 years since his suboptimal treatment. Evaluations were arranged with a periodontist; an orthodontist; a speech therapist; and an ear, nose, and throat specialist. After periodontal treatment, orthodontic decompensation was carried out in preparation for reconstruction. The patient's surgery included maxillary Le Fort I osteotomy in segments (cant correction, transverse expansion, and minimal horizontal advancement); sagittal split ramus osteotomies (asymmetry correction and minimal horizontal advancement); osseous genioplasty (vertical reduction and horizontal advancement); and septoplasty and inferior turbinate reduction. **A,** Frontal and occlusal views before retreatment. **B,** Current profile view shown next to a lateral cephalometric radiograph taken before original surgery, when the patient was 19 years old.

Continued

• **Figure 17-2, cont'd C,** Frontal views in repose before and after retreatment. **D,** Frontal views with smile before and after retreatment.

• **Figure 17-2, cont'd** **E,** Oblique facial views before and after retreatment. **F,** Profile views before and after retreatment.

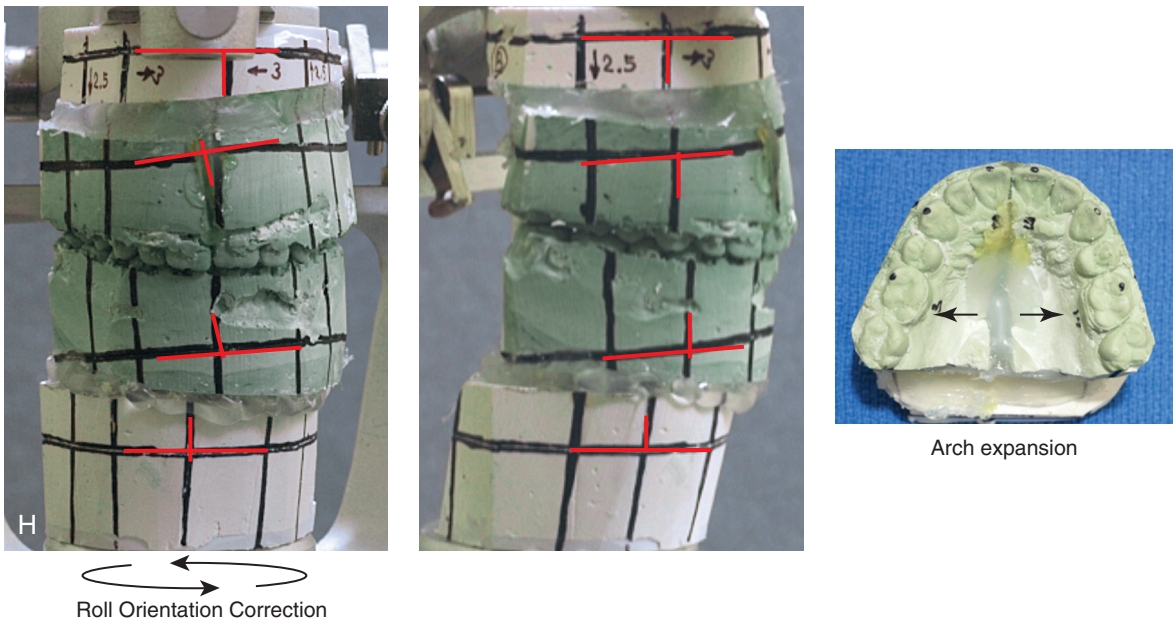
Continued

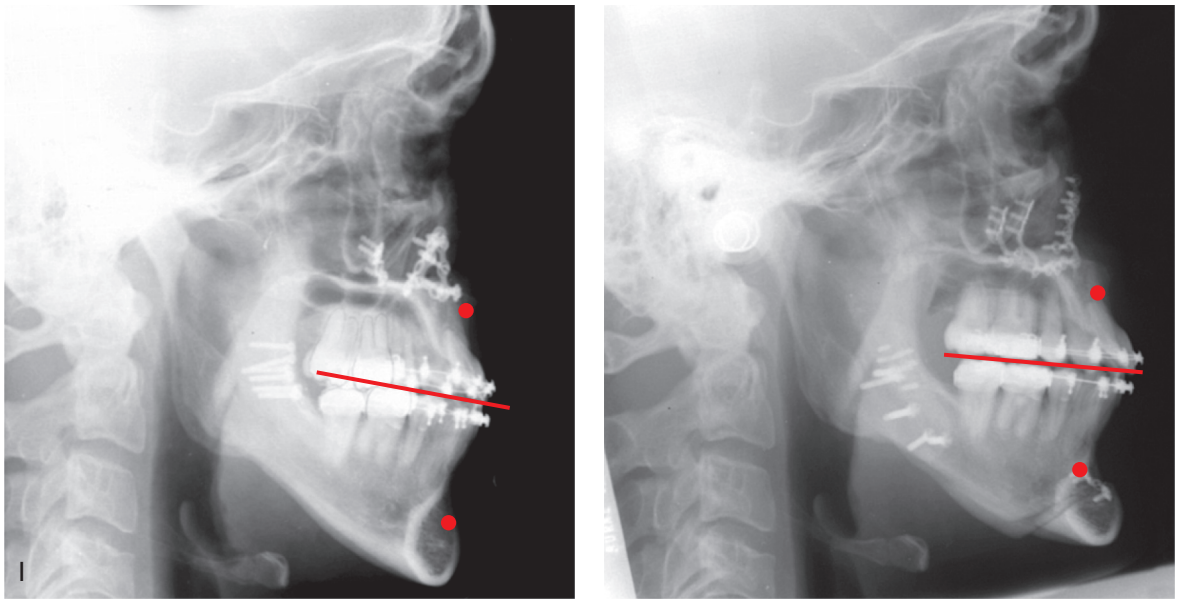
Prior to retreatment with 4 bicuspid extractions

Pre surgery

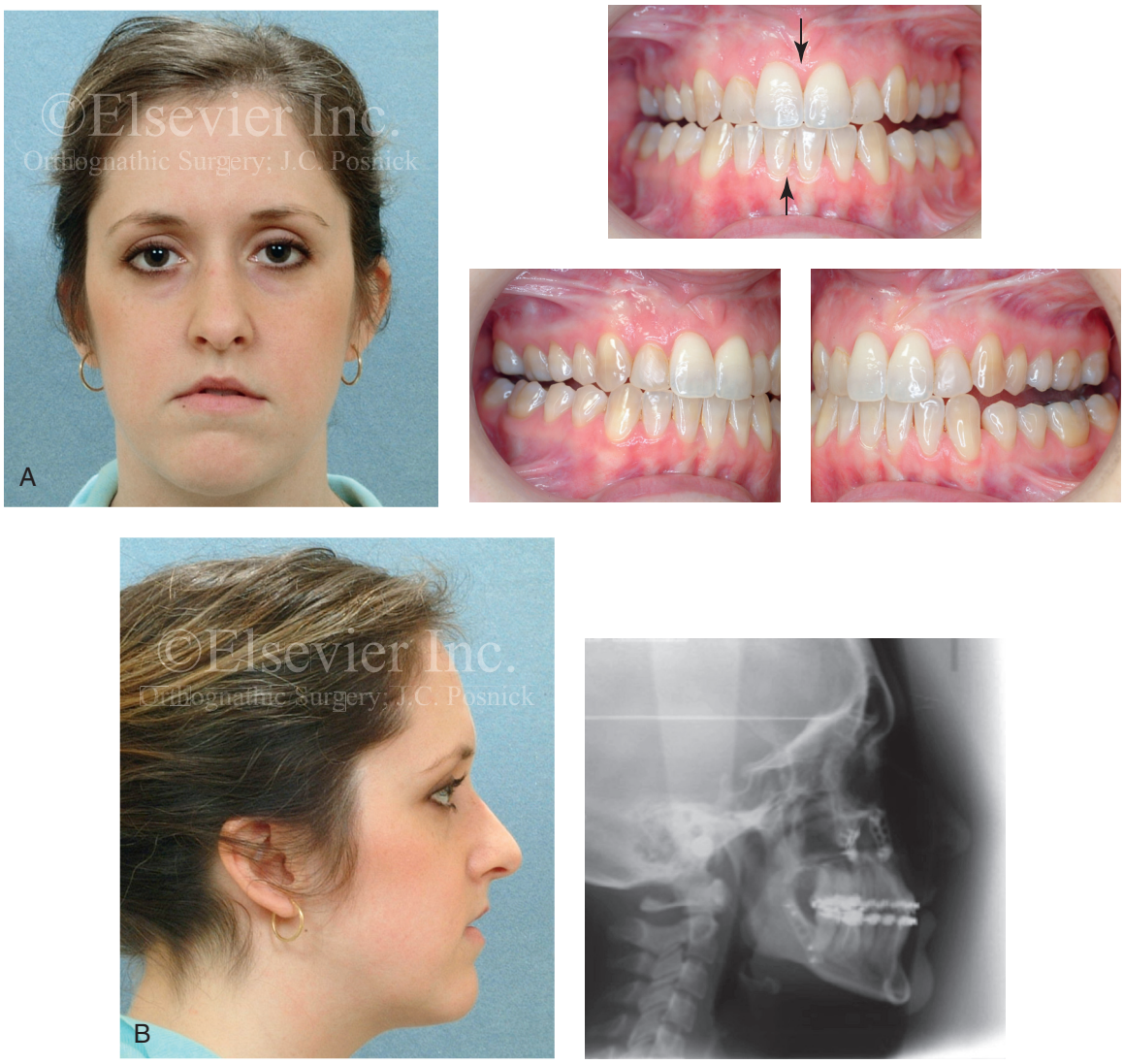
After retreatment

• **Figure 17-2, cont'd G,** Occlusal views before orthodontic retreatment, with orthodontic decompensation in progress, and after retreatment.



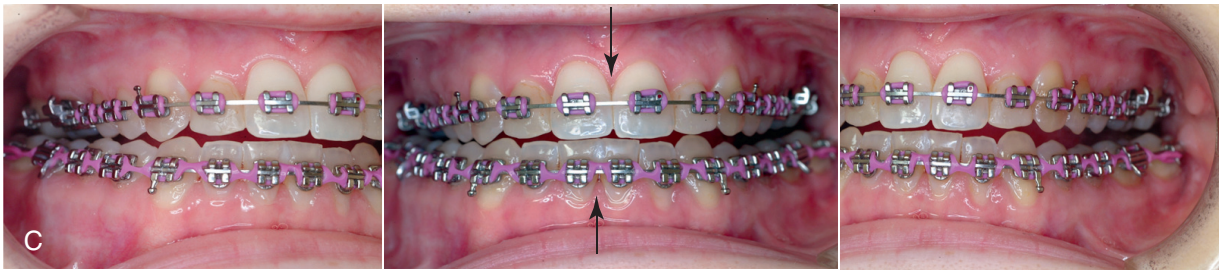


• **Figure 17-2, cont'd** **H,** Articulated dental casts after analytic model planning. **I,** Lateral cephalometric radiographs after first surgery and then after retreatment.

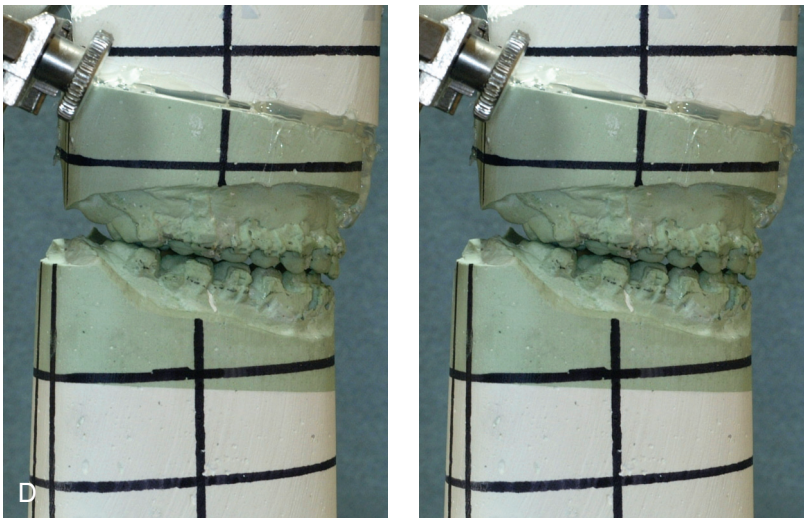


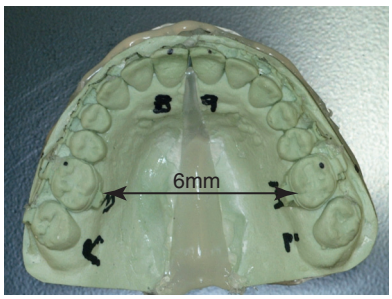
• **Figure 17-3** A 28-year-old woman was referred by her orthodontist for the evaluation of residual malocclusion and facial asymmetry. Eight years earlier, the patient had presented with an asymmetric mandibular excess growth pattern and underwent a combined orthodontic and surgical approach at another institution. The surgery included maxillary Le Fort I osteotomy (three segments) with iliac interpositional grafting in combination with bilateral sagittal split ramus osteotomies. Immediately after surgery, the occlusion that had been achieved was recognized to be unsatisfactory. The patient was taken back to the operating room 1 week later for the removal of the fixation of the right ramus of the mandible, the adjustment of the occlusion, and the reapplication of fixation. The occlusion remained unsatisfactory. Six months later, the patient was taken back to the operating room again, where she underwent a left vertical oblique ramus osteotomy without fixation followed by 6 weeks of intermaxillary fixation. Over the long term, the occlusion remained unsatisfactory, and the patient also had a minor degree of facial asymmetry as seen in the frontal view. At the time of referral to this surgeon, the patient stated that she hoped for the correction of the occlusion and an improvement in her current lip asymmetry. The decision was made to not operate on the mandible again. Options to improve the occlusion included maxillary Le Fort I osteotomy in segments versus surgically assisted rapid palatal expansion. Segmental maxillary surgery with immediate repositioning and interpositional grafting was carried out. The occlusion was maintained with a prefabricated acrylic splint for 6 weeks, which was followed by the immediate placement of a transpalatal arch. **A,** Frontal facial and occlusal views before retreatment. **B,** Profile facial view and lateral cephalometric radiograph before retreatment.

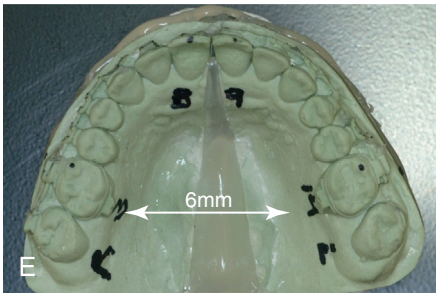
Prior to retreatment



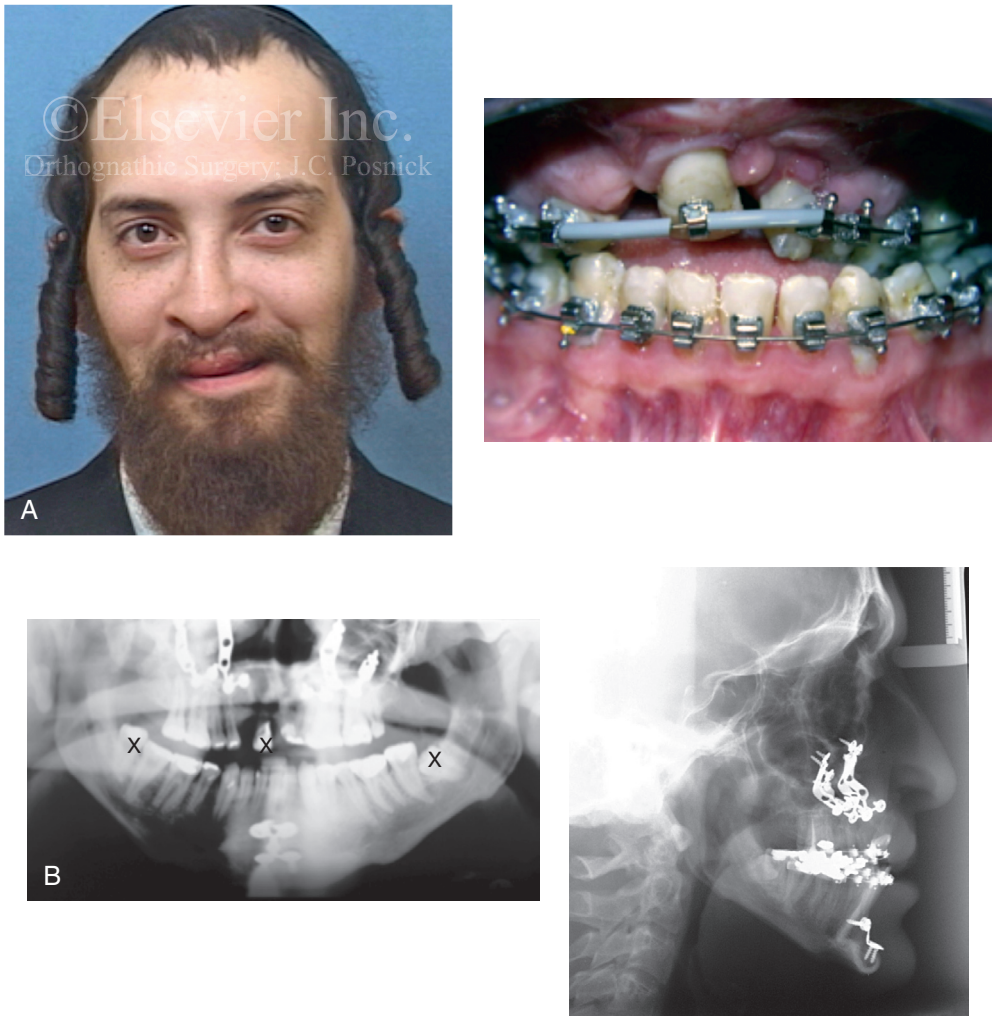
Pre surgery



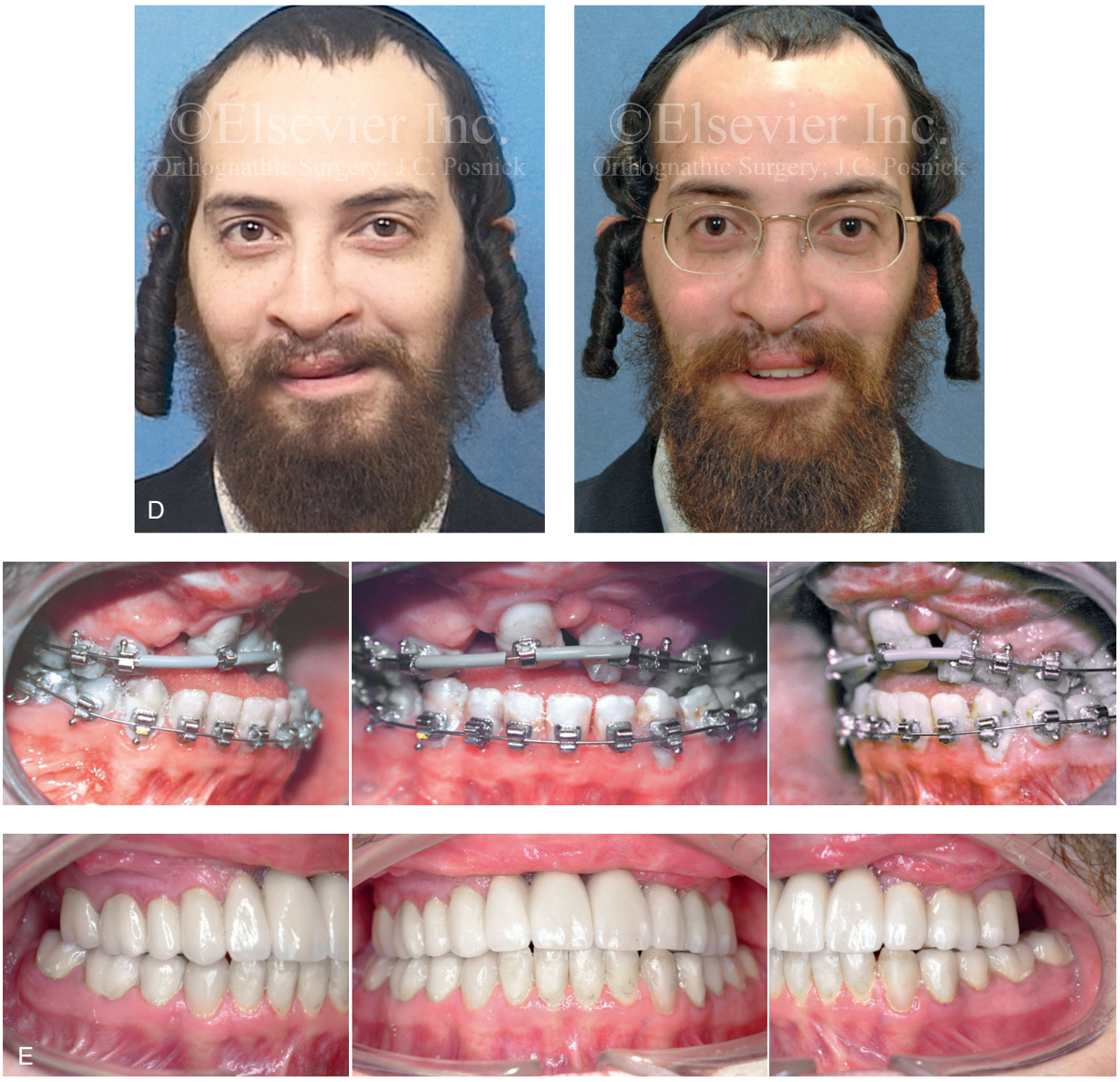




• **Figure 17-3, cont'd** **C,** Occlusal views before retreatment and preoperatively with orthodontic appliances in place. **D,** Articulated dental casts that indicate surgical planning. **E,** Demonstration of the use of the maxillary surgical model to construct a transpalatal arch wire for immediate placement at the time of splint removal (i.e., 5 weeks after surgery). The roof of the patient's mouth is shown with the transpalatal wire in place in accordance with the surgical model. The use of orthodontic mechanics for 6 months to actively maintain the arch width achieved at operation is useful to limit a tendency toward relapse. It is also important to limit any orthodontic mechanics that may position the teeth outside of the alveolar housing, because this situation will be prone to long-term dental relapse.



• **Figure 17-4** A 21-year-old man who was born with bilateral cleft lip and palate arrived for surgical evaluation. He had undergone suboptimal orthognathic surgery (Le Fort I osteotomy and osseous genioplasty) at another institution 4 years earlier. The history, physical examination, and radiographs suggested a combination of both unfavorable initial surgical healing and late dental relapse. The patient was referred to this surgeon and underwent a comprehensive surgical and dental rehabilitative approach. After periodontal evaluation and treatment, orthodontic decompensation was undertaken. The patient's surgery included maxillary Le Fort I osteotomy in three segments with differential repositioning (cant correction, closing down of cleft dental gap, closure of fistula, horizontal advancement, counterclockwise rotation, and vertical adjustment); bilateral sagittal split ramus osteotomies (asymmetry correction); and septoplasty and inferior turbinate reduction. This was followed by planned prosthetic rehabilitation of the maxillary and mandibular arches and ongoing periodontal surveillance. **A,** Frontal and occlusal views at the time of surgical evaluation (i.e., 4 years after the suboptimal orthognathic procedures at another institution). **B,** Cephalometric and Panorex radiographs at the time of surgical evaluation. **C,** Articulated dental casts that indicate analytic model planning.



• **Figure 17-4, cont'd** **D,** Frontal views with smile before and after retreatment including dental rehabilitation. **E,** Occlusal views before and after retreatment including dental rehabilitation.

- Adequate bone contact between the proximal and distal segments, without compression or torquing
- Appropriate alignment of the proximal and distal segments to limit the rotation or torquing of each proximal segment.

4. With the release of IMF, the mandible is put through the range of motion to confirm the adequate stability of the skeletal fixation across the ramus osteotomy site on each side.
5. The occlusion is then checked for any centric relation–centric occlusion discrepancies or other causes of malocclusion. If a discrepancy is seen, each proximal segment must be released of its fixation and then again repositioned until the planned, stable, and reproducible occlusion is achieved (i.e., Steps 1 through 5 are repeated, as needed).

Late Malocclusion after Sagittal Split Ramus Osteotomies

Malocclusion after the successful initial healing of sagittal split ramus osteotomies is the result of either skeletal or dental causes.* *Skeletal causes* may include condylar resorption (see Chapter 36); remodeling or malunion (i.e., relapse) at the osteotomy site; or continued mandibular growth; these conditions are all described in more detail later in this chapter. *Dental causes* of malocclusion after sagittal split ramus osteotomies are often not appreciated

*References 14, 16, 18, 20, 45, 54, 69, 76, 85, 89, 97, 101, 104, 105, 110, 120, 152, 154, 158, 164, 169, 170, 174, 192, 196, 197, 203, 204, 207, 210, 213, 216, 227-229, 232, 252, 256, 272, 273, 285, 297, 308, 312, 316, 326, 328, 334

until much later, but they are generally related to the unstable orthodontic positioning of the teeth outside of the boundaries of the alveolar process before or after surgery; this is discussed later in this chapter (see also Chapters 5 and 6).[208]

Condylar Resorption after Orthognathic Surgery

Although it is rare, *idiopathic condylar resorption* may present after orthognathic surgery (maxillary or mandibular osteotomies), routine orthodontic treatment, or routine dental procedures; it may also occur spontaneously and not in association with any specific form of treatment (see Chapter 36).[12,117,118,143,144,148,159,202,212,237,278,327] *Condylar resorption* is usually bilateral, symmetric, and seen in young females who are between 15 and 35 years old. Although no agreed upon etiology has been delineated for idiopathic condylar resorption, hormonal and autoimmune causes are suspected (see Chapter 36). *Unilateral condylar resorption* in association with compressive forces may also be seen after the excessive use of elastics in an attempt to overcome residual malocclusion. The clinical setting may involve heavy elastic use either as part of an orthodontic camouflage approach to avoid surgery (Fig. 17-5) or after orthognathic procedures that did not achieve the desired occlusion. In either case, heavy compressive forces on the condyle may result in temporomandibular joint symptoms followed by condylar resorption with a loss of condylar (posterior facial) height.

Continued Growth of the Mandible after Orthognathic Surgery

Under normal conditions, the mandible is the last bone in the craniofacial skeleton to complete growth. In general, mandibular growth should be complete between the ages of 14 and 16 years in girls and between the ages of 16 and 18 years in boys. Chronologic and skeletal age will vary from individual to individual, and mandibular growth in men may continue beyond the age of 18 years, but this is uncommon. Jaw growth in girls is usually complete within 2 years after the onset of regular menses. The completion of jaw growth may be assessed in the following ways: 1) via the growth-plate analysis of a hand and wrist radiograph 2) via the analysis of a quantitative condylar head bone scan in comparison to a lumbar vertebra (L2) 3) via the analysis of the mandibular length with the use of serial lateral cephalometric radiographs 4) via the analysis of cervical vertebral maturation with the use of serial lateral cephalometric radiographs 5) via the assessment of serial dental models in centric relation or 6) via the assessment of serial occlusal photographs taken in centric relation.*

Mandibular growth in individuals with micrognathia is generally complete at an earlier age than it is among normal "average" mandibular growers. Alternatively, the cessation of lower jaw growth in individuals with true mandibular excess may occur later than it does in average individuals (Figs. 17-6 and 17-7) (see Chapter 20); this is especially true for individuals with asymmetric mandibular excess growth anomalies (see Chapter 22). Alternatively, the individual with a typical Class II long face growth pattern (i.e., vertical maxillary excess combined with mandibular deficiency) can be assumed to have a normal mandibular growth vector without a tendency for late development (see Chapter 21). I believe that, if the long face patient's chronic nasal obstruction problem is simultaneously managed at the time of orthognathic surgery (i.e., septoplasty, inferior turbinate reduction, the widening of a tight nasal aperture, and the lowering of the nasal floor), further pathologic vertical growth is also likely to be arrested (see Chapter 10).

Ramus Remodeling and Malunion after Sagittal Split Ramus Osteotomy

With the use of rigid internal fixation (e.g., bicortical screws or plates and screws), the remodeling and malunion of the ramus osteotomy site is believed to be an uncommon cause of relapse.[109,115,179,206,236,248,302,324] Although infrequent, infection that has led to sequestration (e.g., of the buccal shelf of the proximal segment) and malunion has been reported. In most clinical settings, there is little evidence to support the concept of late relapse at the ramus osteotomy site after satisfactory initial healing has occurred (by approximately 5 weeks). However, limited bone contact or inadequate fixation of the bony segments may cause initial fibrous union followed by malunion and resulting malocclusion. This would be documented as morphologic change in the mandibular angle (i.e., clockwise rotation of the distal segment) in the presence of unchanged condylar morphology (i.e., no ongoing erosion of the condylar head). There would also be measurable change in either the mandibular length (i.e., as measured from the condyle to the pogonion) or the sella–nasion–B-point angle documented during the analysis of the serial cephalometric radiographs after surgery.

Proffit and colleagues do make a case for ramus remodeling with relapse after mandibular set-back when managing a Class III skeletal pattern.[248] These authors believe that, when the orthognathic procedure for the management of a Class III skeletal pattern is a mandibular set-back only versus a two-jaw surgery (i.e., with the primary movement being maxillary advancement in combination with only minor mandibular adjustment), then relapse at the ramus osteotomy site is a common occurrence. In a recent clinical study, Proffit and colleagues confirmed that—despite improvements in surgical fixation techniques (i.e., rigid fixation versus intraosseous wires and IMF)—when mandibular set-back only procedures are carried out for a Class III malocclusion, skeletal instability is often significant. They confirmed better long-term skeletal and occlusal stability when two-jaw surgery is performed for the management of Class III skeletal patterns (i.e., maxillary advancement with limited mandibular set-back).[251]

Text continued on p. 563

*References 26-28, 30, 33-38, 46-48, 68, 81, 92, 94, 119, 129, 130, 201, 214, 215, 218-224, 226, 258, 266, 270, 274-276, 280, 281, 286, 289, 290, 302, 332

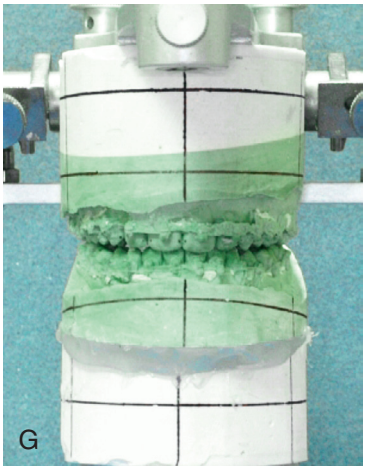
• **Figure 17-5** A 23-year-old woman was referred for surgical evaluation. During her high school years, she was recognized as having a malocclusion, and she underwent four bicuspid extractions and orthodontic treatment to neutralize the occlusion. During her college years, she experienced a closed lock of her jaw as a result of a dislocated disc within the left temporomandibular joint. After 3 months of conservative management, the patient underwent arthroscopy with lysis of adhesions. With a recurrence of the closed lock attributed to the disc in the left temporomandibular joint, she underwent discectomy and fat graft interposition. A review of the patient's earlier medical records, including dental models and radiographic analyses, confirmed the primary cause of current malocclusion to be a longstanding developmental jaw deformity that was initially treated with orthodontics alone. Dental relapse had occurred, and the patient's temporomandibular disorder was likely the result of unstable intercuspation. The relief of the temporomandibular disorder was achieved through an open-joint procedure but with the additional loss of posterior facial height and mandibular asymmetry. The patient was referred to this surgeon and agreed to a comprehensive orthodontic and orthognathic approach. The patient's procedures included bilateral sagittal split ramus osteotomies (horizontal advancement and counterclockwise rotation) and osseous genioplasty (horizontal advancement). Facial balance and satisfactory occlusion were achieved and maintained without the return of temporomandibular disorder. **A,** Frontal views in repose before and after retreatment. **B,** Frontal views with smile before and after retreatment.

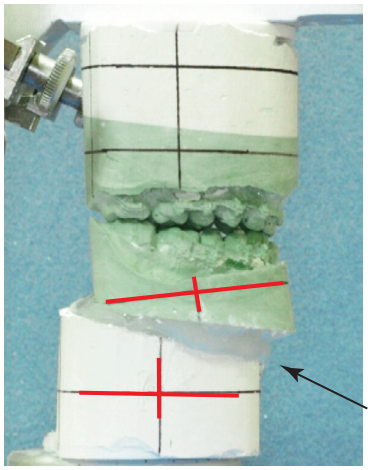
Continued

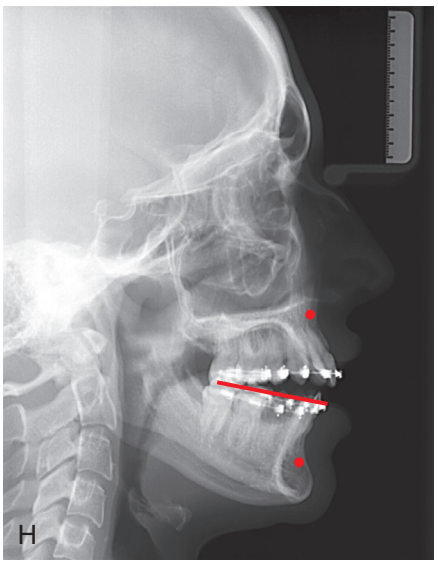
• **Figure 17-5, cont'd C,** Oblique facial views before and after retreatment. **D,** Profile views before and after retreatment. **E,** Panorex radiograph before retreatment. This image indicates a chronic flattening of the left condylar head.

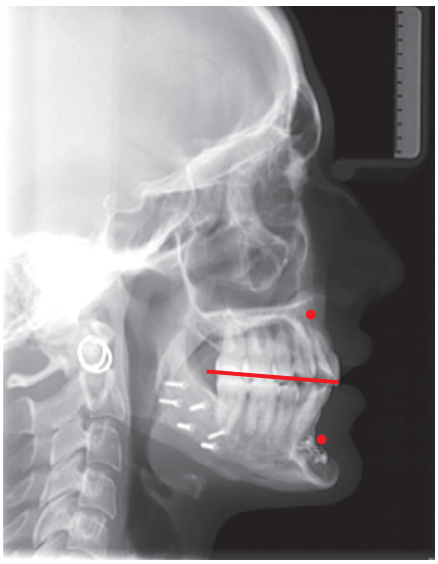
Pre surgery with 4 bicuspid extractions as a teenager

After re-treatment

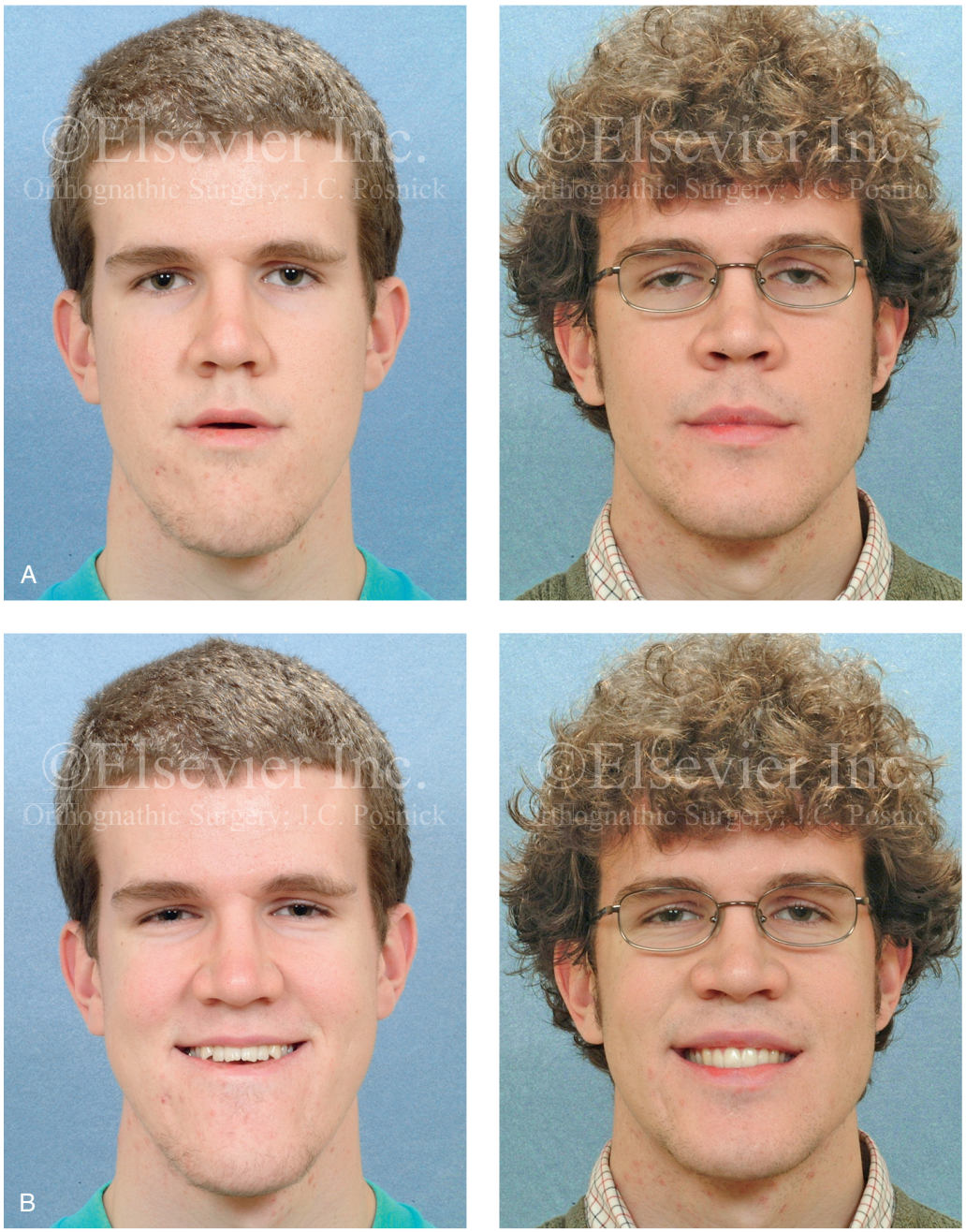








• **Figure 17-5, cont'd** **F,** Occlusal views before retreatment, with orthodontics in progress, and after successful retreatment. **G,** Articulated dental casts indicate analytic model planning. **H,** Lateral cephalometric radiographs before and after successful retreatment.



• **Figure 17-6** An 18-year-old man with malocclusion and a lifelong history of obstructed nasal breathing was referred for the surgical evaluation. The pattern of deformity was maxillary deficiency in combination with relative mandibular excess, which resulted in a severe Class III negative overjet anterior open bite. He had previously undergone growth modification and camouflage orthodontics including lower bicuspid extractions, without success. After a thorough evaluation, he underwent 12 months of orthodontic decompensation followed by jaw and intranasal surgery. The patient's surgery included Le Fort I osteotomy in segments (horizontal advancement, clockwise rotation, transverse widening, and correction of the arch form) with interpositional grafting; sagittal split ramus osteotomy (minimal counterclockwise rotation); osseous genioplasty (bone burring and recontouring); and septoplasty and inferior turbinate reduction. These procedures were followed by finishing orthodontics. The occlusion achieved during the operation and maintained at the patient's debracketing 6 months later was ideal. By 2 years after surgery, however, a symmetric Class III negative overjet malocclusion had recurred. **A,** Frontal views before and 2 years after reconstruction. **B,** Frontal views with smile before and 2 years after reconstruction.

• **Figure 17-6, cont'd** A review of the 2-year postoperative dental models indicated stable maintenance of the arch width and of the correction of the curve of Spee. The models could be articulated into a similar occlusion as had been achieved at the time of operation. Therefore, the cause of change in occlusion could not be attributed to the segmental osteotomies or dental relapse. The cause of the recurrent malocclusion was felt to be either ongoing mandibular growth or Le Fort I osteotomy site relapse. An analysis of the postoperative cephalometric radiographs—which showed an increase in the sella–nasion–B-point angle and the maintenance of the sella–nasion–A-point angle—confirmed maxillary stability. Ongoing mandibular growth was the cause of the malocclusion. Treatment options included Le Fort I osteotomy (3-mm advancement) versus sagittal split ramus osteotomies (3-mm set-back). The patient was pleased with the functional improvements (i.e., swallowing, speech, chewing, lip closure/posture, and breathing) and the enhanced facial aesthetics without temporomandibular disorders. He elected to receive no further treatment. **C,** Oblique facial views before and 2 years after reconstruction. **D,** Profile views before and 2 years after reconstruction.

Continued

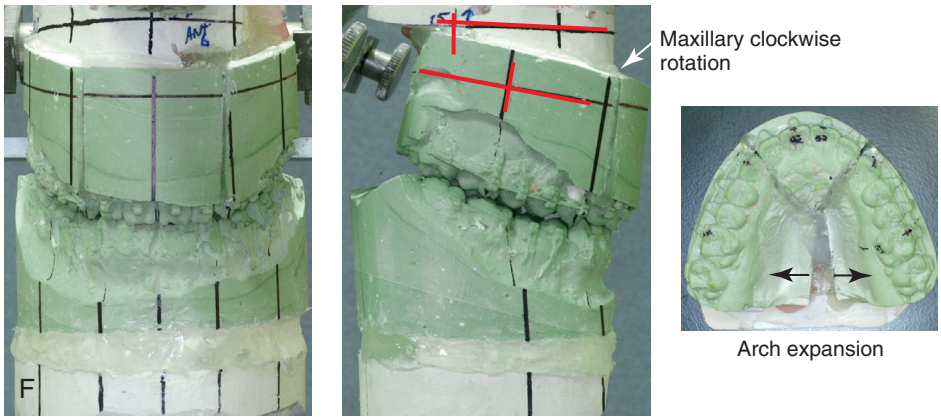
Prior to retreatment with lower bicuspid extractions

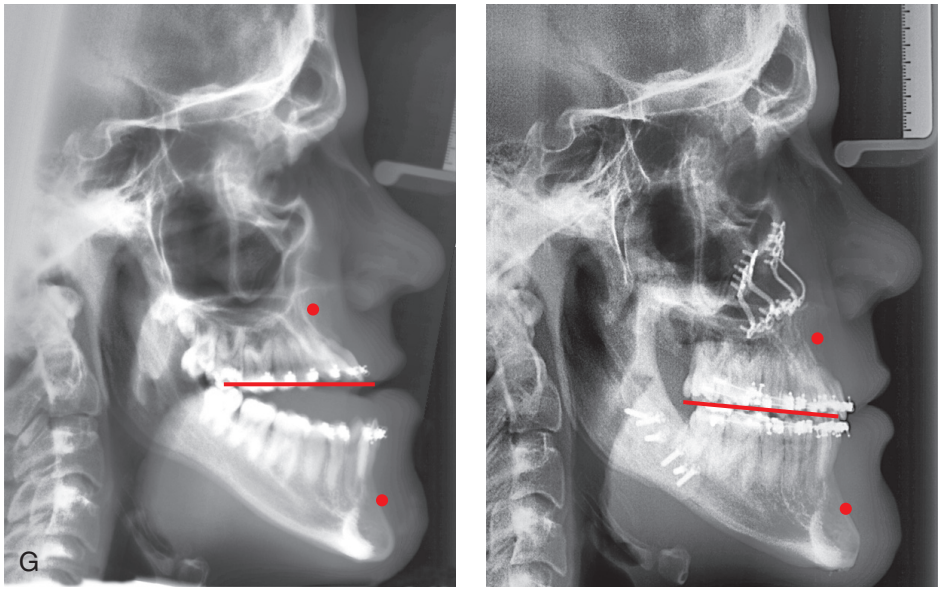
Pre surgery

5 weeks after surgery

2 years after treatment

• **Figure 17-6, cont'd E,** Occlusal views before retreatment, immediately before surgery, 5 weeks after surgery, and 2 years after treatment.





• **Figure 17-6, cont'd F,** Articulated dental casts after analytic model planning. **G,** Lateral cephalometric radiographs before and after treatment.

Le Fort I Osteotomy: Skeletal Stability and Relapse

Immediate (Early) Malocclusion after Le Fort I Osteotomy

Immediate (early) malocclusion that is recognized either intraoperatively or during the first several days or weeks after surgery may occur after a Le Fort I osteotomy unless the following intraoperative conditions are met (see Figs. 17-1 through Fig 17-4)*:

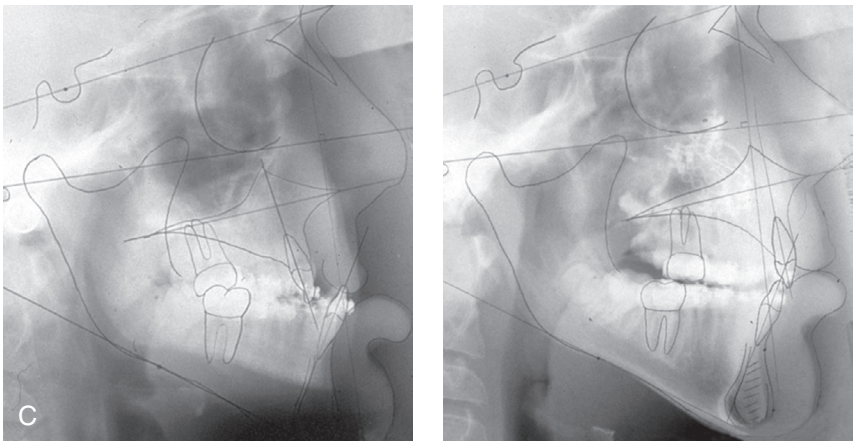
1. After Le Fort I down-fracture osteotomy, full mobilization of the maxilla and the segments (if segmentation has been carried out) is accomplished before the placement of fixation.
2. An accurate prefabricated acrylic splint is secured to the maxillary occlusal surfaces. The splint stabilizes the new maxillary arch form (i.e., the arch width and the curve of Spee) when segmental osteotomies are carried out.
3. After the securing of the splint to the maxillary dentition, IMF is applied to establish the new horizontal position of the maxilla; the dental midline (yaw orientation); the preferred maxillary plane orientation (pitch orientation); and any cant correction (roll orientation) required.
4. With the condyles seated in the terminal hinge position and IMF established via the prefabricated splint, the maxillomandibular complex is rotated until the desired vertical position of the maxilla is achieved.
   - Bony prematurities are common with maxillary impaction. It is easy for the surgeon to overlook interferences in the posterior region of the lateral nasal walls just medial to the descending palatine vessels (see Fig. 15-25, *A* and *B*).

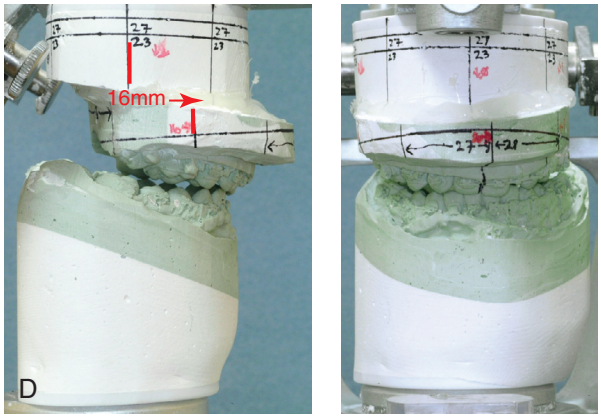
*References 22, 26, 52, 53, 79, 82, 87, 128, 134, 145, 147, 178, 187-189, 191, 193, 194, 233, 234, 239, 241, 244, 259, 264, 279, 287, 307, 317

- Care should be taken to prevent premature contacts across the Le Fort I osteotomy site, which are followed by a "slide" into maximum bone contact during further vertical closure of the maxillomandibular complex. This would cause condylar displacement with malocclusion that should be recognized at the release of the IMF. The malocclusion would typically present as either a symmetric anterior open bite (if both condyles are displaced) or as an asymmetric lateral shift in the bite (if one condyle is displaced).

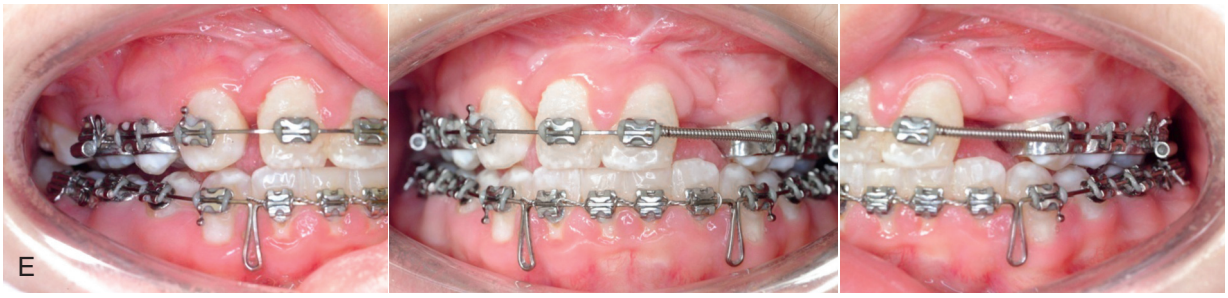
5. There must be adequate bone contact across the osteotomy site after the maxilla is stabilized with plate and screw fixation into the desired position. If not, an interpositional graft of sufficient quality should be placed and secured (see Chapters 15 and 18). Without

• **Figure 17-7** A 13-year-old boy born with bilateral cleft lip and palate arrived for surgical evaluation with a severe cleft jaw deformity. He underwent primary lip and palate repair, a pharyngeal flap procedure, bone grafting, and a columella lengthening procedure at another institution. He was referred to this surgeon with severe maxillary hypoplasia that was causing profound effects on his speech articulation, chewing ability, breathing, and self-esteem. For this reason, early jaw reconstruction was chosen. He underwent maxillary Le Fort I osteotomy (horizontal advancement and vertical lengthening with interpositional [iliac] bone grafting). This was carried out with the understanding that further maxillary growth should not be expected, whereas mandibular growth would likely continue and result in a recurrent Class III. A review of the longitudinal cephalometric radiographs after surgery confirmed no significant horizontal maxillary relapse but ongoing mandibular growth. **A,** Frontal views with smile before and 1 year after surgery just before the removal of the orthodontic appliances. **B,** Oblique facial views before and 1 year after surgery.





Pre surgery



1 year after treatment

• **Figure 17-7, cont'd** **C,** Lateral cephalometric views before and 1 year after surgery. **D,** Articulated dental casts that indicate analytic model planning. **E,** Occlusal views before and 1 year after surgery, just before debanding.

Continued

• **Figure 17-7, cont'd F,** Facial and occlusal views from when the patient was 18 years old (i.e., 5 years after Le Fort I advancement) that indicate recurrent Class III malocclusion as a result of ongoing mandibular growth.

adequate bone contact across the osteotomy site, *fibrous union* may occur (see Chapter 16). Another possibility is that of initial bony union with *relapse* (i.e., *malunion*) occurring as full healing proceeds during the 6 to 12 months after surgery, thereby resulting in late malocclusion.

6. Adequate plate and screw stabilization of the maxilla generally requires the placement of titanium fixation at each pyriform rim and each zygomatic buttress (see Fig. 15-26). When interpositional grafts are required, additional titanium fixation is placed from the superior maxilla and extended across the graft to the dentate segment (see Fig. 15-29, *A, B,* and *C*). Additional plate and screw fixation may also be placed horizontally across any interdental osteotomies, if needed.
7. With release of the IMF, the occlusion is checked as the mandible is rotated (opened and closed) with the condyles seated in the terminal hinge position. The occlusion must evenly contact and fit passively into the splint. If not, the plate and screw fixation across the Le Fort I osteotomy is released. Any bony interference is then identified and removed. The maxilla is again repositioned and the fixation reapplied (i.e., repeat Steps 4 through 7) until the planned occlusion is achieved and confirmed.

Late Malocclusion after Le Fort I Osteotomy

Skeletal causes of a late malocclusion after Le Fort I osteotomy may include the following: 1) remodeling (relapse) at the Le Fort I osteotomy site (as described later in this chapter) 2) fibrous union at the Le Fort I osteotomy site (as described later in this chapter) 3) remodeling (relapse) at the segmental osteotomy sites (as described later in this chapter) 4) condylar resorption (as described previously in this chapter and in Chapter 36)[143] and 5) continued mandibular growth (as described previously in this chapter; see Figs. 17-6 and 17-7).* *Dental causes* of malocclusion after Le Fort I osteotomy are often not realized until much later. They are related to the unstable orthodontic positioning of the teeth outside of the boundaries of the alveolar process before or after surgery (as described later in this chapter and in Chapters 5 and 6).[208]

The precise cause of relapse at the Le Fort I osteotomy site is often difficult to pinpoint , but it is generally related to the following: 1) inadequate mobilization of the down-fractured maxilla before the placement of fixation 2) inadequate bone contact across the osteotomy site after the fixation is applied 3) inadequate (non-rigid) plate and screw fixation across the osteotomy site or 4) excessive occlusal forces or movement transmitted across the osteotomy site during initial healing.†

Fibrous union of the maxilla after Le Fort I osteotomy is an infrequent occurence. When it occurs, expect to find painless mobility of the upper jaw, with or without malocclusion. Risk factors should be recognized, at which point surgical techniques and postoperative management can be adjusted to limit this complication (see Fig. 16-21). The combination of extensive horizontal advancement and vertical lengthening will result in large osteotomy site defects

*References 27, 28, 30, 33-38, 46-48, 68, 81, 92, 94, 119, 129, 130, 201, 214, 215, 218-224, 226, 258, 266, 270, 274-276, 280, 281, 286, 289, 301, 302

†References 3, 4, 11, 14-16, 18-20, 24, 29, 63, 83, 95, 99, 109, 114, 121, 135, 138, 139, 141, 142, 146, 149, 172, 179, 240, 242, 243, 245, 249, 250, 253, 273, 321, 331, 333, 334

with limited bone contact. Failure to form adequate bony bridges at the healing sites is likely to result in delayed healing, fibrous union, or late relapse. Negative influences on bone healing at the Le Fort I osteotomy site may include the following: (1) injury to flap circulation (2) thin osteoporotic bone at the zygomatic buttress and the pyriform rims, with inadequate fixation and (3) the decision to not place interpositional grafts when the bone gaps are of critical size. Additional postoperative healing risk factors include the following: (1) infection or wound dehiscence after surgery (2) the presence of a heavy occlusion (e.g., grinding, clenching) (3) too rapid of a return to normal chewing forces (4) excessive mouth opening against intermaxillary elastics and (5) the breakage (stress fracture) of the fixation plates. Systemic conditions that are known risk factors for poor bone healing include the following: (1) diabetes (2) collagen disease (3) vascular disease (4) osteoporosis or bisphosphonate use (5) nicotine abuse (6) poor nutritional status; and (7) previous radiation treatment.

Relapse across the segmental maxillary osteotomy sites after palatal widening is generally related to the following: (1) inadequate mobilization of the segments (2) inadequate surgical splinting of the new arch shape during initial healing (i.e., the first 5 weeks) (3) inadequate or unstable interdigitating occlusion between the maxillary and mandibular teeth after the splint is removed or (4) inadequate medium-term orthodontic maintenance (i.e., lasting approximately 6 months) with an appropriate appliance after the surgical splint is removed (see Figs. 17-3 and 17-8). Transverse relapse after the removal of the surgical splint may also occur as a result of the use of counterproductive intermaxillary elastic forces. For example, the use of vertical elastics placed on the buccal brackets of the upper and lower molars in an attempt to close posterior open bites may result in the simultaneous unintended collapse of the arch width.

Frequent technical errors that can occur when completing Le Fort I segmental arch expansion and that may result in malocclusion include the following:

1. *Inadequate skeletal mobilization.* When significant widening of the posterior maxilla is required, bilateral (rather than unilateral) parasagittal osteotomies will assist with the tension-free placement of the segments into the splint (see Chapter 15).
2. *Inadequate splint strength.* The use of a non-flexible splint (e.g., a reinforced U-shaped acrylic splint, transpalatal support) is essential to maintain the arch width during initial healing (see Chapter 13).
3. *Inadequate soft-tissue mobilization.* After the completion of segmental osteotomies, the stretching of the palatal soft tissues through the down-fracture (i.e., the use of a spreader forceps) improves the tension-free placement of the segments into the splint.
4. *Inadequate postsurgical care.* After the splint is removed (i.e., 5 weeks after surgery), the seamless transfer of responsibility from the surgeon to the orthodontist for ongoing maxillary arch form maintenance is essential. The effective use of orthodontic mechanics is generally needed for at least 6 months to retain the arch form. (This is described later in this chapter.)

Orthodontic Treatment: Dental Stability and Relapse

The objective of orthodontics in conjunction with jaw surgery is to align the dentition in each arch by placing the maxillary and mandibular teeth fully within the basal bone. When the jaws and jaw segments are then surgically repositioned, the preferred occlusion is achieved. Following Andrews' Six Keys to Normal Occlusion provides the framework for the accomplishment of the orthodontic objectives (see Chapters 5 and 6).[208]

Moen and colleagues carried out a prospective clinical study to evaluate the short-term occlusion achieved and the long-term occlusion maintained after bilateral sagittal split ramus osteotomies with advancement for the treatment of Class II skeletal deformities.[208] Prospectively, 36 patients with Class II skeletal patterns who were to undergo bilateral sagittal split ramus osteotomies agreed to participate in the study. The mean age at operation was 33 years (range, 16 to 59 years). All of the study patients underwent perioperative orthodontics and were followed clinically, with dental models and radiographs (i.e., lateral cephalometric and Panorex). These standard records were obtained at 8 weeks, 1 year, and 13 years after surgery. Interestingly, for the majority of patients, cephalometric analysis confirmed insignificant skeletal changes during long-term follow up after successful initial healing had occurred. Long-term recurrent malocclusion was found to be primarily dental rather than skeletal in origin.[208] *To achieve and then maintain the ideal occlusion, the importance of the stable orthodontic positioning of the teeth in the alveolus cannot be overstated* (Figs. 17-9 through 17-14).

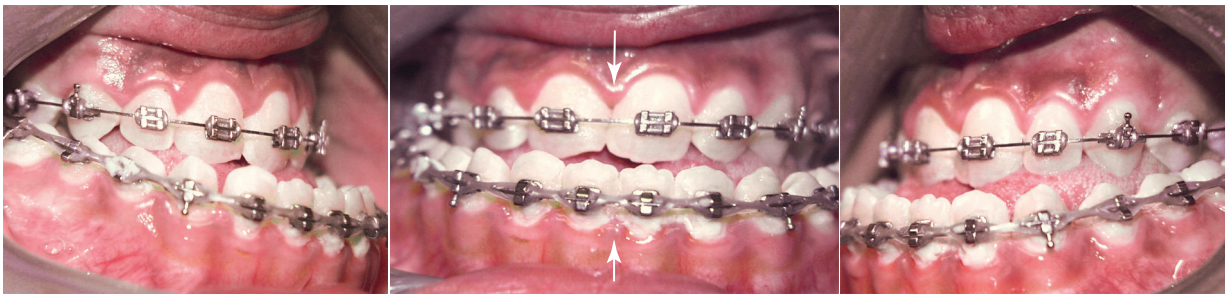
The lips, tongue, cheeks, soft palate, mandible and periodontal ligaments function dynamically with the stationary (immobile) maxilla and the teeth to achieve chewing, swallowing, breathing, speech, and lip closure. Parafunctional habits of these structures (e.g., posturing of the tongue, lips, and jaw) may influence the position of the teeth and the supporting skeletal structures. *Dental compensations* are expected biologic responses when a jaw disharmony is present at birth or when it occurs during growth. The orthodontic mechanical removal of dental compensations should position the teeth within the alveolar process to then maximize the surgical correction and also to provide a framework for long-term occlusal stability and periodontal health.*

Extraction Therapy to Eliminate Crowding

Dental extractions may be necessary when there is inadequate alveolar bone to eliminate the dental crowding and

*References 2, 7, 55, 61, 71, 77, 78, 80, 93, 100, 103, 157, 186, 190, 205, 217, 247, 246, 267, 288, 330, 335, 336

• **Figure 17-8** A 15-year-old high school student was referred by her orthodontist for surgical evaluation of a long-standing developmental jaw deformity with malocclusion and a lifelong history of obstructed nasal breathing. The pattern of growth disturbance was maxillary deficiency in combination with relative mandibular excess, and this resulted in a Class III negative overjet anterior open bite malocclusion. The patient had undergone growth modification and camouflage orthodontics, without success. Referral to this surgeon was followed by orthodontic decompensation in conjunction with jaw and intranasal surgery and then finishing orthodontics. The patient's surgery included maxillary Le Fort I osteotomy in two segments (horizontal advancement, transverse widening, and vertical lengthening); sagittal split ramus osteotomies (counterclockwise rotation and minimal horizontal advancement); and septoplasty and inferior turbinate reduction. The surgical occlusion that was initially achieved at operation and then maintained during the early phase of healing was stable. By 6 to 12 months after surgery, a degree of posterior crossbite had recurred. **A,** Frontal views with smile before and after reconstruction. **B,** Oblique facial views before and after reconstruction.

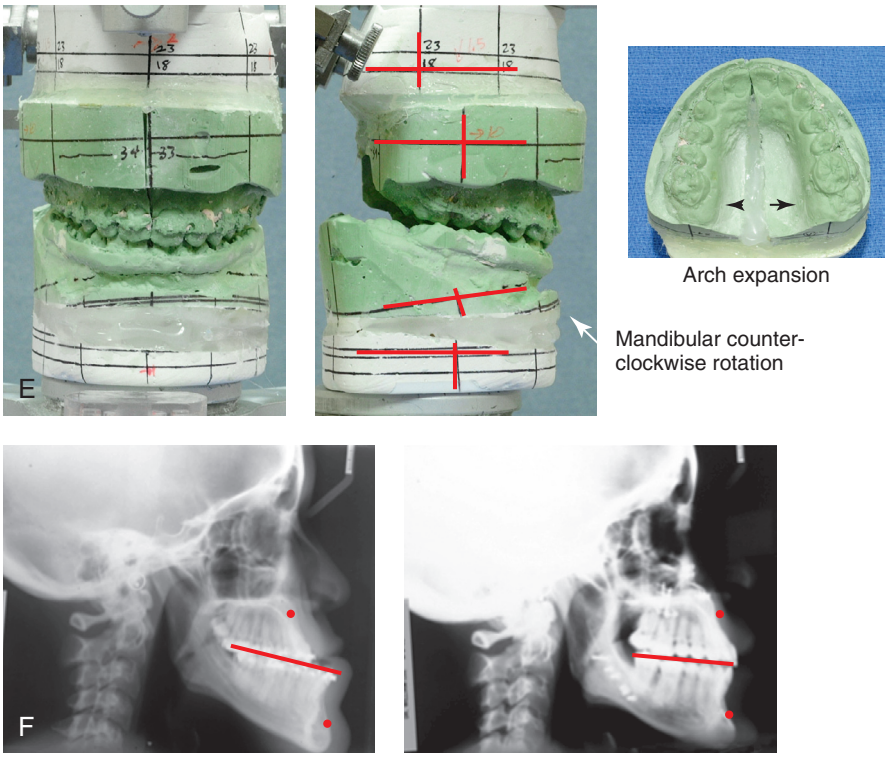


Pre surgery

2 years after treatment

• **Figure 17-8, cont'd** A review of the postoperative dental models indicated intermolar transverse constriction as compared with what was surgically planned, executed, and achieved at 5 weeks after surgery, when the prefabricated splint was removed. A review of the dental models confirmed a combination of skeletal and dental relapse that resulted in maxillary arch constriction. In retrospect, the postoperative orthodontic mechanics of the labial arch wire to maintain the surgically achieved arch width were not adequate. The use of a transpalatal wire or an acrylic palatal plate maintained for 6 months after surgery may have prevented maxillary arch form relapse after the surgical splint was removed (i.e., 5 weeks after surgery). Treatment options for the residual malocclusion include redo Le Fort I segmental osteotomies with expansion versus surgically assisted rapid palatal expansion. The patient was pleased with the improvements (i.e., chewing, speech articulation, lip closure/posture, and breathing) and the enhanced facial aesthetics. She was left with a functional occlusion without centric relation–centric occlusion discrepancies or temporomandibular disorder. She elected to receive no further treatment. **C,** Profile views before and after reconstruction. **D,** Occlusal views with orthodontic decompensation in progress and 3 years after treatment. The positive overjet overbite is maintained but with recurrent maxillary transverse constriction.

Continued



• **Figure 17-8, cont'd E,** Articulated dental casts that indicate analytic model planning. **F,** Lateral cephalometric radiographs before and after reconstruction.

to remove compensations without displacing the roots outside of alveolar housing. Opinions regarding the indications for extractions have changed remarkably over the past 150 years since the beginning of "modern" orthodontics.

During the 1880s, Edward Angle joined other progressive restorative dentists of his time in their opposition to a casual attitude toward the extraction of teeth (see Chapter 2). Angle's belief was that, through diligent effort, the orthodontic alignment of the full complement of teeth was possible. He felt that the orthodontist's ultimate objective was for every person to have the potential for an ideal relationship of all 32 natural teeth.

Dr. Angle believed that the cause of most Class II or Class III problems was often the result of abnormal stresses on the jaws. Through the orthodontist's active application of controlled pressure on the teeth (i.e., arch expansion) and occasionally directly on the bone (i.e., chin cup and headgear), Dr. Angle believed (at least early during his career) that the clinician could change jaw growth and overcome the presenting Class II or Class III deformity.

Angle also taught that proper jaw and dental function on the part of the individual should maintain the teeth in their orthodontically corrected position. He believed—or at least hoped—that, after the teeth were placed in proper occlusion, the natural forces transmitted to the teeth would cause alveolar bone to generate around them.

Through trial and error, he realized that simply tipping the teeth into a new position was often inadequate. He developed the *Ribbon Arch* appliance and then the *Edgewise* appliance to "bodily" move the teeth. These were the first clearly described and reliably designed orthodontic appliances that were capable of controlling root position. At this time, Dr. Angle claimed that these were "bone-growing appliances."[10] For Angle, this work was simply an extension of Wolff's law of bone, which states that the internal architecture of a bone represents the stress pattern that is placed on it.

Angle's treatment philosophy involved arch expansion with the use of vectored rubber bands and other appliances, as needed, to bring the teeth into occlusion. To Angle, at least early during his career, the term *relapse* as it applied after orthodontic expansion that had been carried out to uncrowd and correct overjet or overbite simply meant that the achieved occlusion was inadequate or technically incorrect. According to Angle, "If a correct occlusion had been produced [by the orthodontist] the result would be stable."[10] Therefore, he felt, "if the orthodontic result was not stable, the fault was either that of the orthodontist or poor compliance by the treated patient, not the theory."[10]

For the most part, Angle believed that, for each individual, ideal facial aesthetics would result when the teeth were placed in ideal occlusion. Whether the patient agreed or not, by divine definition, the best facial appearance for that patient would be achieved when the dental arches had been expanded so that all of the teeth were in ideal occlusion. Angle adamantly believed that extractions were not indicated.

Text continued on p. 592

• **Figure 17-9** A female high-school student was referred by her orthodontist for surgical evaluation. Since her mid childhood years, the patient was known to have a long face growth pattern, and she had a lifelong history of obstructed nasal breathing. She had undergone attempted growth modification followed by orthodontic camouflage, without success. At the time of referral, the patient's concerns included lip incompetence, gummy smile, weak profile, malocclusion, and nasal obstruction. She agreed to a surgical correction. The orthodontic treatment plan then changed in preparation for orthognathic surgery. Extractions were not carried out to correct the maxillary and mandibular arch forms. The patient then underwent maxillary Le Fort I osteotomy (horizontal advancement and vertical intrusion); bilateral sagittal split ramus osteotomies (minimal horizontal advancement and counterclockwise rotation); osseous genioplasty (vertical reduction and horizontal advancement); and septoplasty, inferior turbinate reduction, and recontouring of the nasal floor. Successful initial surgical healing was achieved. The occlusion maintained at splint removal (i.e., 5 weeks after surgery) was as planned. The orthodontic appliances were removed 6 months after surgery, with ideal occlusion and orthodontic retention continued. The patient returned 3 years after surgery with complaints of recurrent anterior open-bite malocclusion. **A,** Frontal views in repose before and 3 years after surgery. **B,** Frontal views with smile before and 3 years after surgery.

Continued

• **Figure 17-9, cont'd** An analysis was carried out that included the review of lateral cephalometric and Panorex radiographs taken before surgery, 5 weeks after surgery, and 3 years after surgery. The patient had no temporomandibular disorder, and there was no evidence of condylar resorption. Therefore, idiopathic condylar resorption was ruled out. Cephalometric analysis of the presurgical, early postoperative, and late postoperative cephalometric radiographs revealed satisfactory initial repositioning and healing of the jaws without skeletal relapse at 3 years. A comparison of the 5-week postoperative and 3-year postoperative sella–nasion–A-point and sella–nasion–B-point angles and facial heights confirmed skeletal stability. A review of the arch form (i.e., the dental models) suggested orthodontic relapse that included transverse maxillary constriction and incisor intrusion. The mandibular arch form also indicated dental relapse. Treatment options included redo Le Fort I osteotomy in three segments for the correction of the arch form versus surgically assisted rapid palatal expansion. Orthodontics would also be required. The advantages of four bicuspid extractions as part of dental decompensation were also discussed. The patient was pleased with the enhanced facial aesthetics (i.e., a less gummy smile, an improved profile, and lip closure) and the improved breathing that was achieved. She also did not have any temporomandibular disorder. She elected to receive no further treatment. **C,** Oblique facial views before and 3 years after surgery. **D,** Profile facial views before and 3 years after surgery.

Prior to retreatment

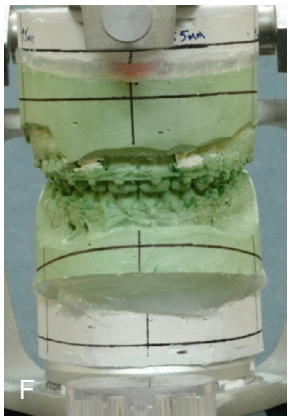
Pre surgery

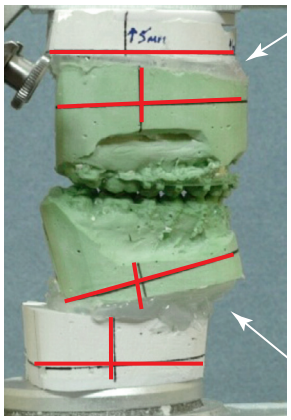
5 weeks after surgery

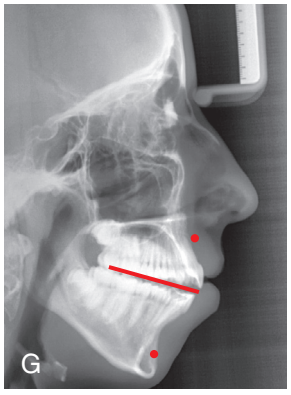
3 years after treatement

• **Figure 17-9, cont'd E,** Occlusal views before the orthodontic retreatment, immediately before surgery, 5 weeks after surgery, and 3 years after surgery.

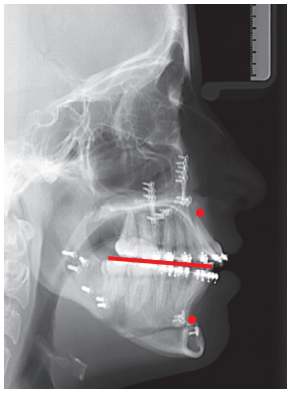
Continued



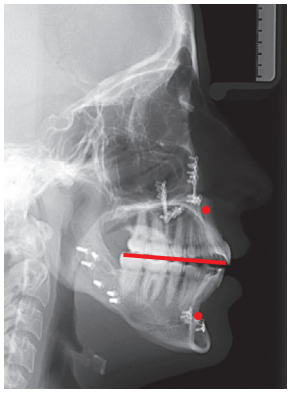




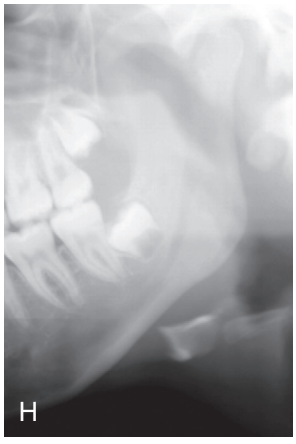
Pre ortho



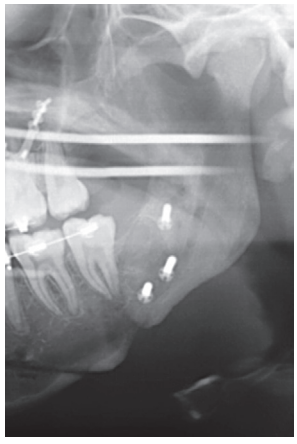
5 weeks after surgery



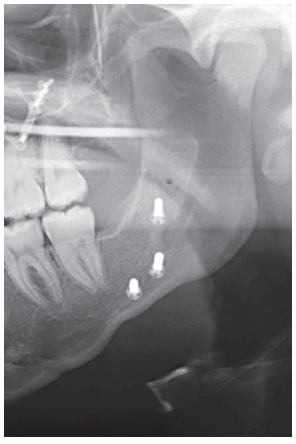
3 years after surgery



Pre surgery

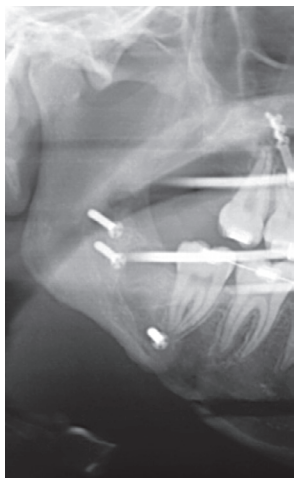


5 weeks after surgery

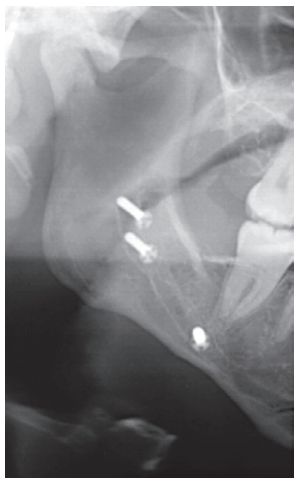


3 years after surgery

Pre surgery



5 weeks after surgery



3 years after surgery

• **Figure 17-9, cont'd** **F,** Articulated dental casts that indicate analytic model planning. **G,** Serial lateral cephalometric views before orthodontic retreatment, 5 weeks after surgery, and 3 years after surgery. **H,** Serial Panorex views of the left condyle–ramus before surgery, 5 weeks after surgery, and 3 years after surgery. **I,** Serial Panorex views of the right condyle–ramus before surgery, 5 weeks after surgery, and 3 years after.

• **Figure 17-10** A 14-year-old girl was referred by her orthodontist for surgical evaluation. She presented with a long face Class II growth pattern and a lifelong history of obstructed nasal breathing. She underwent non-extraction orthodontics, including mechanics to correct the maxillary arch form. The patient's surgery included maxillary Le Fort I osteotomy (vertical intrusion, horizontal advancement, and counterclockwise rotation); sagittal split ramus osteotomies (horizontal advancement and counterclockwise rotation); osseous genioplasty (vertical reduction and horizontal advancement); and septoplasty, inferior turbinate reduction, and nasal floor recontouring. Healing occurred without complications. The occlusion that was initially achieved and maintained at 5 weeks after surgery was as planned. Unfortunately, the patient refused continued postoperative orthodontics and the appliances be removed 3 months after surgery. She was also not compliant with retainer use. By 1 year after surgery, there was a recurrence of the anterior open bite. **A,** Frontal views in repose before and after treatment. **B,** Frontal views with smile before and after treatment.

Continued

• **Figure 17-10, cont'd** A review of the dental models confirmed dental relapse with a recurrent accentuated curve of Spee and arch constriction of the maxilla. In retrospect, segmental orthodontic mechanics followed by segmental maxillary surgery would likely have prevented this problem. At this time, treatment options include segmental orthodontic mechanics and redo Le Fort I osteotomy in segments. The patient and her family were pleased with the improvements in lip closure/posture, a diminished gummy smile, improved profile aesthetics, and better breathing during the day and night. The patient did not have temporomandibular disorder, and she elected to received no further treatment. **C,** Oblique facial views before and after treatment. **D,** Profile views before and after treatment.

Prior to treatment

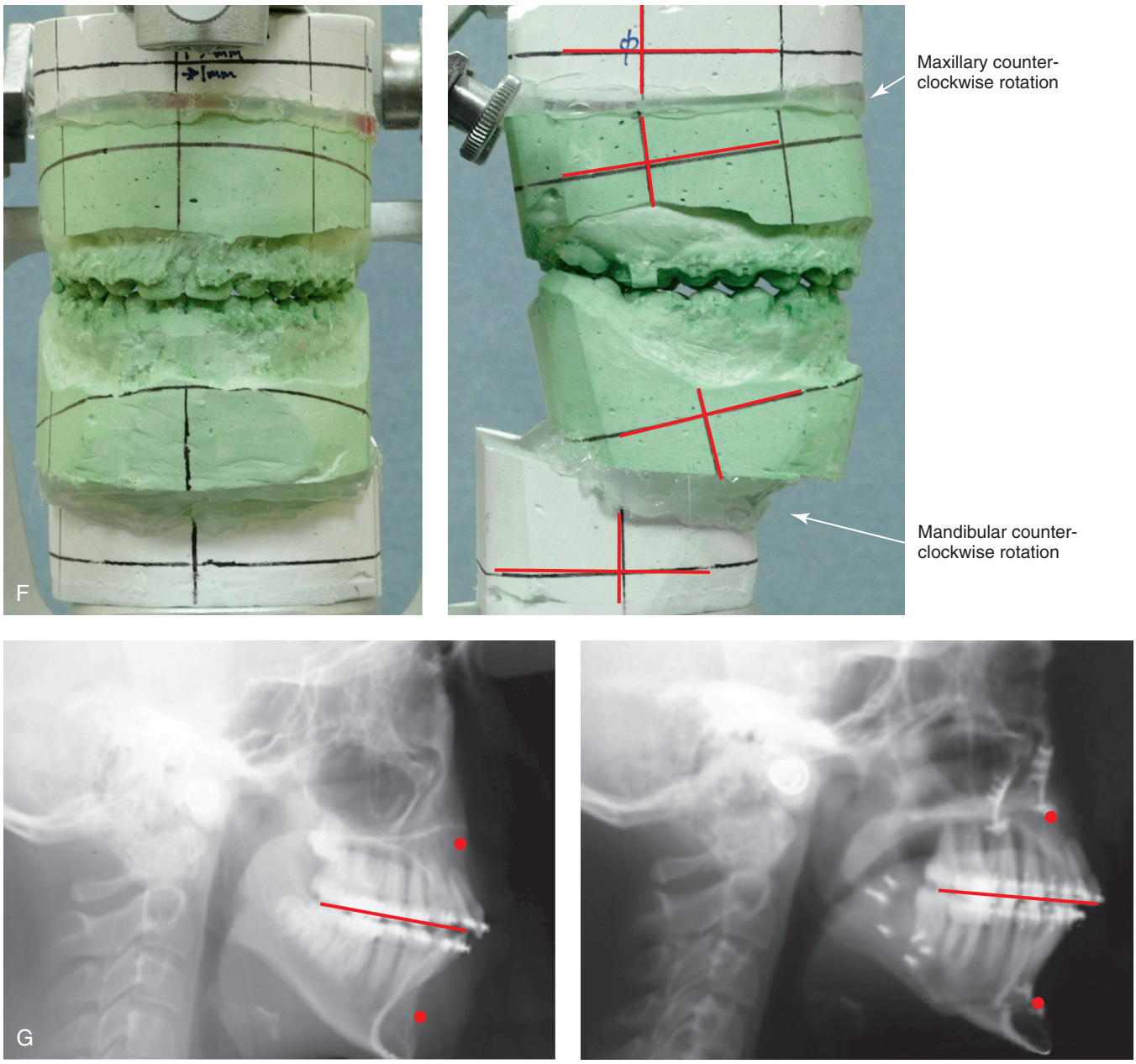
Pre surgery

5 weeks after surgery

1 year after treatment

• **Figure 17-10, cont'd E,** Occlusal views in mixed dentition before treatment, with preoperative orthodontics complete, with initial surgical occlusion achieved (i.e., 5 weeks after surgery), and 1 year after treatment. Note: Discoloration of right maxillary central incisor crown suggests pulpal pathology.

Continued



• **Figure 17-10, cont'd F,** Articulated dental casts that indicate analytic model planning. **G,** Lateral cephalometric views before and after treatment.

• **Figure 17-11** A 17-year-old boy was referred by his orthodontist for the surgical evaluation of a long-standing developmental jaw deformity with malocclusion and a lifelong history of obstructed nasal breathing. The patient had undergone both growth modification and camouflage orthodontics, without success. Examination confirmed a Class III long face growth pattern with an anterior open bite. He then underwent orthodontic mechanics in preparation for jaw surgery. The maxillary arch was leveled with orthodontic mechanics. The patient's surgery included maxillary Le Fort I osteotomy (horizontal advancement, vertical shortening, and clockwise rotation); bilateral sagittal split osteotomies of the mandible (minimal set-back and clockwise rotation); and septoplasty, inferior turbinate reduction, and nasal base recontouring. An osseous genioplasty with horizontal advancement would have further enhanced the patient's facial aesthetics but was refused. The surgery was followed by finishing orthodontics. The patient's occlusion at 5 weeks after surgery was excellent, and it remained so at the time of the debracketing 6 months after surgery. By 1 year after surgery, a slight Class III tendency with posterior constriction was evident. **A,** Frontal views in repose before and after surgery. **B,** Frontal views with smile before and after surgery.

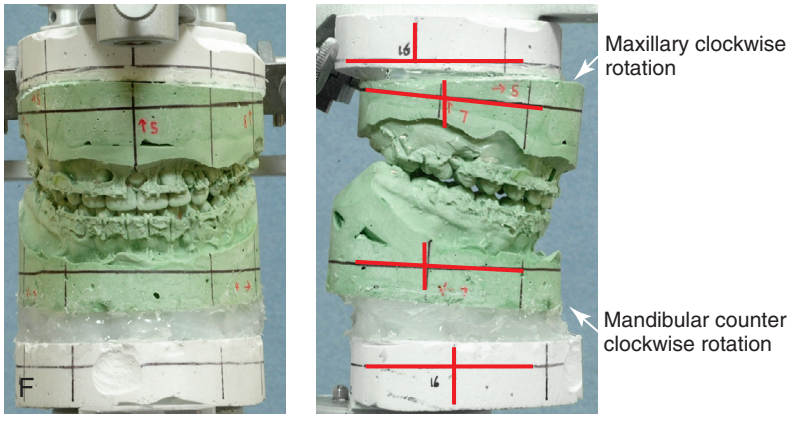
Continued

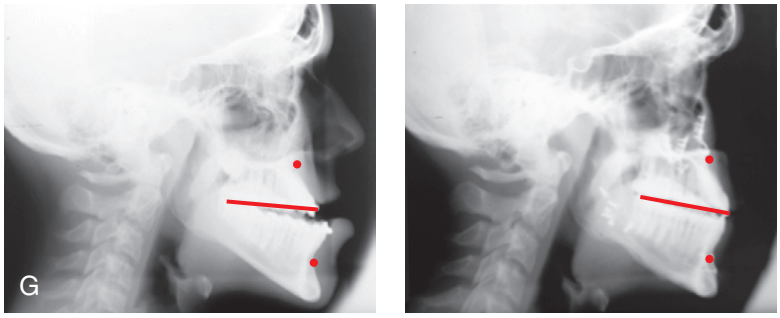
• **Figure 17-11, cont'd** By 2 years after surgery, the anterior occlusion was edge to edge, and the maxillary arch form was irregular. The cause of the recurrent malocclusion was felt to be either ongoing mandibular growth or a loss of maxillary arch form as a result of dental relapse. A review of the cephalometric data suggested stability of the mandible (i.e., the sella–nasion–A-point and sella–nasion–B-point angles were unchanged at intervals after surgery). A review of the dental models confirmed dental relapse with a change in the maxillary arch form (i.e., constriction in the arch width and an accentuated curve of Spee). Treatment options include maxillary Le Fort I osteotomy in segments for the correction of the arch form versus surgically assisted rapid palatal expansion. An osseous genioplasty was also offered to improve the patient's profile aesthetics. The patient was pleased with the functional improvements (i.e., chewing and speech articulation), and he did not have temporomandibular disorder; he also appreciated the enhanced facial aesthetics. He elected to receive no further treatment. **C,** Oblique facial views before and after surgery. **D,** Profile views before and after surgery.

Pre surgery

1 year after surgery

2 years after treatment





• **Figure 17-11, cont'd E,** Occlusal views immediately before surgery and at 1 year and 2 years after surgery. **F,** Articulated dental casts that indicate analytic model planning. **G,** Lateral cephalometric radiographs before and after surgery.

©Elsevier Inc.
Orthognathic Surgery: J.C. Posnick

• **Figure 17-12** A 20-year-old female college student was referred for surgical evaluation. During the mixed dentition, she was noted to have a long face growth pattern with an anterior open bite. She was treated from the time that she was 11 to 15 years old with growth-modification followed by a four-bicuspid orthodontic camouflage approach, without success. She presented for surgical evaluation with complaints of chronic obstructed nasal breathing; an anterior open bite that caused difficulty with chewing and with speech articulation; wide lip separation with lip strain at closure; and a retrusive profile appearance. There was generalized root resorption and early anterior mandibular labial bone loss and gingival recession. A comprehensive evaluation also confirmed septal deviation and inferior turbinate hypertrophy with increased nasal airway resistance. The patient agreed to an orthodontic and orthognathic/intranasal surgical approach. Her surgery included maxillary Le Fort I osteotomy in segments (arch form correction, horizontal advancement, vertical shortening, and counterclockwise rotation); bilateral sagittal split ramus osteotomies (horizontal advancement and counterclockwise rotation); osseous genioplasty (horizontal advancement); and septoplasty, inferior turbinate reduction, and nasal floor recontouring. **A,** Frontal views in repose before and after treatment. **B,** Frontal views with smile before and after treatment.

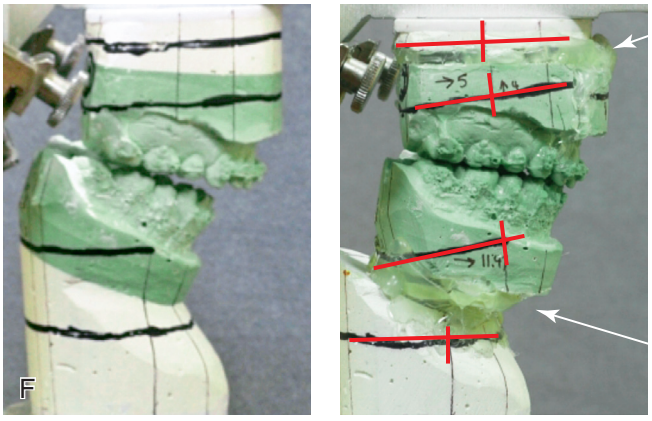
• **Figure 17-12, cont'd** The occlusion that was initially achieved and maintained at 5 weeks after surgery was as planned. Postoperative orthodontic appointments were complicated by the patient's attendance at an out-of-state college. At the time of appliance removal (i.e., 10 months after the operation), there was partial recurrence of the anterior open bite. The occlusion then remained stable at the 18-month postoperative interval. There was no popping, clicking, or pain in either temporomandibular joint. There was mild masticatory muscle discomfort, which was relieved with an effective night guard. An analysis of the postoperative cephalometric radiographs, which were taken at 1 month and 18 months after surgery, confirmed a stable SNB angle. The condyle-to-gonion measurement also remained stable. A review of the condyle morphology on the Panorex radiograph revealed no significant change. A review of the dental models (i.e., the surgical planning models and those from 16 months after surgery) suggested a degree of dental relapse in the form of recurrent curve of Spee issues in the maxilla and the mandible. The patient elected to undergo no further treatment. **C,** Oblique facial views before and after treatment. **D,** Profile views before and after treatment.

Continued

Prior to retreatment with 4 bicuspid extractions

Pre surgery

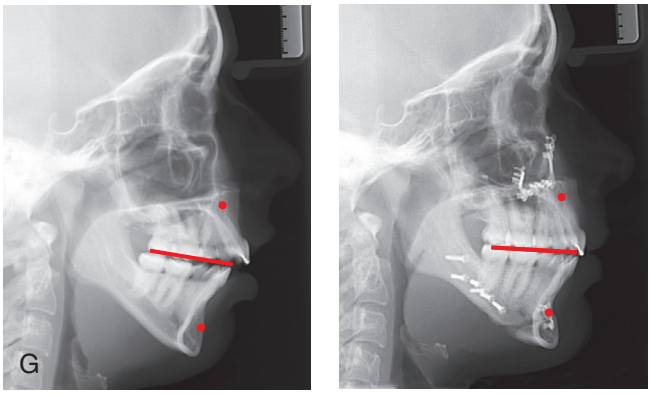
1½ years after treatment



Maxillary counter-clockwise rotation

Curve of Spee

Mandibular counter-clockwise rotation



• **Figure 17-12, cont'd E,** Occlusal views before retreatment, with orthodontics in progress, and at 16 months after surgery. **F,** Articulated dental casts that indicate analytic model planning. **G,** Lateral cephalometric radiographs before and 16 months after surgery.

• **Figure 17-13** A 25-year-old man presented with a long face anterior open-bite growth pattern. He had undergone four bicuspid extractions and a surgically assisted rapid palatal expansion procedure to reduce crowding and to expand the maxillary arch width at another institution. He was referred to this surgeon with residual maxillofacial problems, including jaw discrepancy, residual malocclusion, and chronic obstructed nasal breathing. The patient agreed to additional orthodontic mechanics and surgery, including Le Fort I osteotomy in two segments (vertical shortening, horizontal advancement, and arch narrowing); sagittal ramus osteotomies (horizontal advancement and counterclockwise rotation); septoplasty; the reduction of the inferior turbinates; and osseous genioplasty (vertical shortening and horizontal advancement). He is shown before and after the completion of his final phase of treatment. **A,** Frontal views in repose before and after retreatment. **B,** Frontal views with smile before and after retreatment.

Continued

• **Figure 17-13, cont'd C,** Oblique facial views before and after retreatment. **D,** Profile views before and after retreatment.

Prior to retreatment with 4 bicuspid extractions

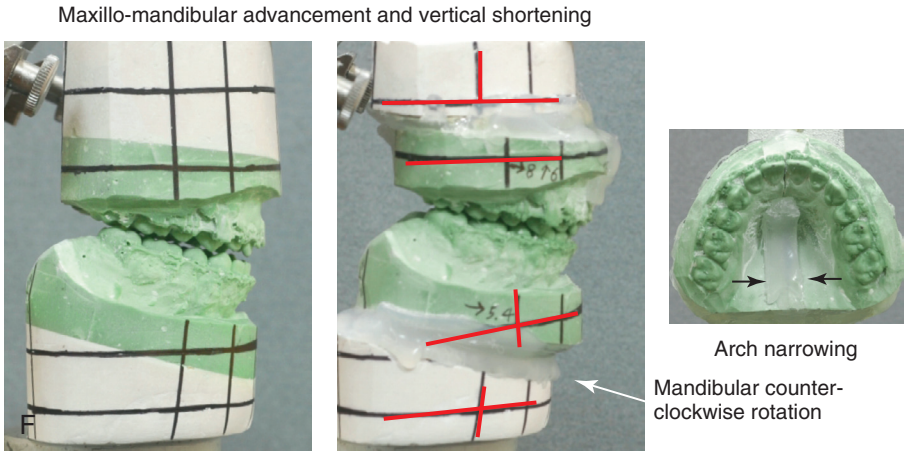
After SARPE

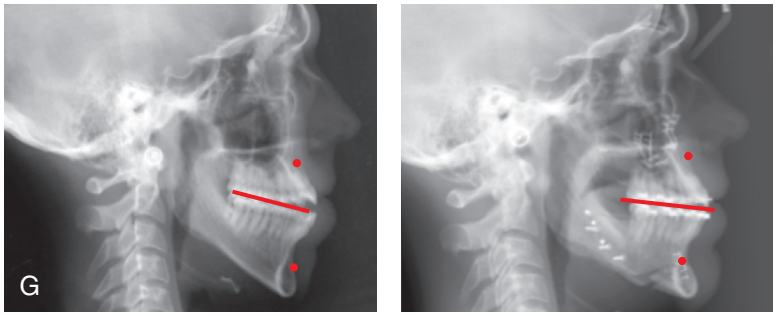
Prior to orthognathic surgery

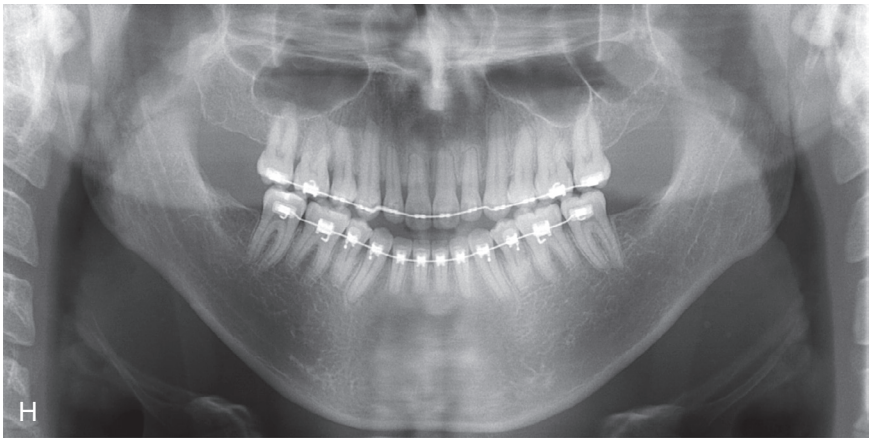
After treatment

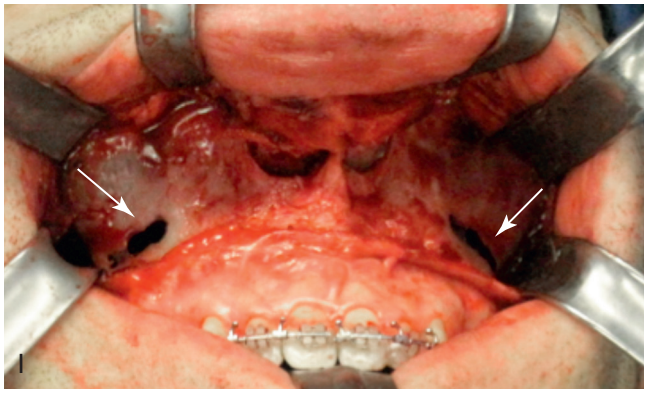
• **Figure 17-13, cont'd E,** Occlusal views after earlier orthodontic treatment including four bicuspid extractions, after surgically assisted rapid palatal expansion (SARPE), just before orthognathic surgery, and after retreatment.

Continued

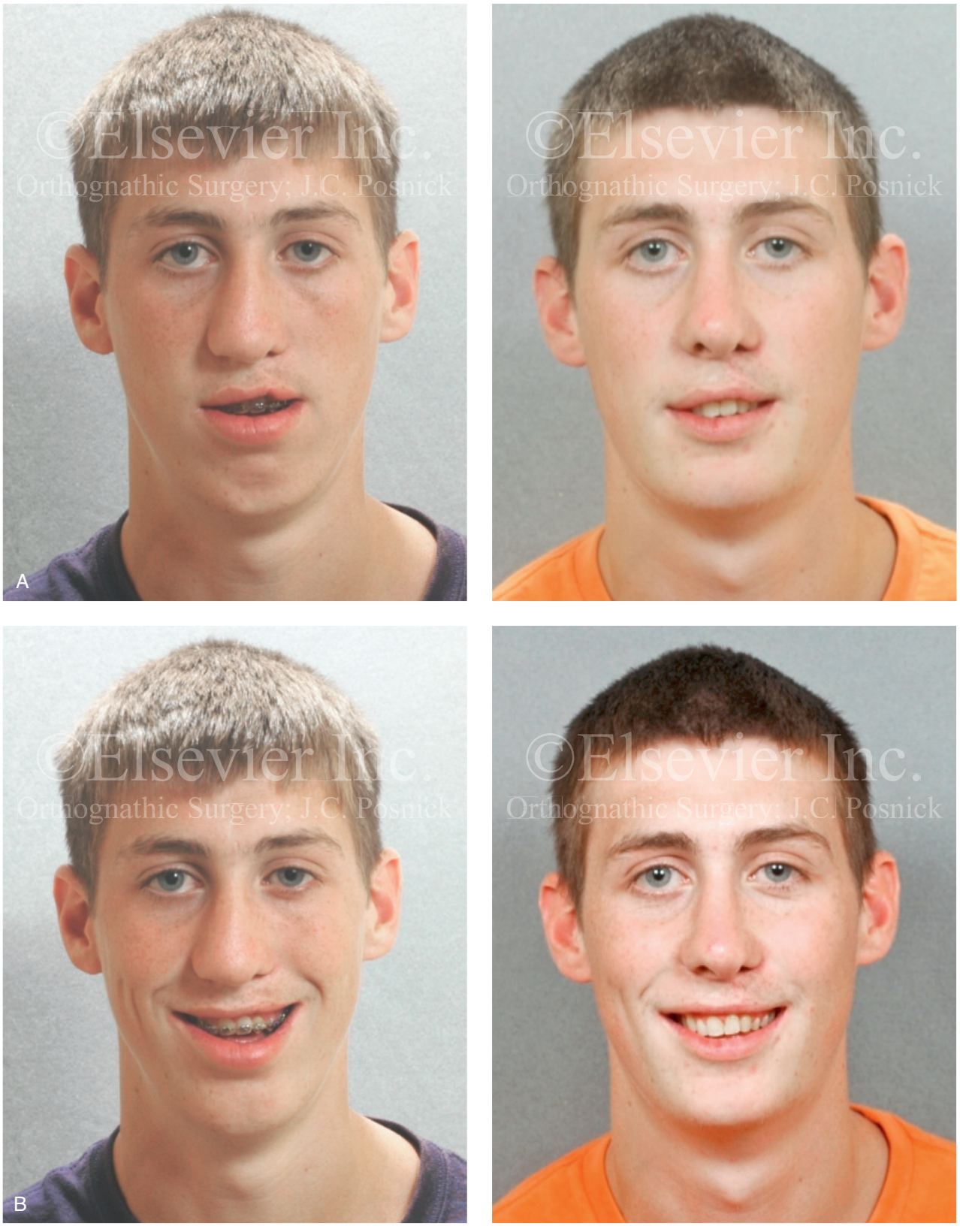








• **Figure 17-13, cont'd F,** Articulated dental casts that indicate analytic model planning. **G,** Lateral cephalometric views before and after retreatment. **H,** Panorex radiograph before retreatment. **I,** Intraoperative view of the anterior maxilla after the surgically assisted rapid palatal expansion (SARPE) procedure and just before Le Fort I osteotomy.

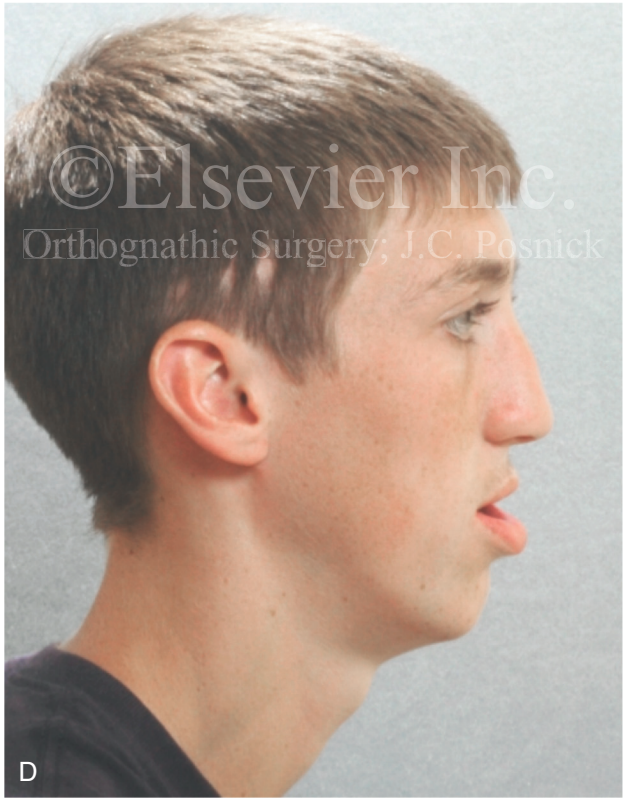


• **Figure 17-14** A young man born with a unilateral cleft lip and palate had been treated by other surgeons. His treatments had included primary cleft lip repair; primary cleft palate repair; two cleft lip revisions; three cleft palate revisions; the placement of a superiorly based pharyngeal flap; iliac grafting for a cleft defect; cleft rhinoplasty; and suboptimal Le Fort I osteotomy with an external RED distraction osteogenesis (DO) device. The patient was referred to this surgeon when he was 17 years old with residual cleft deformities, including the following:

- Nasopharyngeal stenosis with obstructive sleep apnea (apnea–hypopnea index of 11.4 events, with desaturation to 89%)
- Persistent oronasal fistula and alveolar cleft
- Residual cleft jaw deformity with maxillary hypoplasia and Class III malocclusion
- Chronic nasal obstruction as a result of septal deviation and enlarged inferior turbinates
- Residual cleft nasal deformity
- Cleft lip deformity with a lack of continuity of the orbicularis–oris muscle

A, Frontal views before and after reconstruction. **B,** Frontal views with smile before and after reconstruction.

Continued



• **Figure 17-14, cont'd** The patient agreed to a comprehensive orthodontic and surgical approach. During his operation, he underwent redo Le Fort I osteotomy in two segments (+7 mm horizontal advancement with interpositional iliac grafting, transverse widening, and oronasal fistula closure); bilateral sagittal split osteotomies of the mandible (horizontal advancement and counterclockwise rotation); osseous genioplasty (horizontal advancement and vertical shortening); redo septoplasty; redo inferior turbinate reduction; and recontouring of the nasal rims. An attended sleep study performed 1 year after the operation confirmed the complete resolution of obstructive sleep apnea. After successful healing, the patient underwent simultaneous cleft rhinoplasty, including rib cartilage (caudal strut) grafting; cleft lip scar revision, including the repair of the orbicularis oris muscle; and the revision of the pharyngeal flap to relieve stenosis and mucous trapping. He is shown before and 3 years after reconstruction. Note that the residual malocclusion is primarily the result of dental relapse of the teeth within the alveolar bone. This is based on review of dental models and radiographs. **C,** Oblique facial views before and after reconstruction. **D,** Profile views before and after reconstruction.

Pre retreatment ortho

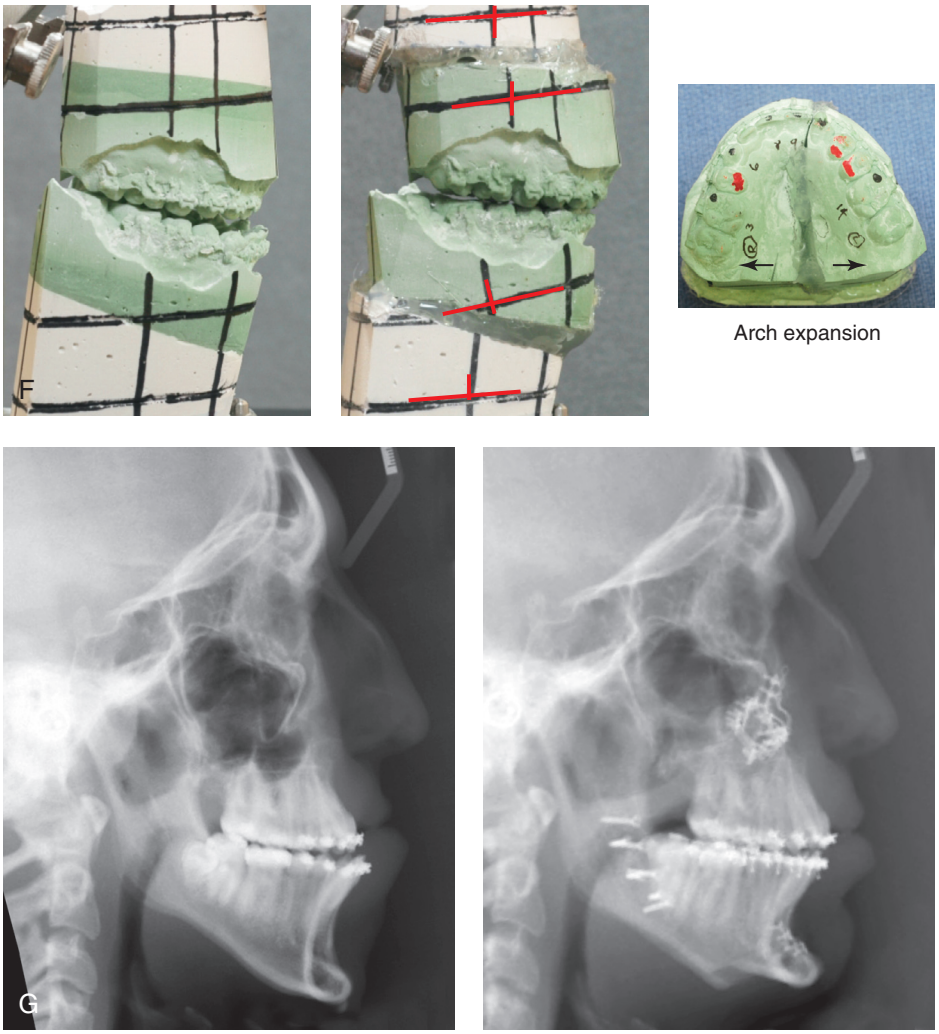
Pre retreatment surgery

5 weeks after surgery

3 years after surgery

• **Figure 17-14, cont'd E,** Occlusal views before retreatment, with orthodontics in progress, 5 weeks after redo jaw surgery, and 3 years after jaw reconstruction.

Continued



• **Figure 17-14, cont'd F,** Articulated dental casts that indicate analytic model planning. **G,** Lateral cephalometric views before and after retreatment.

Calvin Case, who was a prominent contemporary and outspoken critic of Angle, used contouring appliances and light rubber bands to move the teeth. *He argued that, although the arches could be expanded and the teeth aligned, in the long term, occlusal stability was not always satisfactory, and facial aesthetics could be compromised.*

By around 1915, Case had worked out most of the mechanical principles required for the successful management of orthodontic extraction sites. With the use of full-banded appliances, he was able to routinely retract the anterior teeth into the bicuspid extraction sites to correct the inclination of the anterior teeth and to eliminate crowding. Interestingly, only about 10% of his patients were treated with extractions.

Debate in dental circles regarding the issue of extractions continued between Angle's disciples and Case's followers throughout the 1920s. The Dewey–Case "Great Extraction Debate" took place in 1911. Angle's non-extraction/arch expansion approach continued to predominate with the everyday practitioner until after World War II. Angle's philosophy was widely accepted as a result of his well-written and popular textbook; his abilities as a teacher and lecturer; his consistent treatment philosophy; and his success as a diligent clinician.

By the late 1930s, when non-extraction treatment was used on all comers, relapse was a frequent observation that could no longer be ignored. After Angle's death, his student *Charles Tweed* began re-treating a number of Angle's patients who had experienced relapse. Tweed's retreatment approach included *four bicuspid extractions followed by the realignment of the remaining dentition.* He observed greater occlusal stability with this approach. By the mid-1940s, Tweed's well-documented series of cases involving four premolar extractions with orthodontic retraction caused a revolution in orthodontic thinking that led to widespread extraction therapy in everyday orthodontic practice. Interestingly, *Raymond Begg,* who was another of Angle's students and who was at this time halfway around the world in Australia, also *concluded that non-extraction treatment was frequently unstable.* Like Tweed, Begg modified the Angle-designed Edgewise appliance into what he called the "Begg appliance" for use with extraction therapy.

The acceptance of extraction therapy in orthodontics to stably position the teeth into alveolar housing coincided

with an appreciation that genetic variations—with disparities between tooth root volume and jaw size—are common among normal individuals and will differ among races (see Chapter 3). That a presenting jaw or dental discrepancy could benefit from extractions was simply an admission of a patient's individuality rather than the orthodontist's inability to solve all problems in the same way. *By the early 1960s, approximately 50% of American patients who were undergoing orthodontic treatment would experience the extraction of at least some teeth, most commonly the first premolars*.

Orthodontists have also used extractions as a means to achieve a neutralized occlusion in the presence of a skeletal discrepancy (i.e., a *camouflage approach*). This involves achieving an improved occlusion while accepting the jaw (facial) disharmony. Examples of this approach include upper bicuspid extractions to orthodontically treat mandibular retrognathism or lower bicuspid extractions to orthodontically manage Class III skeletal patterns. Interestingly, this orthodontic camouflage approach became common practice during the 20th century, despite the fact that, by the early 1900s, mandibular surgery had been performed for both Class II and Class III skeletal deformities. *In reality, corrective jaw surgery was rarely considered an option (except by a handful of orthodontists and surgeons) until the later decades of the 20th century* (see Chapter 2).

By the 1990s, overemphasis on four bicuspid extractions as part of routine orthodontic treatment began to wane in favor of a more discriminating approach. The rationale for selective extractions as part of a comprehensive surgical and orthodontic approach has now been confirmed to be effective for the removal of dental compensation. Extraction orthodontic therapy in combination with jaw osteotomies to correct the occlusion, for long-term periodontal health, to improve the upper airway, and to enhance facial aesthetics is now considered routine for many patients. The interproximal reduction of enamel as an alternative to extractions was popularized by Dr. J. Sheridan during the 1990s. In borderline cases, it has been found to provide the necessary space for incisor uprighting (see Chapters 5 and 6).

Research confirms that clinicians and the general public prefer a face with full midface projection and with prominent teeth and lips.[231,257,261] The "flat face" orthodontic standards of Tweed, which were typical during the 1950s and the 1960s, retracted rather than expanded (i.e., filled out) the face. In borderline cases, the patient's facial appearance is generally judged as being better without extractions. The decision to extract should be viewed in the light of attaining Andrews' Six Keys to Normal Occlusion and Six Elements of Orofacial Harmony (see Chapter 2). The primary goals are to achieve: (1) the placement of teeth solidly in the alveolar bone (2) ideal dental alignment within and between the arches and (3) the adequate position and projection of the maxillomandibular complex (i.e. facial harmony). If jaw surgery (with or without extractions) is required to achieve these objectives, it should be incorporated into the treatment plan from the outset for the benefit of the patient.

An alternative to the extraction of teeth for the relief of crowding in the maxilla is to expand the volume of the alveolar ridge. This may be accomplished via one of three techniques: (1) rapid palatal expansion (RPE) with the use of a transpalatal appliance if the palatal suture is open (i.e., 0.5 mm of widening per day); (2) surgically assisted rapid palatal expansion (SARPE) followed by transpalatal appliance widening (i.e., 0.5 mm of widening per day); (3) segmental maxillary osteotomies (i.e., Le Fort I down-fracture and full disimpaction) with immediate repositioning to correct the arch form. These three options are not equally appropriate for each individual patient. The level of success of each of these options will be dependent on the clinical circumstances.

Orthodontic Treatment of Specific Dentofacial Growth Patterns: Dental Stability and Relapse

Primary Mandibular Deficiency Growth Pattern with or without Maxillary Arch Constriction (Skeletal Class II)

Findings

For the patient who presents with mandibular deficiency, dental compensations often include crowding and flaring of the lower incisors with an accentuated curve of Spee. This is a natural biologic response to the eruption of a full complement of teeth within a small mandible. This is exacerbated by constant protrusive tongue activity in combination with limited lip forces within a small oral cavity. These dental compensations are inevitable when the combined size of the mandibular teeth is greater than the available alveolar bone volume within a hypoplastic lower jaw. The anterior teeth (i.e. dental roots) follow the path of least resistance and frequently protrude outside of the labial cortex. In this dysfunctional matrix, the lower incisor inclination—when viewed on a lateral cephalometric radiograph—is obtuse, and it often substantially exceeds the normal range (i.e. 90 to 95 degrees). The extent of incisor protrusion is indicative of the degree of mandibular hypoplasia, and the unequal forces of the tongue and lips. In approximately one third of cases, there is also a degree of maxillary arch-width deficiency that requires correction; this is described in more detail later in this chapter.

Orthodontic Approach in Conjunction with Orthognathic Surgery

In the presence of moderate to severe mandibular deficiency, the ideal positioning of the lower anterior teeth is best accomplished by first carrying out extractions to create space. This most often involves the removal of the lower first bicuspids. When less space is required and the occlusion will accommodate it, the extraction of a single lower incisor is an option to consider. In borderline cases and depending on the specific patient's dental anatomy, interproximal reduction of the anterior teeth may provide sufficient space. The orthodontist's ability to achieve lower incisor inclination of approximately 90 to 95 degrees will be a good indication of the overall relief of dental compensation within the mandibular arch.

The failure to remove these compensations limits the surgeon's ability to position the mandible for ideal facial proportions, and for the relief of airway obstruction (i.e., opening of the retroglossal space). An advancement genioplasty to further enhance aesthetics and lip function is also generally beneficial to manage microgenia (see Chapter 19). If the surgeon "over-advances" the chin as a "skeletal compensation" for the inadequate orthodontic removal of incisor flaring, then further facial disproportion (i.e., a deepening of the labiomental fold with an overly prominent pogonion) is likely to occur (see Chapters 37 and 40).

Another aspect of dental compensation frequently seen in the individual with mandibular deficiency is a deep bite and an accentuated curve of Spee. In this case, first bicuspid extractions are often beneficial. At the time of mandibular advancement with incomplete flattening of the curve of Spee, the occlusion may be placed in a "tripod" relationship. After surgery, the resulting mid-arch open bites are closed by extruding the bicuspids with vertical elastics. These mid-arch open bites are generally more efficiently managed after surgery, when the lower anterior facial height is increased and the mandible has been advanced. After surgery, the expanded intraoral volume no longer creates the same degree of anterior and lateral tongue forces that oppose the orthodontic correction of the curve of Spee and the closure of extraction sites. The reduction in bite force early after surgery also assists with the rapid leveling process.

If interarch-width discrepancies are present—as they are in approximately one third of cases—then simultaneous Le Fort I osteotomy with segmentation (i.e., into two or three segments) is generally carried out to widen and correct the maxillary arch form (see "Management of Transverse Maxillary Deficiency" later in this chapter). When only minimal upper arch width deficiency exists and no other reasons warrant maxillary osteotomy, mandibular narrowing to balance the arches can be considered. This may be accomplished by completing a vertical midline osteotomy between the mandibular central incisors. The use of this option for mandibular narrowing is usually a distant second choice (vs. maxillary expansion) for the resolution of interarch discrepancies. Adding width to the upper jaw is generally preferred for the airway and to enhance smile aesthetics (i.e., for the relief of dark buccal corridors).

Maxillary Deficiency with Relative Mandibular Excess Growth Pattern (Skeletal Class III)

Findings

In a patient with maxillary deficiency and relative mandibular excess, the normal dental compensations generally include maxillary incisor protrusion and upright or retroclined mandibular incisors (see Chapter 20).

Orthodontic Approach in Conjunction with Orthognathic Surgery

With this common dentofacial growth pattern, the extraction of the first bicuspids in the maxillary arch to uncrowd and reposition the incisors (approximately 120 degrees to the Frankfort plane or 102 to 104 degrees to the sella–nasion plane) should be considered. With only borderline crowding and limited incisor flaring the decision for maxillary extractions becomes more difficult. Also, a degree of maxillary incisor flaring may be aesthetically tolerated when planning bi-maxillary osteotomies, which incorporate clockwise rotation of the maxillo-mandibular complex to correct any A-point–B-point facial disharmony (see Chapter 12).

If interarch width discrepancies (posterior crossbites) require correction, then simultaneous segmentation of the down fractured maxilla (2 or 3 segments) is carried out (see "Management of Transverse Maxillary Deficiency" later in this chapter).

In the mandibular arch, the incisors are generally retroclined, and a reverse curve of Spee is often excessive. Mandibular extractions are only indicated if there is significant crowding in the lower arch with inadequate alveolar bone.

In the patient with a skeletal Class III growth pattern, the failure to orthodontically remove these specific dental compensations as part of the comprehensive orthodontic and surgical approach is likely to negatively affect long-term occlusal stability, periodontal health, and the achievement of preferred Euclidian facial proportions.

Long Face Growth Pattern: Maxillary Vertical Excess with Mandibular Deformity

Findings

A long face growth pattern classically includes maxillary deformity (vertical excess, transverse deficiency, and a lack of horizontal projection), mandibular deformity (high-angle retrognathia), and deformity of the chin (vertical excess and sagittal deficiency). Of note is that the mandibular deformity may also present as a Class III rather than the more common Class II growth pattern (see Chapters 4 and 21).

The biologic dental compensations in the maxillary arch are reflective of the skeletal transverse deficiency and the differential degrees of maxillary vertical alveolar excess (i.e., the anterior and posterior aspects of the maxilla). If there is marked dental crowding in the maxilla, then bicuspid extractions will be indicated. In the lower arch, depending on the extent of mandibular micrognathia and flaring of the incisors, limited space for the orthodontic retraction of the teeth may be present. If so, mandibular first bicuspid extractions will generally be indicated followed by orthodontic retraction and alignment.

Orthodontic Approach in Conjunction with Orthognathic Surgery

The preoperative orthodontic leveling of a moderate to severe curve of Spee in the maxilla is ideally performed with segmental arch mechanics. This is necessary to match the dental alignment with the extent of dysmorphic anterior and posterior alveolar bone. This is done in preparation for segmental maxillary osteotomies, which will then level the curve of Spee and adjust the arch width. This segmental orthodontic preparation places the teeth solidly in the available alveolar bone within each component (segment) of the

upper jaw. This avoids the extrusion of the anterior teeth and the buccal tipping of the posterior dentition. Given the usual degree of dysmorphic maxillary alveolar bone, attempts to level the dentition solely through orthodontic mechanics (i.e., via incisor extrusion and molar intrusion) are periodontally unsound; they may result in incisor root resorption, and they are prone to late dental relapse with recurrent malocclusion (see Fig. 17-9).

Surgical segmentation of the maxilla between the lateral incisors and the cuspids and then parasagittally through the hard palate is generally performed to achieve the desired arch width, the leveling of the curve of Spee, and the adjustment of incisor inclination. The canine and lateral incisor roots are naturally divergent, so there is no need (or advantage) for the orthodontist to create a space to "make the segmental surgery safe" (see Chapter 15). There is little risk of causing intraoperative injury within the periodontal ligament space or to the tooth roots or of creating periodontal pocketing when completing the interdental osteotomies in these locations.

To review, the orthodontic correction of the maxillary arch form with vertical mechanics (i.e., the extrusion of the incisors and molar intrusion) and dental expansion (i.e., molar tipping) in a patient with a long face anterior open-bite malocclusion is unstable. It is likely to be injurious to the periodontium, with labial bone loss and gingival recession in the molar region, and it is more likely to result in the blunting of the incisor root apices. In addition, attempting closure of the anterior open bite via the orthodontic extrusion of the incisors would accentuate the gummy smile, further flatten the appearance of the face in profile, and not address the lip incompetence.

Management of Transverse Maxillary Deficiency: Techniques and Relapse Patterns

Background

The orthodontic management of isolated transverse maxillary deficiency in children and adolescents with an open palatal suture by *rapid palatal expansion* (RPE) and conventional orthodontic techniques is generally possible, although stability can be troublesome.* In the non-growing individual, if standard orthodontic techniques are used for expansion beyond a few millimeters, they will produce either buccal tipping of the maxillary molars or lingual tipping of the mandibular molars with an unstable long-term occlusion and the potential for injury to the periodontium. The molars will likely relapse palatally, thereby causing clockwise rotation of the mandible with recurrent anterior open bite.

The literature confirms that a transverse maxillary deficiency that presents after palatal suture closure (i.e., an age of >14 years) should be managed surgically.* This can be done with either "surgical assistance" followed by rapid expansion using an in-place palatal appliance (the process known as *surgically assisted rapid palatal expansion* [SARPE]) and then orthodontic realignment or by *segmental Le Fort I maxillary osteotomies* in conjunction with standard orthodontics. The latter approach is carried out when clinically indicated as a one-stage procedure in combination with vertical, horizontal, and other needed surgical repositioning of the maxilla (these two approaches are discussed in more detail later in this chapter). The magnitude of segmental maxillary expansion that a surgeon is comfortable achieving is dependent on that clinician's experience and the unique aspects of the specific patient's palatal anatomy.

Proffit and colleagues reported that individuals who are seeking evaluation for orthognathic surgery have a 30% incidence of transverse jaw discrepancies.[252] True (absolute) transverse discrepancy exists when one or both of the posterior maxillary segments are in a crossbite when the casts are placed into a normal overjet and overbite position after the relief of dental compensation. This will tend to unmask arch-width discrepancies in patients with Class II malocclusions and correct a percentage of those in patients with Class III malocclusions. True transverse arch discrepancies are frequently seen in patients with 1) Class II retrognathic mandibles 2) long face Class II anterior open bites 3) those with previous thumb-sucking habits resulting in anterior open bite and 4) patients with cleft palates and maxillary deficiency.

Vanarsdall and colleagues have defined transverse skeletal discrepancies through measurements that are made on posteroanterior cephalometric radiographs.[314] The authors measured maxillary (skeletal) width from the *jugular point* on one side to the jugular point on the other side. They compared this measurement to the skeletal lower arch width measured from the *antegonial notch* on one side to the antegonial notch on the other side. From these two measurements, the determination of a *differential index* can be made. These researchers suggested that a preferred differential index should be approximately 20 mm in the adult Caucasian individual. Preadolescent patients will normally have a lower differential index. The authors believe that an adult with a differential of 30 mm has a significant transverse problem. According to Vanarsdall and colleagues, a differential index that varies by more than 5 mm from these values requires correction in the maxilla to establish a long-term stable occlusion and satisfactory periodontal health.[314] With the advent of three-dimensional cone beam computed tomography imaging, the more accurate measurement of the transverse maxillary width is likely available. Further research is required to confirm normal values for maxillary and mandibular arch width.

Gingival recession in any given individual is likely to have multifactorial causes. Tooth position within the alveolar

*References 6, 17, 23, 67, 70, 73, 96, 122-125, 133, 151, 168, 185, 199, 200, 268, 269, 277, 282, 283, 306, 322, 323, 337

*References 13, 21, 25, 31, 32, 42, 56, 59, 60, 62, 64-66, 167, 171, 176, 225, 235, 238, 252, 260, 284, 299, 303, 314

process is an important factor, especially in the patient who is biologically susceptible to recession (i.e., with limited attached gingiva; see Chapters 5 and 6). According to Vanarsdall and colleagues, the most common risk factors for the gingival recession of the maxillary posterior dentition include 1) friable attached gingiva 2) genetic factors 3) emotional stress 4) inadequate labial bone and 5) buccally tipped maxillary posterior teeth (i.e., dental compensation).[314] These factors will cause the periodontium to "receive" rather than "transmit" forces, with likely continued bone loss and gingival recession occurring over time.

Vanarsdall and colleagues suggest that periodontal susceptibility to breakdown has a relationship to the position of the teeth within the arch and to the relationship of the opposing teeth when they are in occlusion (see Chapters 5 and 6).[314] The initial experimental work of these researchers shows a decrease in the numbers of pathogenic bacteria around the teeth after the teeth have been aligned into a more natural position (i.e., to more closely agree with Andrews' Six Keys to Normal Occlusion). With good dental alignment, the numbers of harmful bacteria were brought below the threshold at which damage to the periodontal tissues frequently occurs. It is also believed by many clinicians that the achievement of maxillomandibular occlusal harmony (i.e., the correction of molar inclination and crossbites) tends to improve long-term dental and skeletal stability after orthognathic surgery (see Chapters 5 and 6).

History of SARPE and Segmental Osteotomies to Correct Maxillary Arch Deficiencies

The recorded history of transverse maxillary expansion dates back to the mid-1800s, when Angle reported the use of a jackscrew appliance in a 14-year-old girl to create space for a labially displaced canine.[9] This form of arch expansion was frequently used in children, adolescents, and adults until Hass reported that, after the age of 18 years, it was not possible to open the mid-palatal arch with appliance distraction forces because of the presence of bridging bone spicules across the suture and the increased rigidity of contiguous bones.[124] It is now generally accepted that RPE without "surgical assistance" is clinically successful only until the age of approximately 14 years and in the presence of open sutures. Attempting orthopedic expansion beyond this age is likely to result in the tipping and extrusion of teeth followed by dental relapse after the removal of the appliance.

The first recorded use of "surgical separation" of a fused mid-palatal suture for the management of transverse maxillary deficiency was by Brown.[49-51] Since that initial report, the clinical approach that includes the completion of maxillary osteotomies followed by orthodontic appliance expansion has continued to evolve. By the 1980s, it was recognized that a *full Le Fort I osteotomy but without down-fracture*—including the separation of the nasal septum from the maxilla and the separation of both pterygomaxillary sutures—in combination with a parasagittal palatal and interdental osteotomy (generally between the incisors) was required. The immediate activation of an in-place palatal expander in the operating room to verify the completion of the osteotomies is also required to confirm the reliability of postoperative skeletal expansion.

By the 1970s, the biologic safety of a *Le Fort I osteotomy in segments* and the ability to then three-dimensionally reconfigure the segments directly in the operating room without the need for post surgical orthodontic expansion was proven in experimental primate studies and through successful use in clinical practice (see Chapter 2). This led to questions about the value of SARPE as a first-stage procedure for the patient with a dentofacial deformity who also requires maxillary horizontal and vertical adjustments.

Interestingly, it is estimated that only a limited percentage of dentofacial deformity patients have a transverse interarch disharmony in isolation. Arch-width narrowing is generally associated with other maxillary discrepancies (e.g., vertical, horizontal, dental midline to facial midline, occlusal plane, and cant), and it is often seen in combination with deformities of the mandible. For the limited cases in which an isolated maxillary transverse deficiency does occur, the use of SARPE followed by orthodontic alignment makes intuitive sense.

For the patient to undergo a two-stage orthognathic surgical approach (i.e., first-stage SARPE followed by definitive orthognathic surgery 6 to 12 months later) for the management of a multivector maxillary deformity, significant value would have to be demonstrated. Variables to consider include the following: (1) the potential for intraoperative and postoperative complications (2) the risks of multiple rounds of general anesthesia and multiple recovery periods (3) the direct financial costs to the patient, to his or her family, and to the health care system (4) the necessary patient and family time commitment (5) the facial aesthetic effects and (6) the potential for relapse and the stability of the occlusion. Most clinicians agree that the single-stage approach (i.e., Le Fort I osteotomy in segments) has clear advantages when considering each of these variables, except for differences of opinion related to potential relapse and the stability of the occlusion. The patient described in Figure 17-13 could be considered a case that would cause one to favor a two-stage approach, because the end result is favorable. However, in retrospect, the family and the treating clinicians viewed this case as involving an inefficient approach to the correction of the individual's presenting dentofacial deformity. The two-stage approach required 1) two separate general anesthetics and recoveries 2) an extended and more elaborate course of orthodontics; and 3) an increased financial burden to the family in the form of additional surgeon and hospital fees. Also, The SARPE procedure resulted in a degree of root resorption, and the Le Fort I osteotomy (Stage II) was made more difficult by the earlier SARPE procedure (Stage I). The intraoperative view (see Figure 17-13, *F*) shows the resulting fenestrated maxillary walls after the SARPE procedure.

Complications of the SARPE Procedure: Review of the Literature

Although SARPE is a commonly considered procedure, the prevalence of postoperative complications and suboptimal results is not insignificant.[57,72,175,177,182,195,211,254,291,315,329] **Williams and colleagues** examined the prevalence of complications among patients undergoing SARPE.[329] This was a retrospective evaluation conducted on all individuals who underwent SARPE over a 4-year timeframe (2004 to 2008) at a single institution. The study sample (n = 120) included patients with a median age of 29.5 years (range, 22 to 39 years) and was comprised of 51.7% women. Postoperative complications were divided into two main categories: surgical and dental. Thirty-three patients (28%) developed surgical complications and 18 patients (15%) developed dental or periodontal problems. Asymmetric or inadequate expansion was the most frequent surgical complication, and it was found in 13.3% (16 out of 120 patients) of the study cohort. Dental or periodontal problems—including central incisor discoloration, gingival recession, and periodontal bone loss—occurred in 15% of the cohort. Two patients developed catastrophic bone defects with associated loss of the central incisors as a result of eccentric midline osteotomies followed by infections. The study confirms that major complications after SARPE are rare, whereas the incidences of asymmetric and inadequate expansion as well as dental or periodontal problems are not insignificant. These findings confirm that the SARPE procedure should not be considered as a risk-free alternative to segmental Le Fort I osteotomies.

Verlinden and colleagues reported on a large case series of individuals (n = 73 patients) undergoing SARPE with a bone-borne appliance.[315] Although most of the problems documented were related to hardware, six patients (8%) required additional surgery for the correction of an unstable occlusion or for asymmetric expansion. Among those study patients who were followed for more than 6 months, gingival recession, periodontal bone loss, a lack of interdental bone fill, and external apical root resorption were seen in more than one third of cases. **Seitz and colleagues** studied patients who were undergoing SARPE (n = 22) and found that asymmetric expansion was the most frequent surgical complication. In another review of SARPE complications, **Carmen and colleagues** reported a 28.5% incidence of significant gingival recession.[57]

Occlusal Stability and Relapse: Comparing SARPE versus Segmental Le Fort I Osteotomy

When it comes to a single-stage procedure versus a two-stage approach to the management of a complex maxillary deformity, the key aspect of occlusal stability and relapse has been reviewed in the literature and argued by clinical proponents on each side. Shortcomings in the study design of most published research add to the lack of clarity. Most studies have involved only dental casts or direct measurements of dental arch dimensions. Without the use of posteroanterior (PA) cephalograms, skeletal change cannot be differentiated from tooth movement. Another shortcoming is that most studies measure stability from the end of orthodontic treatment rather than from the point of maximum expansion. The clinical practice of over-expanding the arch to end up with a satisfactory occlusion at the end of treatment results in the underestimation of the actual relapse. With limited data points, most studies cannot differentiate skeletal relapse components from dental ones. One exception is the study by **Byloff and Mossaz,** which overcame these limitations by using pre-expansion and postexpansion PA cephalograms and dental casts as data points.[56] After SARPE, the authors observed a mean maximum expansion at the first molar of 8.7 mm, with an average of 36% (3.1 mm) relapse at the time of debanding. Only 1.3 mm (23%) of this relapse was felt to be skeletal, with the rest (77%) being dental. **Berger and colleagues** also used an improved study design and reported an average of 2.49 mm (48%) of skeletal expansion in combination with 2.4 mm (52%) of dental expansion for their patients when using a SARPE approach.[31] Two published meta studies, which were carried out in 2005 by **Koudstaal and colleagues**[167] and in 2006 by **Lagravere and colleagues,**[176] both concluded that, until that time, no good evidence existed regarding the amount of relapse after SARPE.

Chamberland and Proffit studied this controversy, and, in 2008, they published their initial research.[59] They continued to follow their patients for the long term, and they also added numbers to the study group (N = 38). Their study has been recognized for adding significant clarity to this important subject and is therefore reviewed in the next section of this chapter.

Skeletal Stability and Relapse of SARPE: Review of a Benchmark Study

A prospective observational study was carried out by **Chamberland and Proffit** to review the SARPE outcomes in 38 consecutive patients between the ages of 15 and 54 years, with an observation period that extended to 2 years after the completion of treatment.[60] The data collected by these authors included measurements taken from both dental casts and PA cephalograms at the following specific time intervals:

- Before SARPE or any active orthodontic alignment
- After SARPE and at the completion of orthodontic expansion
- At the removal of the orthodontic expander (i.e., approximately 6 months after SARPE and the completion of the orthodontic goals)
- Before the final orthognathic procedure (if any was planned)
- After the final orthognathic surgery and at the end of orthodontic treatment
- 2 years after the completion of treatment

All study patients had a transverse discrepancy of 5 mm or more and were beyond the level of maturity at which palatal expansion without surgery would be possible. The surgical technique involved all bone cuts required for a Le Fort I osteotomy, including the separation of the pterygoid junctions, the nasal septum, and the mid-palatal suture as well as an osteotomy between the central incisors. During surgery, the expansion device was activated to achieve a 1- to 1.5-mm separation of the maxillary central incisors. After latency (7 days), the device was activated twice daily (0.5 mm/day). The patients were monitored twice weekly until the planned expansion was achieved 12 to 20 days later. All patients were expanded 2 mm beyond the desired final position. The expansion device was kept in place for approximately 6 months after the completion of orthodontic goals.

Interestingly, the research data confirm that almost the entire measured relapse was dental rather than skeletal. At 49 months after the SARPE procedure, the maintained expansion at the first molars had relapsed to approach the skeletal expansion (i.e., 4.6 mm total molar expansion [dental and skeletal] as compared with 3.6 mm skeletal expansion). The skeletal expansion that was measured at both the jugula and the nasal cavity was statistically stable.

All patients were maximally expanded 2 mm beyond the desired final position on the basis of the researchers' assumptions that a degree of relapse was inevitable. This overcorrection ensured that, despite a measured dental relapse of 30%, none of the patients showed posterior crossbite at the completion of treatment. One can conclude that, despite retention attempts, over the long term the teeth drift back into the basal bone (see Chapters 5 and 6).

The authors also compared their relapse and stability results with other published SARPE studies. Their measured 30% relapse rate was found to be less than the 36% relapse reported by Byloff and Mossaz.[56] Alternatively, their 30% documented relapse rate was higher than that reported by either Berger and colleagues[31] or Pogrel and colleagues.[238] As previously stated, most SARPE studies—including these two—do not report relapse from a starting point of maximal expansion. However, not doing so will underestimate the level of actual relapse and may explain the differences reported.

Chamberland and Profit showed that, when a SARPE procedure is carried out with the use of optimal surgical and orthodontic techniques, maxillary expansion that is about 47% skeletal and 53% dental can be expected.[59,60] The skeletal expansion that occurred with SARPE followed by strict orthodontic maintenance mechanics for approximately 5 months proved to be statistically stable. The overall relapse rate (30%) was almost totally attributable to lingual retraction (dental relapse) of the posterior teeth. As stated, the authors recommend a 2-mm excess expansion in anticipation of dental relapse. For example, in the case of a desired 8-mm first molar expansion, 10 mm of surgical expansion at the molars is recommended. The researchers documented the advantage of compensating for the inevitable buccal tipping of the posterior maxillary teeth during the expansion phase followed by an inevitable lingual dental relapse after the device is removed. Approximately 25% of their patients had more than 3 mm of dental relapse, whereas 42% had between 1 mm and 3 mm. A full 66% of patients experienced more than 2 mm of dental relapse over the long term (i.e., 2 years after debonding).

Stability and Relapse: Comparing SARPE in Adults versus RPE in Prepubertal Children

The literature confirms that, in the prepubertal child or the adolescent with an open palatal suture, a loss of about one third of the maximum expansion occurs across the first molars after non-surgical RPE. PA cephalograms performed for prepubertal patients with palatal implant markers who underwent RPE demonstrate that approximately 50% of the expansion achieved is skeletal, with the remainder being dental. This can be compared to Chamberland and Proffit's adult SARPE data.[59,60] Those authors demonstrated a mean of 3.47 mm of skeletal expansion and 2.25 mm of dental expansion (i.e., 5.12 mm of total expansion) at the first molars. They then documented a 30% relapse, the majority of which was dental. Therefore, a comparable 30% overall relapse in both the SARPE (adult) group and the RPE (prepubertal) group was found to occur. Interestingly, in the SARPE group, the maintenance of skeletal expansion was greater than that achieved in the RPE group.

Stability and Relapse in Adults: Comparing SARPE versus Segmental Le Fort I Osteotomies

The best data regarding stability after transverse expansion with segmental Le Fort I osteotomies were reported for 32 patients by Phillips and colleagues.[215] Chamberland and Proffit compared the segmental Le Fort I osteotomy group of Phillips and colleagues to their SARPE study patients.[59,60] They used a subgroup of Phillips and colleagues' patients who had undergone levels of maxillary expansion that were equivalent to those of their own SARPE patients. A comparison of the findings demonstrated the following: 1) the mean relapse across the first molars was greater for the Le Fort I group, but the difference was not statistically significant; and 2) the mean relapse across the canines was greater for the SARPE group and was statistically significant.

With reference to the SARPE (two-stage) approach versus the Le Fort I segmental (single-stage) approach for the correction of complex maxillary deformities (i.e. combination of arch-width, vertical, and horizontal deformities), the author's conclude:[59,60]

- Current data do not support the conclusion that SARPE procedures produce more stable expansion than Le Fort I segmental osteotomies.
- Published data regarding both PA cephalograms and measurements of dental models confirm the potential for postoperative relapse that is primarily dental rather than skeletal after transverse maxillary

expansion (i.e., RPE, SARPE, or Le Fort I segmental osteotomies).

- Relapse after the arch width initially achieved by SARPE is comparable to that of other expansion procedures (i.e., segmental Le Fort I osteotomy in adults and RPE in children).
- Published data show that, with properly executed SARPE and a strict maintenance protocol for approximately 6 months, the relapse is almost entirely dental (i.e., 33% dental relapse). At the end of treatment, there is, on average, a net expansion of 67% of the total change.
- With non-surgical expansion carried out in growing individuals (i.e., RPE), the expectation is that only 50% of the total change will be skeletal. No data from sequential PA cephalograms exists for Le Fort I segmental expansion.
- Given the similarity of relapse in intermolar width between SARPE and Le Fort I segmental techniques, routine overcorrection and strict maintenance for 6 months after surgery should be considered part of the protocol.
- When surgery to reposition the maxilla vertically or anteroposteriorly is also required, the routine performance of a preliminary SARPE procedure followed later by a Le Fort I osteotomy with the intention of obtaining better transverse stability does not appear to be warranted.
- According to published literature, the decision to use a two-stage surgical approach (i.e. first stage SARPE) versus a single-stage Le Fort I segmental surgical approach should not be based on the expected stability of the transverse expansion. Instead, it should be based on the risk and morbidity of two surgeries versus the risk and morbidity of a one-stage multisegmented Le Fort I osteotomy for arch expansion and the other necessary vertical or anteroposterior changes required for the correction of the individual's specific dentofacial deformity.
- Clinicians should also take into account that the SARPE procedure will result in areas of fibrous union and defects at the maxillary osteotomy sites. If a Le Fort I is later required to complete the reconstruction, the previous SARPE procedure may add to the complexity of the operation (see Fig. 17-13).

Conclusions

There are inherent challenges to both the orthodontic movement of teeth within the alveolar process and the surgical repositioning of the jaws within the facial soft-tissue envelope. Such challenges include the orthodontist's treatment philosophy; the mechanics used to reposition the teeth; the surgeon's treatment philosophy; and the techniques used to complete the osteotomies and secure them. Other important factors include the initial surgical healing requirements, which take into account patient-specific health-related factors, as well as the difficulties associated with the long-term maintenance of the mechanically relocated teeth, jaws, and jaw segments.

Despite the potential for skeletal and dental relapse, when sound biomechanical and aesthetic principles are followed, the frequent patterns of dentofacial deformity and their associated malocclusions can be safely and reliably corrected and maintained in most cases.

References

1. Abeloos J, De Clercq C, Neyt L: Skeletal stability following miniplate fixation after bilateral sagittal split osteotomy for mandibular advancement. *J Oral Maxillofac Surg* 51:366–369, 1993.
2. Ackerman JL, Proffit WR: Communication in orthodontic treatment planning: Bioethical and informed consent issues. *Angle Orthod* 65:253–262, 1995.
3. Adlam DM, Yau CK, Banks P: A retrospective study of the stability of midface osteotomies in cleft lip and palate patients. *Br J Oral Maxillofac Surg* 27:265–276, 1989.
4. Ahmed MMS: Long-term stability of anterior segmental maxillary osteotomy. *Int J Adult Orthodon Orthognath Surg* 14:297, 1999.
5. Akhtar S, Tuinzing DB: Unfavourable splits in sagittal split osteotomy. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 87:267–268, 1999.
6. Akkaya S, Lorenzon S, Uçem TT: A comparison of sagittal and vertical effects between bonded rapid and slow maxillary expansion procedures. *Eur J Orthod* 21:175–180, 1999.
7. Al Yami EA, Kuijpers-Jagtman AM, Van't Hof MA: Stability of orthodontic treatment outcome: Follow-up until 10 years post-retention. *Am J Orthod Dentofacial Orthop* 115:300–304, 1999.
8. Anavi Y: Condylar positioning devices for orthognathic surgery. Are they necessary? [discussion]. *J Oral Maxillofac Surg* 52:552, 1994.
9. Angell EC: Treatment of irregularities of the permanent or adult teeth. *Dent Cosmos* 540–544, 1860.
10. Angle EH: *Treatment of malocclusion of the teeth and fractures of the mandible: Angle's system*, Philadelphia, 1900, White Dental Manufacturing, p 16.
11. Araujo A, Schendel SA, Wolford LM, Epker BN: Total maxillary advancement with and without bone grafting. *J Oral Surg* 36:849–858, 1978.
12. Arnett GW: A redefinition of bilateral sagittal osteotomy (BSO) advancement relapse. *Am J Orthod Dentofacial Orthop* 104:506, 1993.
13. Arpornmaeklong P, Heggie AA, Shand JM: A comparison of the stability of single-piece and segmental Le Fort I maxillary advancements. *J Craniofac Surg* 14:3, 2003.
14. Arpornmaeklong P, Shand JM, Heggie AA: Stability of combined Le Fort I maxillary advancement and mandibular reduction. *Aust Orthod J* 19:57, 2003.
15. Ayliffe PR, Banks P, Martin IC: Stability of the Le Fort I osteotomy in patients with cleft lip and palate. *Int J Oral Maxillofac Surg* 24:201–207, 1995.
16. Ayoub AF, Trotman CA, Stirrups DR, Wilmot JJ: Stability of bimaxillary osteotomy

following surgical correction of class II skeletal deformities: A two-centre study. *Br J Oral Maxillofac Surg* 35:107–115, 1997.
17. Baccetti T, Franchi L, Cameron CG, McNamara JA, Jr: Treatment timing for rapid maxillary expansion. *Angle Orthod* 71:343–350, 2001.
18. Bailey LJ, Duong HL, Proffit WR: Surgical Class III treatment: Long-term stability and patient perceptions of treatment outcome. *Int J Adult Orthodon Orthognath Surg* 13:35, 1998.
19. Bailey LJ, Phillips C, Proffit WR, Turvey TA: Stability following superior repositioning of the maxilla by Le Fort I osteotomy: Five-year follow-up. *Int J Adult Orthodon Orthognath Surg* 9:163–173, 1994.
20. Bailey LJ, Proffit WR, White RP: Trends in surgical treatment of Class III skeletal relationships. *Int J Adult Orthodon Orthognath Surg* 10:108–118, 1995.
21. Bailey LJ, White RP, Proffit WR, Turvey TA: Segmental Le Fort I osteotomy for management of transverse maxillary deficiency. *J Oral Maxillofac Surg* 55:728–731, 1997.
22. Baker DL, Stoelinga PJW, Blijdorp PA, et al: Long-term stability after inferior maxillary repositioning by miniplate fixation. *Int J Oral Maxillofac Surg* 21:320, 1992.
23. Basciftci FA, Karaman AI: Effects of a modified acrylic bonded rapid maxillary expansion appliance and vertical chin cap on dentofacial structures. *Angle Orthod* 72:61–71, 2002.
24. Baumrind S, Korn EL, Ben-Bassat Y, West EE: Quantitation of maxillary remodeling: 2. Masking of remodeling effects when an "anatomical" method of superimposition is used in the absence of metallic implant. *Am J Orthod Dentofacial Orthop* 91:463–474, 1987.
25. Bays RA, Greco JM: Surgically assisted rapid palatal expansion: An outpatient technique with long-term stability. *J Oral Maxillofac Surg* 50:110, 1992.
26. Behrents RG: *A treatise on the continuum of growth in the aging craniofacial skeleton [PhD thesis]*, Ann Arbor, Mich, 1985, University of Michigan Center for Human Growth and Development.
27. Behrents RG: An atlas of growth in the aging craniofacial skeleton. In McNamara JA, editor: Monograph 18, Craniofacial Growth Series, Vol 18, Ann Arbor, Mich, 1985, University of Michigan.
28. Behrents RG: Growth in the aging craniofacial skeleton. In McNamara JA, editor: Monograph 17, Craniofacial Growth Serie, Vol 17, Ann Arbor, Mich, 1985, University of Michigan.
29. Bell WH, Scheidman GB: Correction of vertical maxillary deficiency: Stability and soft tissue changes. *J Oral Surg* 39:666–670, 1981.
30. Bench RW: Growth of the cervical vertebrae as related to tongue, face, and dentine behavior. *Am J Orthod* 49:183–214, 1963.
31. Berger JL, Pangrazio-Kulbersh V, Borgula T, et al: Stability of orthopedic and surgically assisted rapid palatal expansion over time. *Am J Orthod Dentofacial Orthop* 114:638, 1998.
32. Betts NJ, Vanarsdall RL, Barber HD, et al: Diagnosis and treatment of transverse maxillary deficiency. *Int J Adult Orthodon Orthognath Surg* 10:75–96, 1995.
33. Björk A: Facial growth in man studied with the aid of metallic implants. *Acta Odontol Scand* 13:9–34, 1955.
34. Björk A: Prediction of mandibular growth rotation. *Am J Orthod* 55:585–599, 1969.
35. Björk A, Skieller V: Facial development and tooth eruption: An implant study at the age of puberty. *Am J Orthod* 62:339–383, 1972.
36. Björk A, Skieller V: Growth in width of the maxilla studied by the implant method. *Scand J Plast Reconstr Surg* 8:26, 1977.
37. Björk A, Skieller V: Growth of the maxilla in three dimensions as revealed radiographically by the implant method. *Br J Orthod* 4:53–64, 1977.
38. Björk A, Skieller V: Normal and abnormal growth of the mandible: A synthesis of longitudinal cephalometric implant studies over a period of 25 years. *Eur J Orthod* 5:1–46, 1983.
39. Blomqvist JE, Ahlborg G, Isaksson S, et al: A comparison of skeletal stability after mandibular advancement and use of two rigid internal fixation techniques. *J Oral Maxillofac Surg* 55:568–573, 1997.
40. Blomqvist JE, Ahlborg G, Isaksson S, Svartz K: A comparison of skeletal stability after mandibular advancement and use of two rigid internal fixation techniques [discussion]. *J Oral Maxillofac Surg* 55:574–575, 1997.
41. Blomqvist JE, Isaksson S: Skeletal stability after mandibular advancement: A comparison of two rigid internal fixation techniques. *J Oral Maxillofac Surg* 52:1133–1137, 1994.
42. Bloomquist DS: The stability of segmentalized Le Fort I osteotomies with miniplate fixation in patients with maxillary hypoplasia [discussion]. *J Oral Maxillofac Surg* 53:1412, 1995.
43. Borstlap WA, Stoelinga PJ, Hoppenreijs TJ, et al: Stabilization of sagittal split advancement osteotomies with miniplates: A prospective, multicentre study with two-year follow-up. Part II. Radiographic parameters. *Int J Oral Maxillofac Surg* 33:535, 2004.
44. Bouwman JP, Husak A, Putnam GD, et al: Screw fixation following bilateral sagittal ramus osteotomy for mandibular advancement: Complications in 700 consecutive cases. *Br J Oral Maxillofac Surg* 33:231–234, 1995.
45. Bouwman JPB, Tuinzing DB, Kostense PJ, et al: The value of long-term follow-up of mandibular advancement surgery in patients with a low to normal mandibular plane angle. *Mund Kiefer Gesichtschir* 1:311, 1997.
46. Broadbent BH, Sr, Broadbent BH, Jr, Golden WH: *Bolton standards of dentofacial developmental growth*, St Louis, 1976, CV Mosby Company.
47. Brodie AG: On the growth pattern of the human head from third month to the eighth year of life. *Am J Anat* 68:209–262, 1941.
48. Brodie AG: Late growth changes in the human face. *Angle Orthod* 23:146–157, 1953.
49. Brown GVI: The application of orthodontia principles to the prevention of nasal disease. *Dent Cosmos* 45:765–775, 1903
50. Brown GVI: The surgical and therapeutic aspect of maxillary readjustment with special reference to nasal stenosis, hare lip, cleft palate and speech. *Dent Cosmos* 51:7–17, 1909
51. Brown GVI: *The surgery of oral and facial diseases and malformations*, ed 4, London, 1938, Kimpton.
52. Buijs GJ, Van Der Houwen EB, Stegenga B, et al: Mechanical strength and stiffness of biodegradable and titanium osteofixation systems. *J Oral Maxillofac Surg* 65:2148, 2007.
53. Buijs GJ, van der Houwen EB, Stegenga B, et al: Torsion strength of biodegradable and titanium screws: A comparison. *J Oral Maxillofac Surg* 65:2142, 2007.
54. Burstone CJ: Lip posture and its significance in treatment planning. *Am J Orthod* 53:262–284, 1967.
55. Busby BR, Bailey LJ, Proffit WR, et al: Long-term stability of class III treatment: A study of 5-year postsurgical results. *Int J Adult Orthodon Orthognath Surg* 17:159, 2002.
56. Byloff FK, Mossaz CF: Skeletal and dental changes following surgically assisted rapid palatal expansion. *Eur J Orthod* 7:37, 1992.
57. Carmen M, Marcella P, Giuseppe C, et al: Periodontal evaluation in patients undergoing maxillary expansion. *J Craniofac Surg* 11:491, 2000
58. Caskey RT, Turpin DL, Bloomquist DS: Stability of mandibular lengthening using bicortical screw fixation. *Am J Orthod Dentofacial Orthop* 96:320, 1989.
59. Chamberland S, Proffit WR: Closer look at the stability of surgically assisted rapid palatal expansion. *J Oral Maxillofac Surg* 66:1895–1900, 2008.
60. Chamberland S, Proffit WR: Short-term and long-term stability of surgically assisted rapid palatal expansion revisited. *Am J Orthod Dentofacial Orthop* 139:815–822, 2011.
61. Chang YL, Moon SC: Cephalometric evaluation of the anterior open bite treatment. *Am J Orthod Dentofacial Orthop* 115:29–38, 1999.
62. Chen YR, Yeow V: Multiple-segment osteotomy in maxillofacial surgery. *Plast Reconstr Surg* 104:381, 1999.
63. Cheung LK, Samman N, Hui E, Tideman H: The 3-dimensional stability of maxillary osteotomies in cleft palate patients with

residual alveolar clefts. *Br J Oral Maxillofac Surg* 32:6–12, 1994.
64. Chow J, Hagg U, Tideman H: The stability of segmentalized Le Fort I osteotomies with miniplate fixation in patients with maxillary hypoplasia. *J Oral Maxillofac Surg* 53:1407, 1995.
65. Chung C-H, Goldman AM: Dental tipping and rotation immediately after surgically assisted rapid palatal expansion. *Eur J Orthod* 25:353–358, 2003.
66. Chung C-H, Woo A, Zagarinsky J, et al: Maxillary sagittal and vertical displacement induced by surgically assisted rapid palatal expansion. *Am J Orthod Dentofacial Orthop* 120:144–148, 2001.
67. Ciambotti C, Ngan P, Durkee M, et al: A comparison of dental and dentoalveolar changes between rapid palatal expansion and nickel-titanium palatal expansion appliances. *Am J Orthod Dentofacial Orthop* 119:11–20, 2001.
68. Coben SE: Growth and Class II treatment. *Am J Orthod* 52:5–26, 1966.
69. Costa F, Robiony M, Politi M: Stability of sagittal split ramus osteotomy used to correct Class III malocclusion: Review of the literature. *Int J Adult Orthodon Orthognath Surg* 16:121, 2001.
70. Cross DL, McDonald JP: Effect of rapid maxillary expansion on skeletal, dental, and nasal structures: A postero-anterior cephalometric study. *Eur J Orthod* 22:519–528, 2000.
71. Cunningham SJ, Crean SJ, Hunt NP, et al: Preparation, perceptions and problems: A long-term follow-up study of orthognathic surgery. *Int J Adult Orthodon Orthognath Surg* 11:41–47,1996.
72. Cureton SL, Cuenin M: Surgically assisted rapid palatal expansion: Orthodontic preparation for clinical success. *Am J Orthod Dentofacial Orthop* 116:46, 1999.
73. Davis WM, Kronman JH: Anatomical changes induced by splitting the midpalatal suture. *Angle Orthod* 39:126–132, 1969.
74. Dolce C, Hatch JP, Van Sickels JE, et al: Rigid versus wire fixation for mandibular advancement: Skeletal and dental changes after 5 years. *Am J Orthod Dentofacial Orthop* 121:610, 2002.
75. Dolce C, Van Sickels JE, Bays RA, Rugh JD: Skeletal stability after mandibular advancement with rigid versus wire fixation. *J Oral Maxillofac Surg* 58:1219–1227, 2000; discussion 1227–1228, 2000.
76. Douma E, Kuftinec MM, Moshiri F: A comparative study of stability after mandibular advancement surgery. *Am J Orthod Dentofacial Orthop* 100:141, 1991.
77. Dowling P, Espeland L, Krogstad O, et al: Duration of orthodontic treatment involving orthognathic surgery. *Int J Adult Orthodon Orthognath Surg* 14:146, 1999.
78. Downs WB: Variations in facial relationships: Their significance in treatment and prognosis. *Am J Orthod Dentofacial Orthop* 34:812, 1948.
79. Drommer R, Luhr HG: The stabilization of osteotomized maxillary segments with Luhr miniplates in secondary cleft surgery. *J Maxillofac Surg* 9:166–169, 1981.
80. Edler RJ: Problems in the orthodontic management of orthognathic cases. *Eur J Orthod* 12:420, 1990.
81. Edwards CB, Marshall SD, Qian F, et al: Longitudinal study of facial skeletal growth completion in 3 dimensions. *Am J Orthod Dentofacial Orthop* 132:762, 2007.
82. Egbert M, Hepworth B, Myall R, et al: Stability of Le Fort I osteotomy with maxillary advancement: A comparison of combined wire fixation and rigid fixation. *J Oral Maxillofac Surg* 53:243, 1995.
83. Eggert JH, Götzfried HF, Steinhäuser EW: Relapse potential of maxillary osteotomy in lip, jaw and palatal clefts [German]. *Osterr Z Stomatol* 80:148–156, 1983.
84. Ellis E, III: Condylar positioning devices for orthognathic surgery: Are they necessary? *J Oral Maxillofac Surg* 52:536, 1994.
85. Ellis E, III, Carlson DS: Stability two years after mandibular advancement with and without suprahyoid myotomy: An experimental study. *J Oral Maxillofac Surg* 41:426, 1983.
86. Ellis E, III, Carlson DS, Billups J: Osseous healing of the sagittal ramus osteotomy: A histologic comparison of rigid and nonrigid fixation in *Macaca mulatta*. *J Oral Maxillofac Surg* 50:718, 1992.
87. Ellis E, III, Carlson DS, Frydenlund S: Stability of midface augmentation: An experimental study of musculoskeletal interaction and fixation methods. *J Oral Maxillofac Surg* 47:1062, 1989.
88. Ellis E, III, Esmail N: Malocclusions resulting from loss of fixation after sagittal split ramus osteotomies. *J Oral Maxillofac Surg* 67:2528, 2009.
89. Ellis E, III, Gallo WJ: Relapse following mandibular movement with dental plus skeletal maxillomandibular fixation. *J Oral Maxillofac Surg* 44:509, 1986.
90. Ellis E, III, Hinton RJ: Histologic examination of the temporomandibular joint after mandibular advancement with and without rigid fixation: An experimental investigation in adult *Macaca mulatta*. *J Oral Maxillofac Surg* 49:1316, 1991.
91. Ellis E, III, Reynolds S, Carlson DS: Stability of the mandible following advancement: A comparison of three postsurgical fixation techniques, *Am J Orthod Dentofacial Orthop* 94:38, 1988.
92. Enlow DH: *Facial growth*, ed 3, Pennsylvania, London, 1990, Saunders.
93. Enlow DH, Kuroda T, Lewis AB: Intrinsic craniofacial compensations. *Angle Orthod* 41:271–285, 1971.
94. Enlow DH, Moyers R, Hunter W, McNamara J: A procedure for the analysis of intrinsic facial form and growth. *Am J Orthod* 56:6–23, 1969.
95. Epker BN: Superior surgical repositioning of the maxilla: Long term results. *J Oral Maxillofac Surg* 9:237–246, 1981.
96. Epker BN, Fish LC: Transverse maxillary deficiency. In Epker BN, Fish LC, editors: Dentofacial deformities: Integrated orthodontic and surgical correction, Vol II, St Louis, 1986, CV Mosby Co, pp 618–675.
97. Epker BN, Wessberg GA: Mechanisms of early skeletal relapse following surgical advancement of the mandible. *Br J Oral Surg* 20:175, 1982.
98. Epker BN, Wylie A: Control of the condylar–proximal mandibular segments after sagittal split osteotomies to advance the mandible. *Oral Surg Oral Med Oral Pathol* 62:613, 1986.
99. Escenazi LB, Schendel SA: An analysis of Le Fort I maxillary advancement in cleft lip and palate patients. *Plast Reconstr Surg* 90:779–786, 1992.
100. Espeland L, Dowling PA, Mobarak KA, et al: Three-year stability of open-bite correction by 1-piece maxillary osteotomy. *Am J Orthod Dentofacial Orthop* 134:60, 2008.
101. Farrell CD, Kent JN: Evaluation of the surgical stability of 20 cases of inverted-L and C osteotomies. *J Oral Surg* 35:239, 1977.
102. Ferretti C, Reyneke JP: Mandibular, sagittal split osteotomies fixed with biodegradable or titanium screws: A prospective, comparative study of postoperative stability. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 93:534, 2002.
103. Fink DF, Smith RJ: The duration of orthodontic treatment. *Am J Orthod Dentofac Orthop* 102:45, 1992.
104. Finn RA: Biomechanical considerations in the surgical correction of mandibular deficiency. *J Oral Surg* 38:257–264, 1980.
105. Fish LC, Epker BN: Prevention of relapse in surgical-orthodontic treatment: Part 1. Mandibular procedures. *J Clin Orthod* 20:826, 1986.
106. Foley WL: In-vitro comparison of parameters affecting the fixation strength of sagittal split osteotomies [discussion]. *J Oral Maxillofac Surg* 53:1383, 1995.
107. Foley WL, Frost DE, Paulin WB, et al: Internal screw fixation: Comparison of placement pattern and rigidity. *J Oral Maxillofac Surg* 47:720, 1989.
108. Franco JE, Van Sickels JE, Thrash WJ: Factors contributing to relapse in rigidly fixed mandibular setbacks. *J Oral Maxillofac Surg* 47:451, 1989.
109. Freihofer HP: Results of osteotomies of the facial skeleton in adolescence. *J Maxillofac Surg* 5:267–297, 1977.
110. Freihofer HPM, Jr, Petresevic D: Late results after advancing the mandible by sagittal splitting of the rami. *J Maxillofac Surg* 3:250, 1975.
111. Fujioka M, Fujii T, Hirano A: Comparative study of mandibular stability after sagittal split osteotomies: Bicortical versus

monocortical osteosynthesis. *Cleft Palate Craniofac J* 37:551–555, 2000.
112. Fujioka M, Fujii T, Hirano A: Complete breakage of three-dimensional miniplates: Unusual complication of osteosynthesis after sagittal split osteotomy. *Scand J Plast Reconstr Hand Surg* 34:259, 2000.
113. Gallo W, Moss M, Gaul J: Modification of the sagittal split ramus osteotomy for retrognathia. *J Oral Surg* 34:178–179, 1976.
114. Garrison BT, Lapp TH, Bussard DA: The stability of Le Fort I maxillary osteotomies in patients with simultaneous alveolar cleft bone grafts. *J Oral Maxillofac Surg* 45:761–766, 1987.
115. Gassman CJ, Van Sickels JE, Thrash WJ: Causes, location, and timing of relapse following rigid fixation after mandibular advancement. *J Oral Maxillofac Surg* 48:450, 1990.
116. Gerressen M, Stockbrink G, Smeets R, et al: Skeletal stability following bilateral sagittal split osteotomy (BSSO) with and without condylar positioning device. *J Oral Maxillofac Surg* 65:1297, 2007.
117. Gill DS, El Maaytah M, Naini FB: Risk factors for post-orthognathic condylar resorption: A review. *World J Orthod* 9:21, 2008.
118. Gonçalves JR, Cassano DS, Wolford LM, et al: Postsurgical stability of counterclockwise maxillomandibular advancement surgery: Affect of articular disc repositioning. *J Oral Maxillofac Surg* 66:724, 2008.
119. Greulich WW, Pyle SI: *Radiographic atlas of skeletal development of the hand and wrist*, ed 2, Stanford, Calif, 1959, Stanford University Press.
120. Guernse YL: Stability of treatment results in Class II malocclusion corrected by full mandibular advancement surgery. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 37:668, 1974.
121. Gurstein KW, Sather AH, An KN, et al: Stability after inferior or anterior maxillary repositioning by Le Fort I osteotomy: A biplanar stereocephalometric study. *Int J Adult Orthodon Orthognath Surg* 13:131, 1998.
122. Haas AJ: Rapid expansion of the maxillary dental arch and nasal cavity by opening the midpalatal suture. *Angle Orthod* 31:73–90, 1961.
123. Haas AJ: The treatment of maxillary deficiency by opening the midpalatal suture. *Angle Orthod* 35:200–217, 1965.
124. Haas AJ: Palatal expansion: Just the beginning of dentofacial orthopedics. *Am J Orthod* 57:219–255, 1970.
125. Haas AJ: Rapid palatal expansion: A recommended prerequisite to Class III treatment. *Trans Eur Orthod Soc* 311–318, 1973.
126. Hackney FL, Van Sickels JE, Nummikoski PV: Condylar displacement and temporomandibular joint dysfunction following bilateral sagittal split osteotomy and rigid fixation. *J Oral Maxillofac Surg* 47:223, 1989.
127. Hadjianghelou O, Luder HU, von Weydlich G: Rezidivursachen nach korrektur der progenie mit sagittaler spaltung. *Informationen aus der kieferorthopädie* 17:185–204, 1985.
128. Haers PE, Sailer HF: Biodegradable self-reinforced poly-L/DL-lactide plate and screws in bimaxillary orthognathic surgery: Short-term skeletal stability and material related failures. *J Craniomaxillofac Surg* 26:363, 1998.
129. Hagg U, Taranger J: Skeletal stages of the hand and wrist as indicators of the pubertal growth spurt. *Acta Odontol Scand* 38:187, 1980.
130. Hagg U, Taranger J: Maturational indicators and the pubertal growth spurt. *Am J Orthod* 82:299–309, 1982.
131. Harada K, Enomoto S: Stability after surgical correction of mandibular prognathism using the sagittal split ramus osteotomy and fixation with poly-L-lactic acid screws. *J Oral Maxillofac Surg* 55:464, 1997.
132. Harada K, Tajima K, Enomoto S: Stability after sagittal split ramus osteotomy without post-operative maxillomandibular fixation in the treatment of prognathic patients with asymmetric mandibles. *Clin Orthod Res* 2:27, 1999.
133. Hartgerink DV, Vig PS, Abbot DW: The effect of rapid maxillary expansion on nasal airway resistance. *Am J Orthod* 92:381–389, 1987.
134. Haymond CS, Stoelinga PJW, Blijdorp PA, et al: Surgical orthodontic treatment of anterior skeletal open bite using small plate internal fixation. One to five year follow up. *Int J Oral Maxillofac Surg* 20:223, 1991.
135. Hedemark A, Freihofer HP: The behaviour of the maxilla in vertical movements after the Le Fort I osteotomy. *J Maxillofac Surg* 6:244–249, 1978.
136. Hilbe M, Puelacher W: Die stabilität nach sagittaler unterkieferosteotomie zur korrektur distaler und progener bisslagen. *Inf Kieferortho* 26:63, 1994.
137. Hing RN: Early relapse following bilateral sagittal split advancement. *Aust Orthod J* 11:100, 1989.
138. Hirano A, Suzuki H: Factors related to relapse after Le-Fort I maxillary advancement osteotomy in patients with cleft lip and palate. *Cleft Palate Craniofac J* 38:1–10, 2001.
139. Hochban W, Ganss C, Austermann KH: Long-term results after maxillary advancement in patients with clefts. *Cleft Palate Craniofac J* 30:237–243, 1993.
140. Hoffman GR, Moloney FB: The stability of facial osteotomies: 2. Mandibular advancement with bicortical screw fixation. *Aust Dent J* 40:213, 1995.
141. Höltje WJ, Scheuer H: Skeletal stability and relapse patterns after Le Fort I osteotomy using miniplate fixation in patients with isolated cleft palate [discussion]. *Plast Reconstr Surg* 94:59–60, 1994.
142. Hoppenreijs TJ, Freihofer HP, Stoelinga PJ, et al: Skeletal and dento-alveolar stability of Le Fort I intrusion osteotomies and bimaxillary osteotomies in anterior open bite deformities: A retrospective three-centre study. *Int J Oral Maxillofac Surg* 26:161–175, 1997.
143. Hoppenreijs TJ, Freihofer HP, Stoelinga PJ, et al: Condylar remodelling and resorption after Le Fort I and bimaxillary osteotomies in patients with anterior open bite: A clinical and radiological study. *Int J Oral Maxillofac Surg* 27:81, 1998.
144. Hoppenrejis TJM, Stoelinga PJW, Grace KL, et al: Long-term evaluation of patients with progressive condylar resorption following orthognathic surgery. *Int J Oral Maxillofac Surg* 28:411, 1999.
145. Hörster W: Experience with functionally stable plate osteosynthesis after forward displacement of the upper jaw. *J Maxillofac Surg* 8:176–181, 1980.
146. Houston WJB, James DR, Jones E, Kavvadia S: Le Fort I maxillary osteotomies in cleft palate cases: Surgical changes and stability. *J Craniomaxillofac Surg* 17:9–15, 1989.
147. Houston WJB, Jones E, James DR: A method of recording change in maxillary position following orthognathic surgery. *Eur J Orthod* 9:9–14, 1987.
148. Hwang SJ, Haers PE, Sailer HF: The role of a posteriorly inclined condylar neck in condylar resorption after orthognathic surgery. *J Craniomaxillofac Surg* 28:85, 2000.
149. Iannetti G, Chimenti C, Di Paolo C: Five-year follow-up of Le Fort I osteotomies. *J Craniomaxillofac Surg* 15:238, 1987.
150. Ingervall B, Thüer U, Vuillemin T: Stability and effect on the soft tissue profile of mandibular setback with sagittal split osteotomy and rigid internal fixation. *Int J Adult Orthodon Orthognath Surg* 10:15–25, 1995.
151. Isaacson RJ, Ingram AH: Forces produced by rapid maxillary expansion. II. Forces present during treatment. *Angle Orthod* 34:261–270, 1964.
152. Jäger A, Kubein-Meesenburg D, Luhr HG: Longitudinal study of combined orthodontic and surgical treatment of class II malocclusion with deep overbite. *Int J Adult Orthodon Orthognath Surg* 6:29, 1991.
153. Jeter TS, Van Sickels JE, Dolwick MF: Modified techniques for internal fixation of sagittal ramus osteotomies. *J Oral Maxillofac Surg* 42:270–272, 1984.
154. Joss CU, Thüer UW: Stability of hard and soft tissue profile after mandibular advancement in sagittal split osteotomies: A longitudinal and long-term follow-up study. *Eur J Orthod* 30:16–23, 2008.
155. Kahnberg KE, Kashani H, Owman-Moll P: Sagittal split advancement osteotomy: Comparison of the tendency to relapse after two different methods of rigid fixation.

Scand J Plast Reconstr Surg Hand Surg 41:167, 2007.
156. Kallela I, Laine P, Suuronen R, et al: Skeletal stability following mandibular advancement and rigid fixation with polylactide biodegradable screws. *Int J Oral Maxillofac Surg* 27:3, 1998.
157. Keeling SD, Dolce C, Van Sickels JE, et al: A comparative study of skeletal and dental stability between rigid and wire fixation for mandibular advancement. *Am J Orthod Dentofacial Orthop* 117:638, 2000.
158. Keeling SD, LaBanc JP, Van Sickels JE, et al: Skeletal change at surgery as a predictor of long-term soft tissue profile change after mandibular advancement. *J Oral Maxillofac Surg* 54:134, 1996.
159. Kerstens JCK, Tuinzig DB, Golding RP, et al: Condylar atrophy and osteoarthritis after bimaxillary surgery. *Oral Surg Oral Med Oral Pathol* 69:274, 1990.
160. Kierl MJ, Nanda RS, Currier GF: A 3-year evaluation of skeletal stability of mandibular advancement with rigid fixation. *J Oral Maxillofac Surg* 48:587, 1990.
161. Kim YJ, Oh KM, Hong JS, et al: Do patients treated with bimaxillary surgery have more stable condylar positions than those who have undergone single jaw surgery. *J Oral Maxillofac Surg* 70:2143–2152, 2012
162. Kirkpatrick TB, Woods MG, Swift JQ, et al: Skeletal stability following mandibular advancement and rigid fixation. *J Oral Maxillofac Surg* 45:472, 1987.
163. Knaup CA, Wallen TR, Bloomquist DS: Linear and rotational changes in large mandibular advancements using three or four fixation screws. *Int J Adult Orthodon Orthognath Surg* 8:245, 1993.
164. Kobayashi T, Watanabe I, Ueda K, Nakajima T: Stability of the mandible after sagittal ramus osteotomy for correction of prognathism. *J Oral Maxillofac Surg* 44:693–697, 1986.
165. Kohn DH, Richmond EM, Dootz ER, et al: In vitro comparison of parameters affecting the fixation strength of sagittal split osteotomies. *J Oral Maxillofac Surg* 53:1374, 1995.
166. Komori E, Aigase K, Sugisaki M, Tanabe H: Skeletal fixation versus skeletal relapse. *Am J Orthod Dentofacial Orthop* 92:412–421, 1987.
167. Koudstaal MJ, Poort LJ, van der Wal KG, et al: Surgically assisted rapid maxillary expansion (SARME): A review of the literature. *Int J Oral Maxillofac Surg* 34:709, 2005.
168. Krebs A: Midpalatal suture expansion studied by the implant method over a seven-year period. *Trans Eur Orthod Soc* 40:131–142, 1964.
169. Krekmanov L: Orthognathic surgery without the use of postoperative intermaxillary fixation: A clinical and cephalometric evaluation of surgical correction of mandibular and maxillary deformities. *Swed Dent J Suppl* 61:8–62, 1989.
170. Krekmanov L, Lilja J, Ringqvist M: Simultaneous correction of maxillary and mandibular dentofacial deformities without the use of postoperative intermaxillary fixation: A clinical and cephalometric study. *Int J Oral Maxillofac Surg* 17:363–370, 1988.
171. Kretschmer WB, Baciut G, Baciut M, et al: Transverse stability of 3-piece Le Fort I osteotomies. *J Oral Maxillofac Surg* 69:861–869, 2011.
172. Kufner J: Four-year experience with major maxillary osteotomy for retrusion. *J Oral Surg* 29:549–553, 1971.
173. Kundert M, Hadjianghelou O: Condylar displacement after sagittal splitting of the mandibular rami. *J Maxillofac Surg* 8:278–287, 1980.
174. LaBanc JP, Turvey TA, Epker BN: Results following simultaneous mobilization of the maxilla and mandible for the correction of dentofacial deformities: Analysis of 100 consecutive patients. *Oral Surg* 54:607–612, 1982.
175. Lagravere MO, Carey J, Heo G, et al: Transverse, vertical, and anteroposterior changes from bone-anchored maxillary expansion: A randomized clinical trial. *Am J Orthod Dentofacial Orthop* 137:e1, 2010
176. Lagravere MO, Major PW, Flores-Mir C: Dental and skeletal changes following surgically assisted rapid maxillary expansion. *Int J Oral Maxillofac Surg* 35:481, 2006.
177. Lanigan DT, Romanchuk K, Olson CK: Ophthalmic complications associated with orthognathic surgery. *J Oral Maxillofac Surg* 51:480, 1993.
178. Larsen AJ, Van Sickels JE, Thrash WJ: Postsurgical maxillary movement: A comparison study of bone plate versus wire osseous fixation. *Am J Orthod Dentofacial Orthop* 95:334, 1989.
179. Lavergne J, Gasson N: A metal implant study of mandibular rotation. *Angle Orthod* 46:144–150, 1976.
180. Lee J, Piecuch JF: The sagittal ramus osteotomy: Stability of fixation with internal miniplates. *Int J Oral Maxillofac Surg* 21:326, 1992.
181. Leonard MS: Maintenance of condylar position after sagittal split osteotomy of the mandible. *J Oral Maxillofac Surg* 43:391, 1985.
182. Li KK, Meara JG, Rubin PA: Orbital compartment syndrome following orthognathic surgery. *J Oral Maxillofac Surg* 53:964. 1995
183. Lindorf HH: Functionally stable tandem screw fixation in sagittal ramus osteotomy. Surgical technique. *Dtsch Z Mund Kiefer Gesichtschir* 8:367–373, 1984.
184. Lindorf HH: Sagittal ramus osteotomy with tandem screw fixation: Technique and results. *J Maxillofac Surg* 14:311, 1986.
185. Lines PA: Adult rapid maxillary expansion with corticotomy. *Am J Orthod* 67:44–56, 1975.
186. Lo FM, Shapiro PA: Effect of presurgical incisor extrusion on stability of anterior open bite malocclusion treated with orthognathic surgery. *Int J Adult Orthodon Orthognath Surg* 13:23–34, 1998.
187. Louis PJ, Waite PD, Austin RB: Long-term skeletal stability after rigid fixation of Le Fort I osteotomies with advancements. *Int J Oral Maxillofac Surg* 22:82–86, 1993.
188. Luhr HG: Zur stabilen osteosynthese bei unterkiefer–frakturen. *Dtsch Zahnarztl Z* 23:754, 1968.
189. Luhr HG: Skelettverlagernde operationen zur harmonisierung des gesichtsprofils—probleme der stabilen fixation von osteotomiesegmenten. In Pfeifer G von, editor: *Die esthetik von form und funktion in der plastischen und wiederherstellungs–chirurgie*, Berlin, 1985, Springer-Verlag, p 87.
190. Luther F, Morris DO, Hart C: Orthodontic preparation for orthognathic surgery: How long does it take and why? A retrospective study. *Br J Oral Maxillofac Surg* 41:401, 2003.
191. Luyk NH, Ward BR: The stability of Le Fort I advancement osteotomies using bone plates without bone grafts. *J Maxillofac Surg* 13:250–253, 1985.
192. Macintosh RB: Experience with the sagittal split osteotomy of the mandibular ramus. A 13-year review. *J Maxillofac Surg* 9:151–164, 1981.
193. Major PW, Philippson GE, Glover KE, et al: Stability of maxilla downgrafting after rigid or wire fixation. *J Oral Maxillofac Surg* 54:1287, 1996.
194. Marchetti C, Gentile L, Bianchi A, Bassi M: Semirigid fixation of the mandible in bimaxillary orthognathic surgery: Stability after 18 months. *Int J Adult Orthodon Orthognath Surg* 14:37–45, 1999.
195. Marchetti C, Pironi M, Bianchi A, et al: Surgically assisted rapid palatal expansion vs. segmental Le Fort I osteotomy: Transverse stability over a 2-year period. *J Craniomaxillofac Surg* 37:74, 2009.
196. Mayo KH, Ellis E, III: Stability of the mandible after advancement and use of dental plus skeletal maxillomandibular fixation: An experimental investigation in *Macaca mulatta*. *J Oral Maxillofac Surg* 45:243, 1987.
197. McCance AM, Moss JP, James DR: Stability of surgical correction of patients with skeletal III and skeletal II anterior open bite, with increased maxillary mandibular planes angle. *Eur J Orthod* 14:198, 1992.
198. McDonald WR, Stoelinga PJ, Blijdorp PA, Schoenaers JA: Champy bone plate fixation in sagittal split osteotomies for mandibular advancement. *Int J Adult Orthodon Orthognath Surg* 2:89–97, 1987.
199. Melsen B: A histological study of the influence of sutural morphology and skeletal maturation on rapid palatal expansion in children. *Trans Eur Orthod Soc* 48:499–507, 1972.

200. Memikoglu TU, Iseri H: Effect of a bonded rapid maxillary expansion appliance during orthodontic treatment. *Angle Orthod* 69:251–256, 1999.
201. Meng H, Goorhuis J, Kapila S, Nanda S: Growth changes in the nasal profile from 7 to 18 years of age. *Am J Orthod Dentofacial Orthop* 94:317–326, 1988.
202. Merkx MA, Van Damme PA: Condylar resorption after orthognathic surgery: Evaluation of treatment in 8 patients. *J Craniomaxillofac Surg* 22:53, 1994.
203. Miguel JA, Turvey TA, Phillips C, Proffit WR: Long-term stability of two-jaw surgery for treatment of mandibular deficiency and vertical maxillary excess. *Int J Adult Orthodon Orthognath Surg* 10:235–245, 1995.
204. Miguel JA, Turvey TA, Phillips G, Proffit WR: Long-term stability of two-jaw surgery for treatment of mandibular treatment outcome. *Int J Adult Orthodon Orthognath Surg* 13:35–44, 1998.
205. Mihalik CA, Proffit WR, Phillips C: Long-term follow-up of Class II adults treated with orthodontic camouflage: A comparison with orthognathic surgery outcomes. *Am J Orthod Dentofacial Orthop* 123:266–278, 2003.
206. Mobarak KA, Espeland L, Krogstad O, et al: Mandibular advancement surgery in high-angle and low-angle class II patients: Different long-term skeletal responses. *Am J Orthod Dentofacial Orthop* 119:368, 2001.
207. Mobarak KA, Krogstad O, Espeland L, Lyberg T: Long-term stability of mandibular setback surgery: A follow-up of 80 bilateral sagittal split osteotomy patients. *Int J Adult Orthodon Orthognath Surg* 15:83–95, 2000.
208. Moen K, Wisth PJ, Skaale S, et al: Dental or skeletal relapse after sagittal split osteotomy advancement surgery? Long-term follow up. *J Oral Maxillofac Surg* 69:e461–e468, 2011.
209. Moenning JE, Bussard DA, Lapp TH, et al: A comparison of relapse in bilateral sagittal split osteotomies for mandibular advancement: Rigid internal fixation (screws) versus inferior border wiring with anterior skeletal fixation. *Int J Adult Orthodon Orthognath Surg* 5:175, 1990.
210. Moldez MA, Sugawara J, Umemori M, et al: Long-term dentofacial stability after bimaxillary surgery in skeletal Class III open bite patients. *Int J Adult Orthodon Orthognath Surg* 15:309, 2000.
211. Mommaerts MY, Collado J, Mareque Bueno J: Morbidity related to "endo-corticotomies" for transpalatal osteodistraction. *J Craniomaxillofac Surg* 36:198, 2008.
212. Moore KE, Gooris PJ, Stoelinga PJ: The contributing role of condylar resorption to skeletal relapse following mandibular advancement surgery: Report of five cases. *J Oral Maxillofac Surg* 49:448, 1991.
213. Moser K, Freihofer HPM: Long-term experience with simultaneous movement of the upper and lower jaw. *J Maxillofac Surg* 10:271–277, 1980.
214. Moyers RE, van der Linden FPGM, Riolo ML, et al: *Standards of human occlusal development*. Ann Arbor, Mich, 1976, University of Michigan Center for Human Growth and Development.
215. Muller G: Growth and development of the middle face. *J Dent Res* 42:385–399, 1963.
216. Nagai I, Tanaka N, Noguchi M, et al: Changes in occlusal state of patients with mandibular prognathism after orthognathic surgery: A pilot study. *Br J Oral Maxillofac Surg* 39:429, 2001.
217. Nance HN: The limitations of orthodontic treatment. *Am J Orthod* 33:177–223, 1947.
218. Nanda RS: The rates of growth of several facial components measured from serial cephalometric roentgenograms. *Am J Orthod* 41:658–673, 1955.
219. Nanda RS: Growth changes in skeletal-facial profile and their significance in orthodontic diagnosis. *Am J Orthod* 59:501–513, 1971.
220. Nanda RS: Patterns of vertical growth in the face. *Am J Orthod* 93:103–116, 1988.
221. Nanda RS: Circumpubertal growth spurt related to vertical dysplasia. *Angle Orthod* 59–60:113–122, 1989.
222. Nanda RS: Growth patterns in subjects with long and short faces. *Am J Orthod Dentofacial Orthop* 98:247–258, 1990.
223. Nanda RS, Meng H, Kapila S, Goorhuis J: Growth changes in the soft tissue facial profile. *Angle Orthod* 60:177–190, 1990.
224. Nanda RS, Nanda SK: Considerations of dentofacial growth in long-term retention and stability: Is active retention needed? *Am J Orthod Dentofacial Orthop* 101:297–302, 1992.
225. Northway WM, Meade JB, Jr: Surgically assisted rapid maxillary expansion: A comparison of technique, response, and stability. *Angle Orthod* 67:309, 1997.
226. Ochoa BK, Nanda RS: Comparison of maxillary and mandibular growth. *Am J Orthod Dentofacial Orthop* 125:148, 2004.
227. Oliveira JA, Bloomquist DS: The stability of the use of bilateral sagittal split osteotomy in the closure of anterior open bite. *Int J Adult Orthodon Orthognath Surg* 12:101–108, 1997.
228. Ow A, Cheung LK: Skeletal stability and complications of bilateral sagittal split osteotomies and mandibular distraction osteogenesis: An evidence-based review. *J Oral Maxillofac Surg* 67:2344, 2009.
229. Pangrazio-Kulbersh V, Berger JL, Kaczynski R, et al: Stability of skeletal class II correction with 2 surgical techniques: The sagittal split ramus osteotomy and the total mandibular subapical alveolar osteotomy. *Am J Orthod Dentofacial Orthop* 120:134, 2001.
230. Paulus GW, Steinhauser EW: A comparative study of wire osteosynthesis versus bone screws in the treatment of mandibular prognathism. *Oral Surg Oral Med Oral Pathol* 54:2–6, 1982.
231. Peck H, Peck S: A concept of facial esthetics. *Angle Orthod* 40:284–318, 1970.
232. Pepersack WJ, Chausse JM: Long term follow-up of the sagittal splitting technique for correction of mandibular prognathism. *J Maxillofacial Surg* 6:117–140, 1978.
233. Perez MM, Sameshima GT, Sinclair PM: The long-term stability of Le Fort I maxillary downgrafts with rigid fixation to correct vertical maxillary deficiency. *Am J Orthod Dentofacial Orthop* 112:104, 1997.
234. Persson G, Hellem S, Nord PG: Bone-plates for stabilizing Le Fort I osteotomies. *J Maxillofac Surg* 14:69–73, 1986.
235. Phillips C, Medland WH, Fields HW, Jr, et al: Stability of surgical maxillary expansion. *Int J Adult Orthodon Orthognath Surg* 7:139, 1992.
236. Phillips C, Zaytoun HS, Thomas PM, Terry BC: Skeletal alterations following TOVRO or BSSO procedures. *Int J Adult Orthodon Orthognath Surg* 3:203–213, 1986.
237. Phillips RM, Bell WH: Atrophy of mandibular condyles after sagittal ramus split osteotomy: Report of case. *J Oral Surg* 36:45, 1978.
238. Pogrel MA, Kaban LB, Vargerik K, Baumrind S: Surgically assisted rapid maxillary expansion in adults. *Int J Adult Orthodon Orthognath Surg* 7:37–41, 1992.
239. Posnick JC, Dagys AP: Skeletal stability and relapse patterns after Le Fort I maxillary osteotomy fixed with miniplates: The unilateral cleft lip and palate deformity. *Plast Reconstr Surg* 94:924–932, 1994.
240. Posnick JC, Ewing MP: Skeletal stability after Le Fort I maxillary advancement in patients with unilateral cleft lip and palate. *Plast Reconstr Surg* 85:706–710, 1990.
241. Posnick JC, Taylor M: Skeletal stability and relapse patterns after Le Fort I osteotomy using miniplate fixation in patients with isolated cleft palate. *Plast Reconstr Surg* 94:51–58, 1994.
242. Posnick JC, Tompson B: Modification of the maxillary Le Fort I osteotomy in cleft-orthognathic surgery: The unilateral cleft lip and palate deformity. *J Oral Maxillofac Surg* 50:666–675, 1992.
243. Posnick JC, Tompson B: Cleft-orthognathic surgery: Complications and long-term results. *Plast Reconstr Surg* 96:255–266, 1995.
244. Proffit WR: Stability of Le Fort I osteotomy with maxillary advancement: A comparison of combined wire fixation and rigid fixation [discussion]. *J Oral Maxillofac Surg* 53:248, 1995.
245. Proffit WR, Bailey LJ, Phillips C, Turvey TA: Long-term stability of surgical open-bite correction by Le Fort I osteotomy. *Angle Orthod* 70:112, 2000.
246. Proffit WR, Miguel JA: The duration and sequencing of surgical-orthodontic treatment. *Int J Adult Orthodon Orthognath Surg* 10:35, 1995.
247. Proffit WR, Phillips C, Dann C, IV: Who seeks surgical-orthodontic treatment? *Int J Adult Orthodon Orthognath Surg* 5:153–160, 1990.

248. Proffit W, Phillips C, Dann C, Turvey T: Stability after surgical-orthodontic correction of skeletal class III malocclusion: I. Mandibular set-back. *Int J Adult Orthodon Orthognath Surg* 6:7–18, 1991.
249. Proffit WR, Phillips C, Prewitt JW, Turvey TA: Stability after surgical-orthodontic correction of skeletal Class III malocclusion: II. Maxillary advancement. *Int J Adult Orthodon Orthognath Surg* 6:71–80, 1991.
250. Proffit WR, Phillips C, Turvey TA: Stability following superior repositioning of the maxilla by Le Fort I osteotomy. *Am J Orthod Dentofacial Orthop* 92:151–161, 1987.
251. Proffitt WR, Phillips C, Turvey, TA: Stability after mandibular setback: Mandible-only versus 2-jaw surgery. *J Oral Maxillofac Surg* 70:e408–e414, 2012.
252. Proffit WR, Turvey TA, Philips C: Orthognathic surgery: A hierarchy of stability. *Int J Adult Orthodon Orthognath Surg* 11:191–204, 1996.
253. Quejada JG, Bell WH, Kawamura H, Zhang X: Skeletal stability after inferior maxillary repositioning. *Int J Adult Orthodon Orthognath Surg* 2:67–74, 1987.
254. Ramieri GA, Spada MC, Austa M, et al: Transverse maxillary distraction with a bone-anchored appliance: dento-periodontal effects and clinical and radiological results. *Int J Oral Maxillofac Surg* 34:357, 2005
255. Raveh J, Vuillemin T, Lädrach K, Sutter F: New techniques for reproduction of the condyle relation and reduction of complications after sagittal ramus split osteotomy of the mandible. *J Oral Maxillofac Surg* 46:751–757, 1988.
256. Reitzik M: Skeletal and dental changes after surgical correction of mandibular prognathism. *J Oral Surg* 38:109, 1980.
257. Ricketts RM: The biologic significance of the divine proportion and Fibonacci series. *Am J Orthod* 81(5):351–370, 1982.
258. Riolo ML, Moyers RE, McNamara JA, Jr, Hunter WST: *An atlas of craniofacial growth*, Ann Arbor, Mich, 1974, University of Michigan.
259. Rosen HM: Miniplate fixation of Le Fort I osteotomies. *Plast Reconstr Surg* 78:748–754, 1986.
260. Rosen HM: Segmental osteotomies of the maxilla. *Clin Plast Surg* 16:785, 1989.
261. Rosen HM: Facial skeletal expansion: Treatment strategies and rationale. *Plast Reconstr Surg* 89:798, 1992.
262. Rosenquist B, Rune B, Peterssen A, et al: Condylar displacement after oblique sliding osteotomy of the mandibular rami: A stereometric and plain radiographic study. *J Craniomaxillofac Surg* 16:301, 1988.
263. Rotskoff KS, Herbosa EG, Villa P: Maintenance of condyle-proximal segment position in orthognathic surgery. *J Oral Maxillofac Surg* 49:2, 1991.
264. Rotter BE, Zeitler DL: Stability of the Le Fort I maxillary osteotomy after rigid internal fixation. *J Oral Maxillofac Surg* 57:1080, 1999.
265. Rubens BC, Stoelinga PJ, Blijdorp PA, et al: Skeletal stability following sagittal split osteotomy using monocortical miniplate internal fixation. *Int J Oral Maxillofac Surg* 17:371–376, 1988.
266. Ruston MA: Growth at the mandibular condyle in relation to some deformities. *Br Dent J* 76:57–68, 1944.
267. Sabri R: Orthodontic objectives in orthognathic surgery: State of the art today. *World J Orthod* 7:177–191, 2006.
268. Sandlkçloglu M, Hazar S: Skeletal and dental changes after maxillary expansion in the mixed dentition. *Am J Orthod Dentofacial Orthop* 111:321–327, 1997.
269. Sarver DM, Johnston MW: Skeletal changes in vertical and anterior displacement of the maxilla with bonded rapid palatal expansion appliances. *Am J Orthod Dentofacial Orthop* 95:462–466, 1989.
270. Savara BS, Singh IJ: Norms of size and annual increments of seven anatomical measures of maxillae in boys from 3 to 16 years of age. *Angle Orthod* 38:104, 1968.
271. Schatz J-P, Tsimas P: Cephalometric evaluation of surgical-orthodontic treatment of skeletal Class III malocclusion. *Int J Adult Orthodon Orthognath Surg* 10:173–180, 1995.
272. Scheerlinck JP, Stoelinga PJW, Blijdorp PA, et al: Sagittal split advancement osteotomies stabilized with miniplates: A 2–5-year follow-up. *Int J Oral Maxillofac Surg* 23:127–131, 1994.
273. Schubert P, Bailey LJ, White RP, Proffit WR: Long-term cephalometric changes in untreated adults compared to those treated with orthognathic surgery. *Int J Adult Orthodon Orthognath Surg* 14:91–99, 1999.
274. Schudy F: Vertical growth versus anteroposterior growth as related to function and treatment. *Angle Orthod* 34–35:75–93, 1964.
275. Scott J: The analysis of facial growth: I. The anteroposterior and vertical dimensions. *Am J Orthod* 44:507–512, 1958.
276. Seigel ML: Mechanisms of early maxillary growth: Implications for surgery. *J Oral Surg* 34:106, 1976.
277. Seitz O, Landes CA, Philipp DJ, et al: Reliable surgically assisted rapid palatal expansion by maxillary widening device. *J Craniofac Surg* 19:846, 2008.
278. Sesenna E, Raffaini M: Bilateral condylar atrophy after combined osteotomy for correction of retrusion: A case report. *J Oral Maxillofac Surg* 13:263, 1985.
279. Shand JM, Heggie AA: Use of a resorbable fixation system in orthognathic surgery. *Br J Oral Maxillofac Surg* 38:335, 2000.
280. Shuttleworth FK: Sexual maturation and the physical growth of girls age six to nineteen. Monograph No 5. *Soc Res Child Dev (serial no 12)* 1937.
281. Sillman JH: Dimensional changes in the dental arches: Longitudinal study from birth to 25 years. *Am J Orthod Dentofacial Orthop* 50:824, 1964.
282. Silva Filho OG, Montes LA, Torelly LF: Rapid maxillary expansion in the deciduous and mixed dentition evaluated through posteroanterior cephalometric analysis. *Am J Orthod Dentofacial Orthop* 107:268–275, 1995.
283. Silva Filho OG, Villas Boas MC, Capelozza Filho L: Rapid maxillary expansion in the primary and mixed dentitions: A cephalometric evaluation. *Am J Orthod Dentofacial Orthop* 100:171–181, 1991.
284. Silverstein K, Quinn PD: Surgically-assisted rapid palatal expansion for management of transverse maxillary deficiency. *J Oral Maxillofac Surg* 55:725–727,1997.
285. Simmons KE, Turvey TA, Phillips C, Proffit WR: Surgical-orthodontic correction of mandibular deficiency: Five-year follow-up. *Int J Adult Orthodon Orthognath Surg* 7:67–79, 1992.
286. Singh IJ, Savara BS: Norms of size and annual increments of seven anatomical measures of maxillae in girls from 3 to 17 years of age. *Angle Orthod* 36:312, 1968.
287. Sittitavornwong S, Waite PD, Dann JJ, et al: The stability of maxillary osteotomies fixated with biodegradable mesh in orthognathic surgery. *J Oral Maxillofac Surg* 64:1631, 2006.
288. Skidmore KJ, Brook KJ, Thomson WM, et al: Factors influencing treatment time in orthodontic patients. *Am J Orthod Dentofacial Orthop* 129:230, 2006.
289. Skieller V, Björk A, Linde-Hanson T: Prediction of mandibular growth rotation evaluated from a longitudinal implant sample. *Am J Orthod* 86:359–370, 1984.
290. Snow MD, Turvey TA, Walker D, Proffit WR: Surgical mandibular advancement in adolescents: postsurgical growth related to stability. *Int J Adult Orthodon Orthognath Surg* 6:143–151, 1991.
291. Sokucu O, Kosger HH, Bicakci AA, et al: Stability in dental changes in RME and SARME: A 2-year followup. *Angle Orthod* 79:207, 2009.
292. Sorokolit CA, Nanda RS: Assessment of the stability of mandibular setback procedures with rigid fixation. *J Oral Maxillofac Surg* 48:817–822, 1990.
293. Souyris F: Sagittal splitting and bicortical screw fixation of the ascending ramus. *J Maxillofac Surg* 6:198, 1978.
294. Spiessel B: Osteosynthese bei sagittaler osteotomie nach Obwegeser/Dal Pont. *Fortschr Kiefer Gesichts Chir* 18:145, 1974.
295. Spiessel B: Rigid internal fixation after sagittal split osteotomy of the ascending ramus. In Spiessl B, editor: *New Concepts in Maxillofacial Bone Surgery*, New York, 1976, Springer-Verlag, pp 115–122.
296. Steinhäuser EW: Advancement of the mandible by sagittal ramus split and suprahyoid myotomy. *J Oral Surg* 31:516, 1973.
297. Stella JP, Astrand P, Epker BN: Patterns and etiology of relapse after correction of class III open bite via subcondylar ramus osteotomy.

Int J Adult Orthodon Orthognath Surg 1:91, 1986.
298. Stoelinga PJW, Borstlap WA: The fixation of sagittal split osteotomies with miniplates: The versatility of a technique. *J Oral Maxillofac Surg* 61:1471–1476, 2003.
299. Stromberg C, Holm J: Surgically-assisted rapid maxillary expansion in adults: A retrospective long-term follow-up study. *J Craniomaxillofac Surg* 23:222, 1995.
300. Stroster TG, Pangrazio-Kulbersh J: Assessment of condylar position following bilateral sagittal split ramus osteotomy with wire fixation or rigid fixation. *Int J Adult Orthodon Orthognath Surg* 9:55–63, 1994.
301. Subtelny JD: The soft tissue profile, growth and treatment changes. *Angle Orthod* 31:105–122, 1961.
302. Tanner JM: *Growth at adolescence*, Oxford, UK, 1962, Blackwell Scientific.
303. Thaller SR: Re: A comparison of the stability of single-piece and segmental Le Fort I maxillary advancements. *J Craniofac Surg* 14:599, 2003.
304. Thomas PM, Tucker MR, Prewitt JR, et al: Early skeletal and dental changes following mandibular advancement and rigid internal fixation. *Int J Adult Orthodon Orthognath Surg* 3:171, 1986.
305. Thüer U, Ingervall B, Vuillemin T: Stability and effect on the soft tissue profile of mandibular advancement with sagittal split osteotomy and rigid internal fixation. *Int J Adult Orthodon Orthognath Surg* 9:175, 1994.
306. Timms DJ: A study of basal movement with rapid maxillary expansion. *Am J Orthod* 77:500–507, 1980.
307. Turvey TA, Bell RB, Phillips C, et al: Self-reinforced biodegradable screw fixation compared with titanium screw fixation in mandibular advancement. *J Oral Maxillofac Surg* 64:40, 2006.
308. Turvey TA, Philips C, Zaytoun HS, et al: Simultaneous superior repositioning of the maxilla and mandibular advancement. *Am J Orthod Dentofacial Orthop* 94:372, 1988.
309. Van Sickels JE: A comparative study of bicortical screw and suspension wires versus bicortical screws in large mandibular advancements. *J Oral Maxillofac Surg* 49:1293, 1991.
310. Van Sickels JE, Dolce C, Keeling S, et al: Technical factors accounting for stability of bilateral sagittal split osteotomy advancement. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 89:19, 2000.
311. Van Sickels JE, Flanary CM: Stability associated with mandibular advancement treated by rigid osseous fixation. *J Oral Maxillofac Surg* 43:338–341, 1985.
312. Van Sickels JE, Richardson DA: Stability of orthognathic surgery: A review of rigid fixation. *Br J Oral Maxillofac Surg* 34:279, 1996.
313. Van Sickels JE, Tiner BD, Keeling SD, et al: Condylar position with rigid fixation versus wire osteosynthesis of a sagittal split advancement. *J Oral Maxillofac Surg* 57:31, 1999.
314. Vanarsdall RL: Transverse dimension and long-term stability. *Semin Orthod* 5(3):171–180, 1999.
315. Verlinden CR, Gooris PG, Becking AG: Complications in transpalatal distraction osteogenesis: A retrospective clinical study. *J Oral Maxillofac Surg* 69:899, 2011.
316. Vijayaraghaven K, Richardson A, Whitlock R: Post-operative relapse following sagittal split osteotomy. *Br J Oral Surg* 12:63, 1974.
317. Wardrop RW, Wolford LM: Maxillary stability following downgraft and/or advancement procedures with stabilization using rigid fixation and porous block hydroxyapatite implants. *J Oral Maxillofac Surg* 47:336–342, 1989.
318. Watzke IM, Tucker MR, Turvey TA: Lag screw versus position screw techniques for rigid internal fixation of sagittal osteotomies: A comparison of stability. *Int J Adult Orthodon Orthognath Surg* 6:19, 1991.
319. Watzke IM, Turvey TA, Phillips C, et al: Stability of mandibular advancement after sagittal osteotomy with screw or wire fixation: A comparative study. *J Oral Maxillofac Surg* 48:108, 1990.
320. Weinberg S, Taliano A, Taliano A: Condylar dislocation: An unusual complication observed after mandibular osteotomy. *Oral Surg Oral Med Oral Pathol* 56:581, 1983.
321. Welch TB: Stability in the correction of dentofacial deformities: A comprehensive review. *J Oral Maxillofac Surg* 47:1142–1149, 1989.
322. Wertz R, Dreskin M: Midpalatal suture opening: A normative study. *Am J Orthod* 71:367–381, 1977.
323. Wertz RA: Skeletal and dental changes accompanying rapid midpalatal suture opening. *Am J Orthod* 58:41–66, 1970.
324. Wessberg GA, Schendel SA, Epker BN: The role of suprahyoid myotomy in surgical advancement of the mandible via sagittal split ramus osteotomies. *J Oral Maxillofac Surg* 40:273, 1982.
325. Westermark A: LactoSorb resorbable osteosynthesis after sagittal split osteotomy of the mandible: A 2-year follow-up. *J Craniofac Surg* 10:519, 1999.
326. White RP, Jr, Peters PB, Costich ER, Page HL, Jr: Evaluation of sagittal split-ramus osteotomy in 17 patients. *J Oral Surg* 27:851, 1969.
327. Will LA, Joondeph DR, Hohl TH, et al: Condylar position following mandibular advancement: its relationship to relapse. *J Oral Maxillofac Surg* 42:578, 1984.
328. Will LA, West RA: Factors influencing the stability of sagittal split osteotomy for mandibular advancement. *J Oral Maxillofac Surg* 47:813, 1989.
329. Williams BJD, Currimbhoy S, Silva A, O'Ryan, FS: Complications following surgically assisted rapid palatal expansion: A retrospective cohort study. *J Oral Maxillofac Surg* 70:2394–2402, 2012.
330. Williams RL: Occlusal treatment for the postorthodontic patient, *Am J Orthod* 59:431, 1971.
331. Willmar K: On Le-Fort I osteotomy: A follow-up study of 106 operated patients with maxillo-facial deformity. *Scand J Plast Reconstr Surg* 12(0):suppl 12:1–68, 1974.
332. Wolford LM, Karras SC, Mehra P: Considerations for orthognathic surgery during growth: Part 1. Mandibular deformities. *Am J Orthod Dentofacial Orthop* 119:95, 2001.
333. Wolford LM, Karras SC, Mehra P: Considerations for orthognathic surgery during growth: Part 2. Maxillary deformities. *Am J Orthod Dentofacial Orthop* 119:102, 2001.
334. Worms FW, Spiedel TM, Bevis RR, Waite DE: Post treatment stability and aesthetics of orthognathic surgery. *Angle Orthod* 50(4):251–273, 1980.
335. Yaqoob O, Patel A, O'Neill J: Active postsurgical orthodontic treatment: How long does it take? *Br Orthod Soc Clin Effect Bull* 19:12, 2006.
336. Yashiro K, Takada K: Tongue muscle activity after orthodontic treatment of anterior open bite: A case report. *Am J Orthod Dentofacial Orthop* 115:660–666, 1999.
337. Zimring JF, Isaacson RJ: Forces produced by rapid maxillary expansion: III. Forces present during retention. *Angle Orthod* 35:178–186, 1965.

18

Grafts Frequently Used During Orthognathic Surgery and for Adjunctive Procedures

JEFFREY C. POSNICK, DMD, MD

- Bone Grafting: Definitions and Options
- Grafting Interpositional Defects and Gaps of the Mandible and the Maxilla
- Grafting Condyle and Ascending Ramus Defects
- Cartilage Grafting for Nasal Reconstruction
- Alloplastic Grafts and Implants for the Augmentation of the Facial Skeleton
- Conclusions

Bone Grafting: Definitions and Options

During maxillofacial surgery, grafting is a procedure that replaces missing structures with tissues from the patient's own body or with artificial, synthetic, or natural substitutes.[24,33,40,50,56,60,62,69-71,78,106,111,112] It is estimated that more than 500,000 bone-grafting procedures are performed annually in the United States, and these numbers easily double globally. Approximately half of the procedures that involve grafts relate to spinal fusion. The exact number of grafts used in head and neck reconstruction is not known, but it is significant.

Bone grafting is possible because bone—unlike most other biologic tissues—has the ability to regenerate completely if it is provided with space into which it can grow.[3-5,7,13,29,34,49,64,65,83,125-128] As native bone grows, it will generally replace any resorbable graft material in its path, which results in a fully integrated region of new bone. The biologic mechanisms that provide a rationale for bone grafting are osteoconduction, osteoinduction, and osteogenesis.

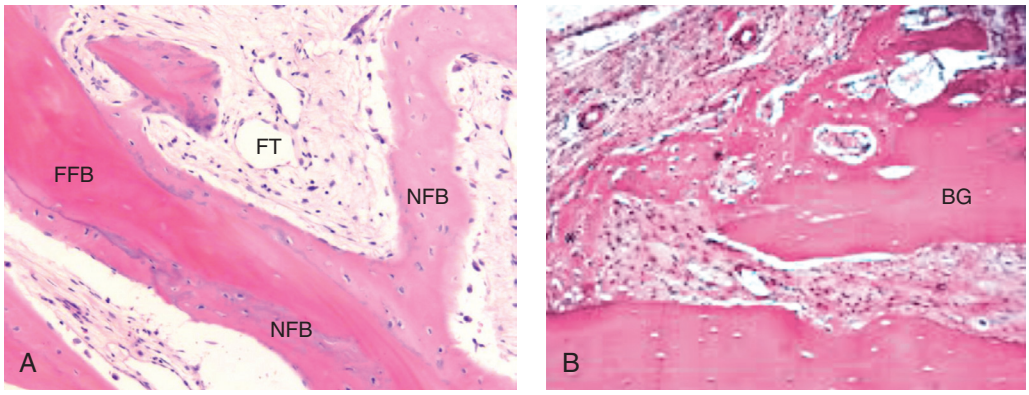
Osteoconduction occurs when the graft material serves as a scaffold that facilitates new growth from the native bone (Fig. 18-1, *A*). Osteoblasts from the margin of the defect use the graft material as a framework upon which to spread and generate new bone. The selected graft material for the management of a bone defect should at the very least be osteoconductive.

Osteoinduction involves the stimulation of osteoprogenitor cells to differentiate into osteoblasts that then begin new bone formation (Fig. 18-1, *B*). The most widely studied types of osteoinductive cell mediators are bone morphogenic proteins (BMPs). A bone graft material that is both osteoconductive and osteoinductive will serve as a scaffold for existing osteoblasts, and it will also stimulate the formation of new osteoblasts, thereby theoretically promoting the faster integration of the graft.

Osteogenesis occurs when vital osteoblasts that originate from the bone graft material itself contribute to new growth along with bone growth generated via the mechanisms of osteoinduction and osteoconduction. Fresh autogenous bone graft has the capability of supporting new bone growth by all three means (i.e., osteogenesis, osteoinduction, and osteoconduction). Because all three mechanisms may not be required at the particular recipient site to establish successful healing, it should be possible in many circumstances to select a non-autogenous graft, thereby avoiding the need for harvesting donor bone from the patient and still achieving success.

The sheer volume of bone grafting procedures carried out in the United States and throughout the world has created a relative shortage of available musculoskeletal donor tissue that is traditionally used for reconstructive autografts and allografts. This opportunity has stimulated corporate interest in supplying what is seen as a growing market for bone replacement materials. Currently, these graft alternatives are subjected to varying degrees of regulatory scrutiny; their true effectiveness in patients is not known until validated clinical trials are eventually carried out.

In general, it is known that osteoconductive materials work well when filling defects of non-critical size that would normally be expected to heal. However, when more



• **Figure 18-1 A,** Osteoconduction. A photomicrograph of a bone biopsy, where NFB surrounds the pre-existing bone and the residual of FFB. NFB presents features of mature bone, with well-organized lamellae and numerous small osteocytic lacunae. **B,** Osteoinduction. Peripheral region of the graft at 10 days. New bone formation, with primary bone tissue from the bone graft *(BG)* fragment and the parietal bone. **A** *from Acocella A, Bertolai R, Nissan J, Sacco R: Clinical, histological and histomorphometrical study of maxillary sinus augmentation using cortico-cancellous fresh frozen chips.* J Craniomaxillofac Surg *39:192–199, 2011.* **B** *from Esteves JC, Borrasca AG, Aranega AM, et al: Histomorphometric analysis of the repair process of autogenous bone grafts fixed at rat calvaria with cyanoacrylate.* J Appl Oral Sci *19:529–534, 2011.*

challenging defects are present, either fresh autologous bone graft or grafts with an osteoinductive agent appear to be necessary or are at least preferable for favorable healing.

Types of Bone Grafts

Bone Autografts

Fresh autogenous cancellous and corticocancellous bone are benchmark graft materials that allograft and bone substitutes attempt to match in their in vivo performance.[2,25,117-124] They incorporate all of the aforementioned properties, and they have no associated risk of viral transmission. They are incorporated into surrounding bone through creeping substitution. Unfortunately, the availability of an autograft for a specific need may be limited, and the harvest may be associated with donor site morbidity.[10]

Bone Allografts

The advantages of bone allografts that are recovered from deceased human donor sources include their availability in various shapes and sizes, the avoidance of the need to sacrifice host structures, and the lack of donor-site morbidity. Bone allografts are distributed through regional tissue banks and by a variety of reputable orthopedic medical device companies. The grafts are not without controversy, particularly because of their association with the transmission of infectious agents. It is essential to know the tissue bank provider's methods of handling the allografts to understand infectious disease issues and the potency of the graft material. Allografts should be compared with autogenous bone grafts with reference to their structural strength, osteoconduction, osteoinduction, and osteogenesis.

Bone Graft Substitutes

The ideal bone graft substitute is biocompatible, bioresorbable, osteoconductive, osteoinductive, structurally similar to bone, easy to use, and cost-effective. A growing number of bone graft alternatives are commercially available for orthopedic and, to a lesser extent, head and neck use. They are used in the reconstruction of cavitary bony deficiencies (i.e., interpositional defects) and for augmentation purposes.

Not all bone graft alternatives perform equally well. This presents a challenge to the orthognathic surgeon. It is important to appreciate the varied but repetitively seen graft-healing environments related to orthognathic surgery, including the following: (1) an interpositional defect at the chin osteotomy site; (2) an interpositional defect at the Le Fort I osteotomy site; (3) an interpositional defect at the ramus of the mandible osteotomy site; (4) the augmentation needs of the chin, the angle of the mandible, the zygomas, the perinasal area, and the infra-orbital rim; (5) a radiated tissue bed that requires osteotomy site healing; and (6) the proximity of the Le Fort I osteotomy site to the maxillary sinus. Each recipient environment will present with different graft-healing needs. Therefore, the validation of any bone graft alternative in one clinical site will not necessarily predict its performance in another location.

The clinician should seek a high burden of proof when using clinical or preclinical studies to justify the use of an osteoinductive graft material and the choice of one alternative over another. Only human trials can best determine the efficacy of a bone graft substitute and its site-specific effectiveness. Whenever feasible, surgeons should practice evidence-based medicine and tailor the treatment of their patient according to published medical literature and the levels of evidence claimed.

U.S. Food and Drug Administration approvals of bone graft substitutes for human use include the following:

The use of premarket-approved rh-BMP-2 (Infuse Bone Graft; Medtronic, Minneapolis, Minn) as an autograft

replacement for spinal fusion and the treatment of open tibia fractures. This substance has also been approved for maxillary sinus augmentation and localized alveolar ridge augmentation.

The use of rh-BMP-7 (OP-1 Implant; Olympus Biotech, Hopkington, Mass) as a humanitarian device. This substance has been approved as an autograft substitute for the non-union of long bones.

The use of rh-BMP-7 (Op-1 Putty; Olympus Biotech, Hopkington, Mass) as a humanitarian device alternative to autograft in compromised patients. This substance is approved for patients who require revision posterolateral lumbar spinal fusion or for those in whom autologous bone and bone marrow recovery (i.e., harvesting) are not feasible or are not expected to promote fusion.

The specific applications of bone substitutes, as described previously, have demonstrated clinically useful osteoinductive capabilities in humans. They have paved the way for broader clinical applications in the future. The recommended methods for the clinical application of these bone substitutes include direct placement into the surgical site, but the results have been more promising when the growth factors have been administered in combination with substrates to facilitate timed-release delivery or to provide a material scaffold for bone formation.

Please review the following salient points when considering the use of bone graft substitutes during orthognathic surgery:

- Fresh autogenous cancellous bone and, to a lesser extent, cortical bone are the benchmark graft materials. Their shortcomings include limited availability, donor site morbidity, and the potential for resorption.
- The advantages of allogenic bone include its availability in various sizes and shapes as well as the avoidance of host donor harvesting. The transmission of infection (particularly of human immunodeficiency virus) has been virtually eliminated as a concern when the grafts are properly treated.
- The ideal bone graft substitute is biocompatible, bioresorbable, osteoconductive, osteoinductive, structurally similar to bone, easy to use, and cost-effective. Bone substitute products are currently available on the market. They vary with regard to their composition and their claimed mechanism of action.
- U.S. Food and Drug Administration approvals of specific uses of recombinant human growth factors (i.e., rh-BMP-2 and rh-BMP-7) for the spine and the long bones have been made on the basis of demonstrated bone repair in human trials. Approved head and neck applications are limited but are likely to increase.
- The orthognathic surgeon has choices in the realm of bone grafting. Selection should be based on reasonable burdens of proof. Examining the products' claims and whether such claims are supported by preclinical and clinical studies for the specific sites to be used during clinical practice should be considered.

Grafting Interpositional Defects and Gaps of the Mandible and the Maxilla

Autogenous Iliac (Particulate Cancellous) Graft Donor Site (Video 8)

Iliac (hip) particulate cancellous bone remains the preferred graft material for the management of congenital cleft defects of the alveolus, the palate, and the floor of the nose.[1,9,12,15,26,28,46-48,52,63,66,77,82,90,115] This is especially true when the canine tooth is expected to erupt through the graft (i.e., cleft defect); when a tooth will be orthodontically moved into the grafted site; or when a dental implant will later be placed (Fig. 18-2). The cleft defect that requires a graft may be on one side (i.e., with unilateral cleft lip and palate) or on both sides (i.e., with bilateral cleft lip and palate). Previous attempts at grafting a bone deficiency may have failed as a result of an unsuitable graft material being selected; a soft-tissue deficiency or poor flap management; or inadequate postoperative management or patient cooperation (see Chapters 32 and 33).

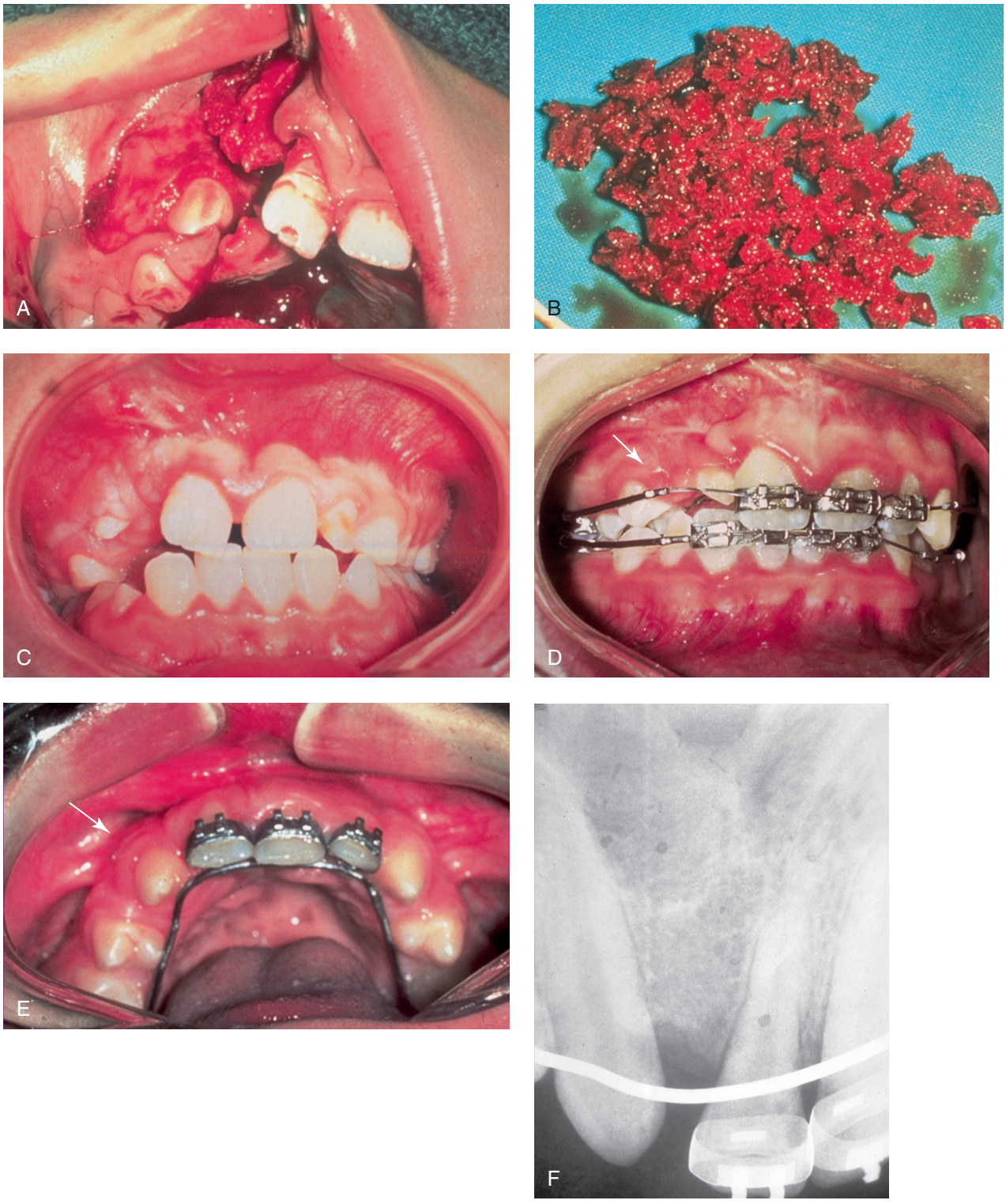
Donor site discomfort in the anterior iliac region is generally caused by the injury or contusion of the surrounding muscles on the medial (i.e., external oblique, internal oblique, and rectus abdominis) and lateral (i.e., medial gluteus) sides of the bone.* In a thin individual with a minimal fat layer and without baseline ankle, knee, or lumbar region arthritis or myalgia who is relatively young (i.e., <50 years), only minimum discomfort should occur. However, there are reports of complications associated with anterior iliac crest graft harvesting, including gait disturbance, paresthesia, superficial infections, hematoma, poor cosmesis, and chronic donor site pain.[6,20,41,42,53,57,81,92,103-105,110,113,114,131] The experienced surgeon can harvest the graft rapidly and efficiently. There is no need for the placement of a drain, minimal blood loss (i.e., <50 cc) is expected, and the head and neck region will have been simultaneously prepped and draped.

Step-by-Step Approach (Fig. 18-3)

Preparation and Draping

- An endotracheal tube is placed and secured in accordance with the patient's head and neck reconstruction needs.
- Ophthalmic ointment and corneal shields are placed for eye protection.
- The patient is supine on the operating room table.
- The patient's arm is tightly tucked to his or her side.
- Betadine solution is used for both hip and head and neck preparation.

*Reference 8, 10, 16, 21-23, 35, 36, 51, 54, 55, 58, 61, 72-75, 88, 100, 130, 143, 138



• **Figure 18-2** A child was born with unilateral cleft lip and palate and underwent arch expansion orthodontic treatment. This was followed by autogenous iliac bone grafting and fistula closure during the mixed dentition. **A,** Intraoperative view after the elevation of the mucogingival flap indicating the extent of the cleft defect. **B,** Harvested autogenous cancellous iliac graft used to fill the cleft defect. **C,** Occlusal view early after surgery. **D,** Later occlusal view with the early eruption of the canine. **E,** Occlusal and palate view after the eruption of the permanent canine through the bone graft. The canine and the other posterior teeth are orthodontically guided anteriorly to close the cleft and dental gap in the region of the congenitally missing lateral incisor. **F,** Periapical radiograph indicating normal alveolar ridge formation after the eruption of the canine through the bone-grafted cleft site.

Incision marked

Incision through skin

Scissor dissect to cartilage

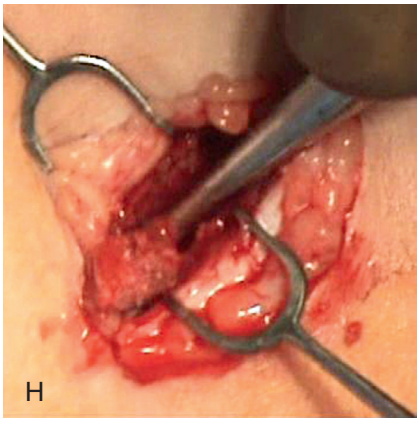
Incision through cartilage cap

• **Figure 18-3** Autogenous iliac (particularly cancellous) graft donor site harvesting. **A,** The planned incision is parallel and 1 cm lateral to the iliac crest. The incision starts 1 cm posterior to the anterior superior iliac spine. **B,** After the injection of local anesthesia, the incision is carried out through the skin. **C,** Scissors dissection continues through the subcutaneous tissue and the superficial and deep fascia while avoiding the cutting and contusion of the gluteus muscle laterally or the abdominal oblique and transverse abdominis muscles medially. **D,** With a knife, the cartilaginous cap is split down the center directly over the crest and down to the marrow cavity.

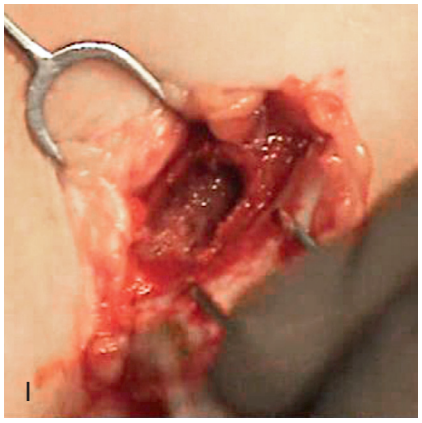
Incision down to marrow

Elevation of cartilage flap

Flaps curette



Curette used to remove cancellous bone

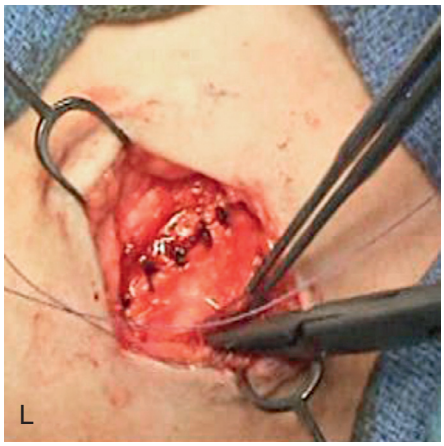


Defect in ilium

Cancellous bone harvested

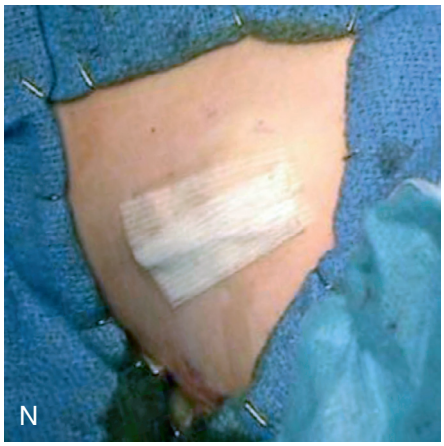
• **Figure 18-3, cont'd** **E,** With a knife, perpendicular relaxing incisions are completed through the cartilage at either end of the main incision. **F,** With a periosteal elevator, each half of the cartilaginous cap (i.e., medial and lateral) is elevated off of the underlying marrow space. **G,** The cancellous marrow bone is now exposed. **H,** Exposed cancellous bone is harvested with curettes. **I,** With the necessary cancellous bone harvested, a defect in the marrow space can be seen. **J,** Harvested cancellous marrow bone to be used for reconstruction.

Cartilage cap sutured



Fascial layer sutured

Skin closure



Dressing applied

• **Figure 18-3, cont'd K,** The cartilaginous cap is repositioned back in place with interrupted ties (3-0 Vicryl). **L,** The deep and superficial fascia layers are closed with interrupted suture ties (3-0 Vicryl). **M,** Subdermal closure is performed with interrupted suture ties (4-0 Vicryl). The skin is closed with subcuticular running suture (5-0 Monocryl). **N,** Steri-Strips and then an occlusive dressing (Tegaderm) are placed over the skin.

- At the hip, the patient is prepped halfway down the thigh, to the groin, across the abdominal midline, up to the rib cage, and posterior down the side of the buttock.
- Draping is completed, with limited exposed skin left around the planned incision site.

Surgical Markings

- The planned hip region incision (i.e., 3 cm) is located and marked with a surgical pen before prep and draping.
- The planned incision is parallel and 1 cm lateral to the iliac crest. The incision starts 1 cm posterior to the anterior superior iliac spine.

Local Anesthesia

- Xylocaine (1% with 1:100,000 epinephrine) is injected below the skin, directly over the surgical marking, and down to the periosteum over the iliac crest.

Skin Incision and Flap Elevation

- The incision is carried through the skin, the subcutaneous tissue, and the superficial and deep fascia directly over the iliac crest where the graft is to be harvested.
- The cutting or contusion of the gluteus muscle laterally or of the abdominal oblique and transverse abdominis muscles medially is to be avoided. Injury to these muscles when approaching the iliac crest should be avoided, because this is the primary cause of buttock and abdominal wall discomfort after surgery.

Graft Harvesting

Cartilage Cap Over Crest (Patient Typically <12 Years Old)

- If a cartilaginous cap remains over the crest, it is split down the center directly over the crest and down to the marrow cavity. Perpendicular relaxing incisions are completed through the cartilage at either end of the main incision. This allows for the lifting of each half of the

cartilage cap (i.e., medial and lateral) off of the underlying marrow space. The lifting of the vascularized cartilage flaps is accomplished with a periosteal elevator.
- The exposed cancellous bone is then harvested with curettes.

No Cartilage Cap Over Crest (Patient Typically >12 Years Old)

- When the iliac crest is mature, no cartilaginous cap remains. In this case, a window of cortical bone is removed from the medial aspect of the crest and down the medial plate for approximately 2 cm. This is an efficient method for the exposure of the medullary cavity.
- The subperiosteal dissection of the medial crest and down the medial plate is followed by the placement of a toed-out retractor down the medial plate.
- The removal of the cortex is facilitated with the following: 1) an oscillating saw with a short fan blade on a long shaft to cut through the cortex of the medial plate at the inferior aspect; 2) an oscillating saw with a wide fan blade on a short shaft to cut through the cortex of the medial crest; and 3) a reciprocating saw with a short, straight blade to cut through the cortices of the medial plate to connect the other two cuts.
- The removal of the window of cortical bone is then easily accomplished with the use of a chisel and a mallet.
- With the window of cortical bone removed, cancellous marrow is harvested with curettes.
- The removed cortical bone may also be chopped or used as an intact cortical graft. There is no advantage to replacing the small cortical bone segment, because the strength of the crest has not been compromised. The bone defect will not be visible or palpable through the closed skin wound. The removed marrow and cortical bone regenerate themselves.

Wound Closure

- After adequate graft is harvested, bone wax is used for hemostasis. The bone wax is not used to fill the cavity; rather, a thin layer is placed directly over the bleeding medullary surfaces. Interestingly, the wax does not prevent the regeneration of bone that will fill the cavity.
- If present, the cartilaginous cap is repositioned and sutured back into place with interrupted ties (3-0 Vicryl). If a cap is not present, then the overlying periosteum is closed with interrupted ties (3-0 Vicryl).
- The deep and superficial fascia layers are closed with interrupted suture ties (3-0 Vicryl).
- Subdermal closure is performed with interrupted suture ties (4-0 Vicryl).
- The skin is closed with subcuticular running suture (5-0 Monocryl).

Placement of Dressing

- The skin surface is cleaned with saline. It is then dried, and benzoin is applied.
- A limited number of Steri-Strips are placed directly over the closed wound.
- A small piece of cut gauze is placed directly over the Steri-Strips.
- An occlusive dressing (e.g., Tegaderm) is placed over the skin.

Postoperative Care

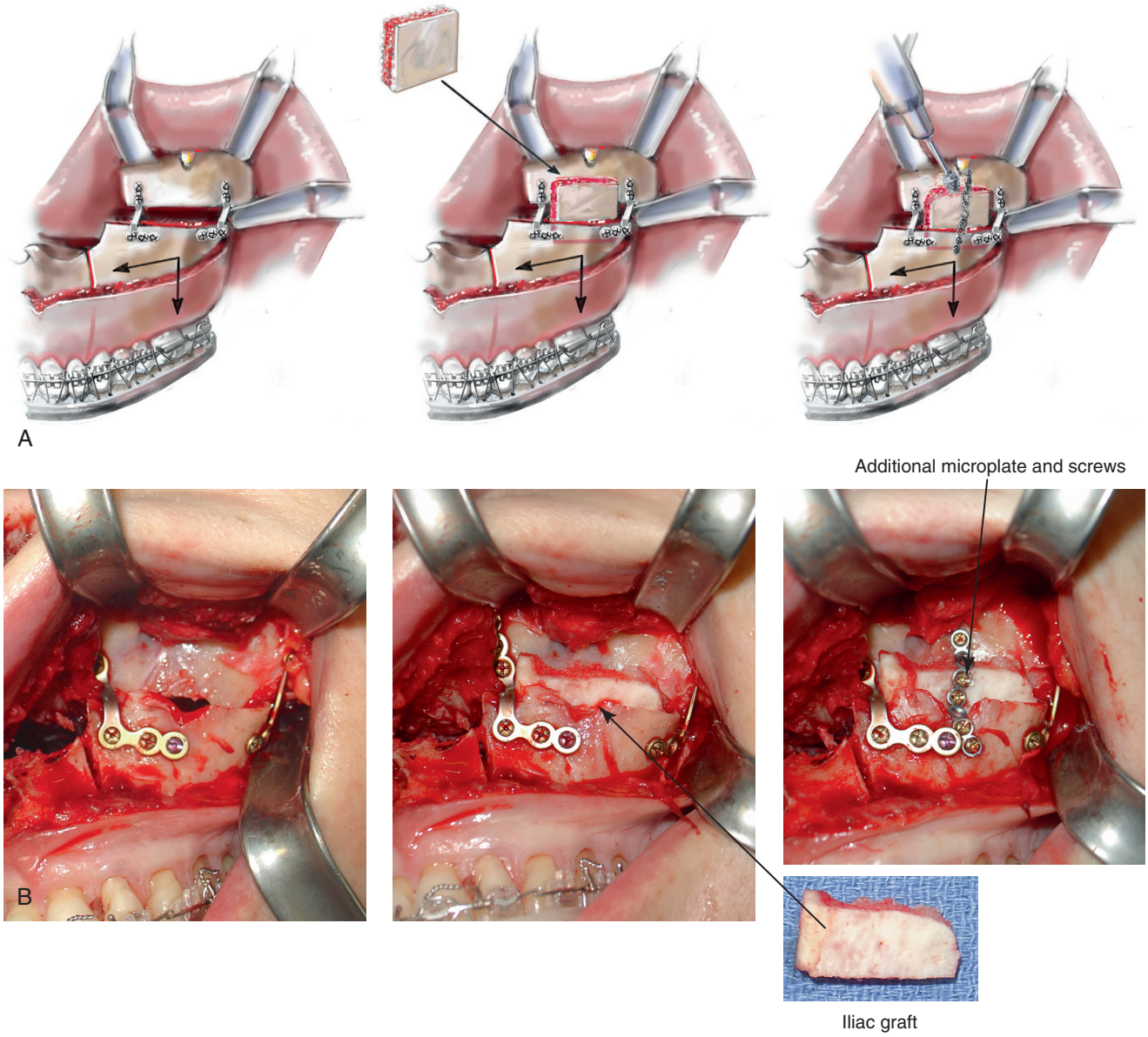
- The patient may shower without concern about moisture getting on the occlusive dressing that is covering the wound.
- The dressing remains intact for approximately 7 to 10 days.
- After the dressing is removed, no additional bandages are placed.

Autogenous Iliac (Corticocancellous Bloc) Graft Donor Site (Video 9)

When a Le Fort I osteotomy with significant horizontal advancement and vertical lengthening is carried out, an interpositional defect (i.e., dead space) is created. Despite rigid plate and screw fixation of the repositioned maxilla, inadequate bone contact may jeopardize successful healing (e.g., fibrous union; see Chapter 16) or leave the upper jaw prone to skeletal relapse (see Chapter 17). The advantage of placing an interpositional graft is sometimes obvious and at other times borderline. When an interpositional graft is deemed necessary, I prefer to use either autogenous or allogenic iliac bone.[107] A crafted corticocancellous anterior iliac (hip) graft is interposed between the pyriform and the zygomatic titanium plates that are used to secure the upper jaw in its new location (Fig. 18-4). The graft is tightly wedged between the advanced and lengthened anterior maxillary wall and the more posterior baseline maxillary wall. Any sharp edges of the inset graft are smoothed using a rotary drill with a watermelon bur. The graft is then fixed in place with an additional titanium plate and screws.

Another indication for a corticocancellous bloc graft is when a ramus osteotomy (e.g., an inverted L or straight horizontal osteotomy) is completed. After the proximal segment is seated with the condyle in the glenoid fossa (i.e., the terminal hinge position) and the distal mandible is secured to the maxillary teeth via intermaxillary fixation, a significant interpositional gap may remain. In these circumstances, a crafted corticocancellous iliac graft can be tightly interposed between the proximal and distal segments with additional cancellous bone packed into the remaining dead space. Rigid plate and screw fixation of the osteotomy segments and the graft is always required (see Chapter 28).

When an anterior maxillary or mandibular segmental alveolar defect requires reconstruction (e.g., after trauma or tumor resection), a crafted corticocancellous anterior iliac graft is generally preferable to other options. After successful graft healing (i.e., 4 to 6 months), dental rehabilitation including implant and crown placement may follow (see Chapter 35).[2]



• **Figure 18-4** When the extent of horizontal advancement and vertical lengthening of the maxilla after Le Fort I osteotomy are judged to be significant, then an interpositional bone graft may be required and beneficial for the achievement of precise healing. In these circumstances, I prefer to use either autogenous or allogenic iliac bone graft. A bloc of corticocancellous bone is crafted and tightly wedged into the gap on each side. Each graft is inset in between the pyriform rim and zygomatic buttress titanium plates. An additional plate is then contoured to extend from the anterior maxilla above, across the graft, and then onto the alveolar process of the anterior maxilla. Titanium screws (1.2 mm in diameter) are then used to secure the plate to the bone. **A,** Illustrations and **B,** intraoperative views of interpositional corticancellous iliac graft in a Le Fort I osteotomy site.

Donor site discomfort in the anterior iliac region is generally caused by the injury or contusion of the surrounding muscles on the medial (i.e., external oblique, internal oblique, and rectus abdominis) and lateral (i.e., medial gluteus) sides of the bone.* In a thin individual with a minimal fat layer and without baseline ankle, knee, or lumbar region arthritis or myalgia who is relatively young (i.e., <50 years old), only minimum muscle contusion and discomfort should occur. However, there are reports of complications associated with anterior iliac crest graft harvesting, including gait disturbance, paresthesia, superficial infections, hematoma, poor cosmesis, and chronic donor site pain.[6,20,41,42,53,57,81,92,103,104,105,110,113,114,131] The experienced surgeon can harvest the graft rapidly and effectively. In general, there is no need for the placement of a drain, minimal blood loss (i.e., <50 cc) is expected, and the head and neck region will have been simultaneously prepped and draped.

*Reference 8, 10, 16, 21-23, 35, 36, 51, 54, 55, 58, 61, 72-75, 80, 88, 100, 130, 143, 138

Step-by-Step Approach (Fig. 18-5)

Preparation and Draping

- An endotracheal tube is placed and secured in accordance with the patient's head and neck reconstruction needs.
- Ophthalmic ointment and corneal shields are placed for eye protection.
- The patient is supine on the operating room table.
- The patient's arm is tightly tucked to his or her side.
- Betadine solution is used for both hip and head and neck preparation.
- At the hip, the patient is prepped halfway down the thigh, to the groin, across the abdominal midline, up to the rib cage, and posterior down the side of the buttock.
- Draping is completed, with limited exposed skin left around the planned incision.

Surgical Markings

- The planned hip region incision (i.e., 3 cm to 4 cm) is located and marked with a surgical pen before preparation and draping.
- The planned incision is parallel and 1cm lateral to the iliac crest. It starts just posterior to the anterior superior iliac spine.

Local Anesthesia

- Xylocaine (1% with 1:100,000 epinephrine) is injected below the skin, directly over the surgical marking, and down to the periosteum over the iliac crest.

Skin Incision and Flap Elevation

- The incision is carried through the skin, the subcutaneous tissue, and the superficial and deep fascia directly over the iliac crest where the graft is to be harvested.
- The cutting or contusion of the gluteus muscle laterally or of the abdominal oblique and transverse abdominis muscles medially is to be avoided. Injury to these muscles when approaching the iliac crest is the primary cause of buttock and abdominal wall discomfort after surgery.

Graft Harvesting

- With the use of a knife (no. 15 blade), the periosteum is scored directly over the midline of the crest from just posterior of the anterior superior iliac spine. The incision continues posteriorly for a distance that is dependent on the extent of exposure required for the planned graft.
- Subperiosteal dissection of the medial crest and down the medial plate continues. A toed-out retractor is placed down the medial plate.
- The efficient removal of a bloc of corticocancellous bone graft is accomplished using an oscillating saw (with a short fan blade on a long shaft and a wide fan blade on a short shaft) and a reciprocating saw (with a short, straight blade) to cut through all four bordering cortical walls.
- The corticocancellous bloc graft is then removed from the medial aspect of the crest and down the medial plate. The length of the graft along the crest and its depth into the medullary cavity are variable, depending on the patient's reconstructive needs.
- The actual removal of the corticocancellous bloc is accomplished with the use of a 10-mm chisel and a twisting motion to fully separate the graft.
- With the bloc graft removed, additional cancellous marrow is harvested with curettes, as needed.
- The strength of the crest and of the ilium as a whole should not be compromised by the bloc graft removal. In general, the bone defect will not be detectable through the closed skin wound, because the lateral aspect of crest is preserved. The removed marrow and the cortical bone will regenerate, often with greater than normal thickness.

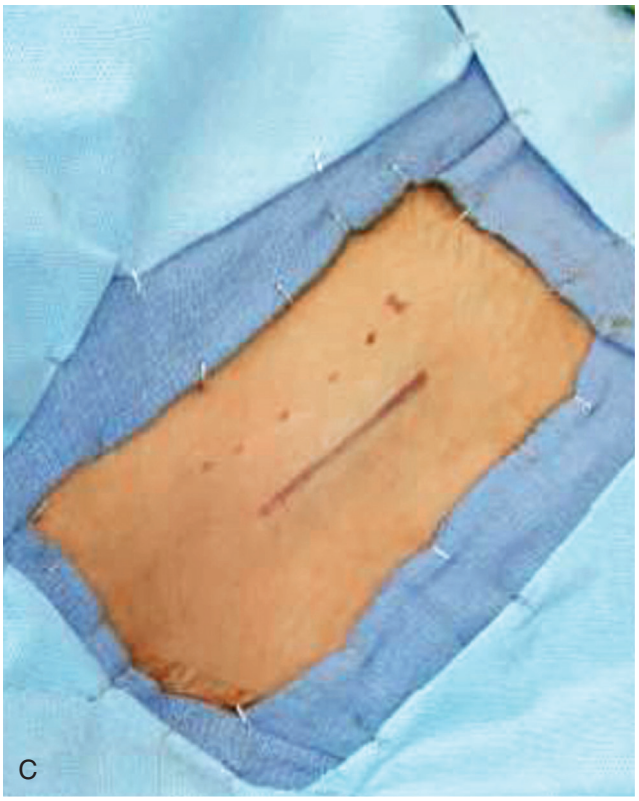
Wound Closure

- After adequate graft is harvested, bone wax is used for hemostasis. The bone wax is not used to fill the cavity; rather, a thin layer is placed directly over the bleeding medullary surfaces. The wax should not prevent bone regeneration.

Handheld instruments

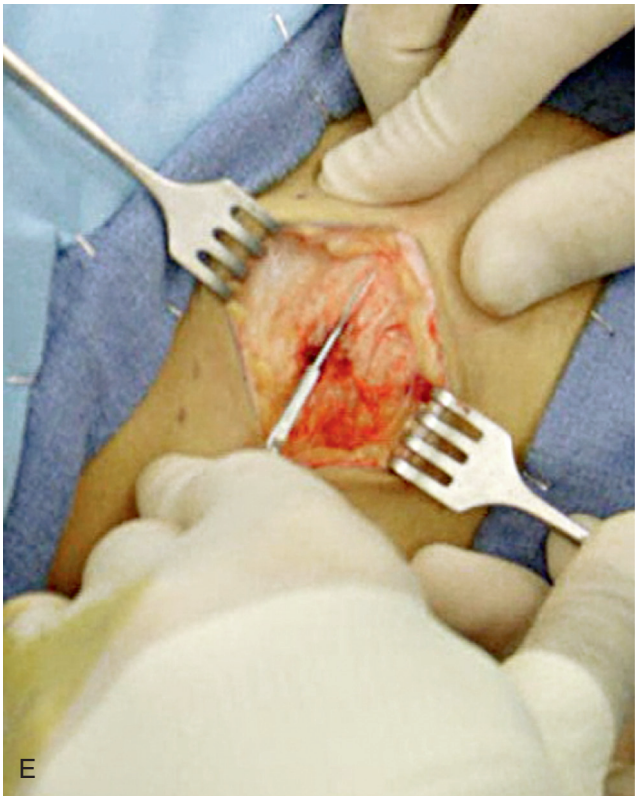
Incision marked and local anesthesia injected

• **Figure 18-5** Intraoperative views of the harvesting of autogenous iliac corticocancellous bone. **A,** The basic handheld instruments that are used to harvest the graft. **B,** The planned incision is parallel and lateral to the iliac crest. It starts just posterior to the anterior superior iliac spine. Local anesthesia is injected below the skin directly over the surgical marking and down to the periosteum over the iliac crest.



Incision marked

Incision through skin

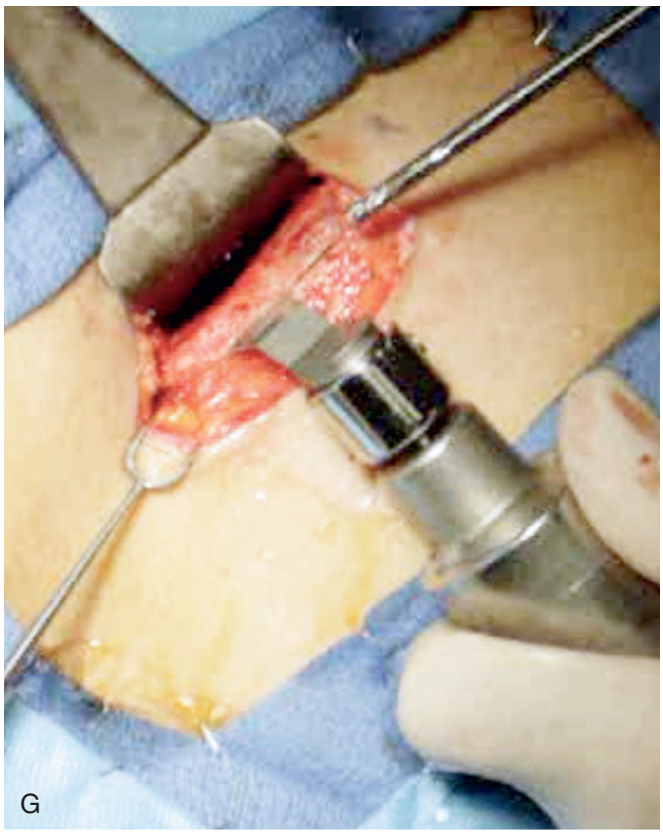


Flaps elevated and periosteum scored

Subperiosteal dissection of medial crest and plate

• **Figure 18-5, cont'd C,** The planned incision is shown. **D,** With a knife, the incision is carried through the skin and the subcutaneous tissue. The scissors dissection continues through superficial and deep fascia directly over the crest, where the graft is to be harvested. The contusion of the gluteus muscle laterally or the abdominal oblique and transverse abdominis muscles medially is avoided. **E,** With the use of a knife, the periosteum is scored directly over the midline of the crest from just posterior to the anterior superior iliac spine. The incision continues posteriorly for a distance that depends on the extent of exposure required for the planned graft. **F,** Subperiosteal dissection of the medial crest and down the medial plate continues.

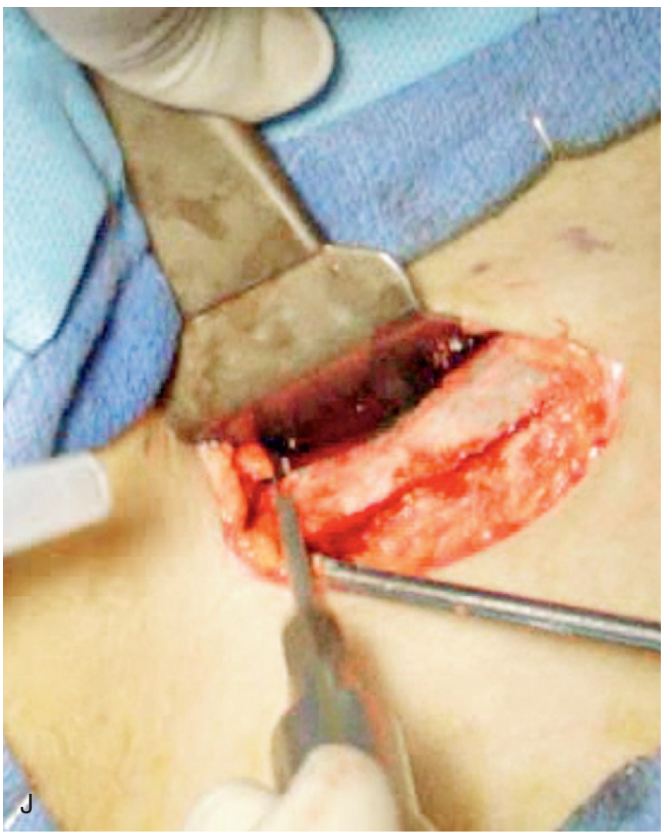
Continued



Oscillating saw through crestal cortex

Oscillating saw through lower cortex

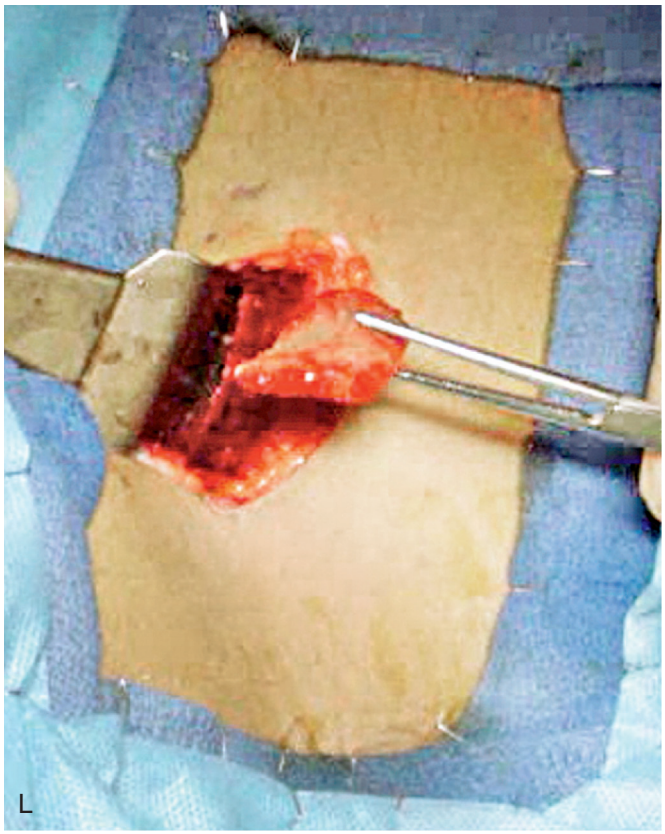
Reciprocating saw through third cortex wall



Reciprocating saw through final cortex wall

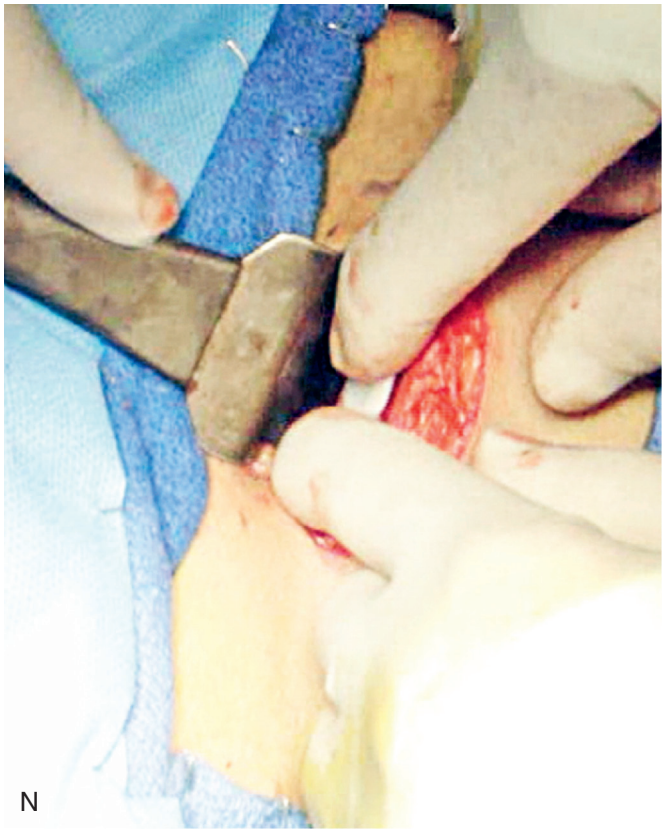
• **Figure 18-5, cont'd** **G,** A towed-out wide retractor is placed down the medial plate. The efficient removal of a bloc of corticocancellous bone graft is performed by first cutting through all four cortical wall borders. An oscillating saw with a wide blade on a short shaft is used to cut through the wall directly over the medial aspect of the crest. **H,** An oscillating saw with a short fan blade on a long shaft is next used to cut through the inferior cortical wall. **I,** A reciprocating saw with a short straight blade is used to cut through the lateral wall. **J,** A reciprocating saw with a short straight blade is used to cut through the last cortical wall.

Chisel used to release graft



Kocher clamp to remove graft

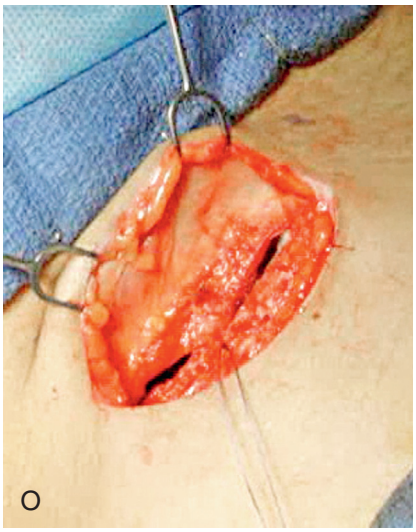
Curette to remove cancellous bone



Thin coat of bone wax for hemostasis

• **Figure 18-5, cont'd K,** The separation and removal of the corticocancellous block is accomplished with the use of a chisel that is 10 mm in width. After the chisel is in place, a twisting motion is carried out to fully separate the graft. **L,** The bloc graft is removed with a Kocher clamp. **M,** Additional cancellous marrow is harvested with curettes as needed. **N,** A thin coat of bone wax is placed onto the marrow walls for hemostasis.

Continued



Periosteum sutured

Suturing of skin

Dressing in place

• **Figure 18-5, cont'd** **O,** The periosteum is closed with interrupted suture ties (3-0 Vicryl). **P,** After closing the deep and superficial fascia layers with interrupted sutures (3-0 Vicryl), subdermal and then subcuticular closure are accomplished (5-0 Monocryl). **Q,** Steri-Strips and then an occlusive dressing (Tegaderm) are placed over the skin.

- The periosteum is closed with interrupted suture ties (3-0 Vicryl).
- The deep and superficial fascia layers are closed with interrupted suture ties (3-0 Vicryl).
- Subdermal closure is performed with interrupted suture ties (4-0 Vicryl).
- The skin is closed with subcuticular running suture (5-0 Monocryl).

Placement of Dressing

- The skin surface is cleaned with saline. It is then dried, and benzoin is applied.
- A limited number of Steri-Strips are placed directly over the closed wound.
- A small piece of cut gauze is placed directly over the Steri-Strips.
- An occlusive dressing (e.g., Tegaderm) is placed over the skin.

Postoperative Care

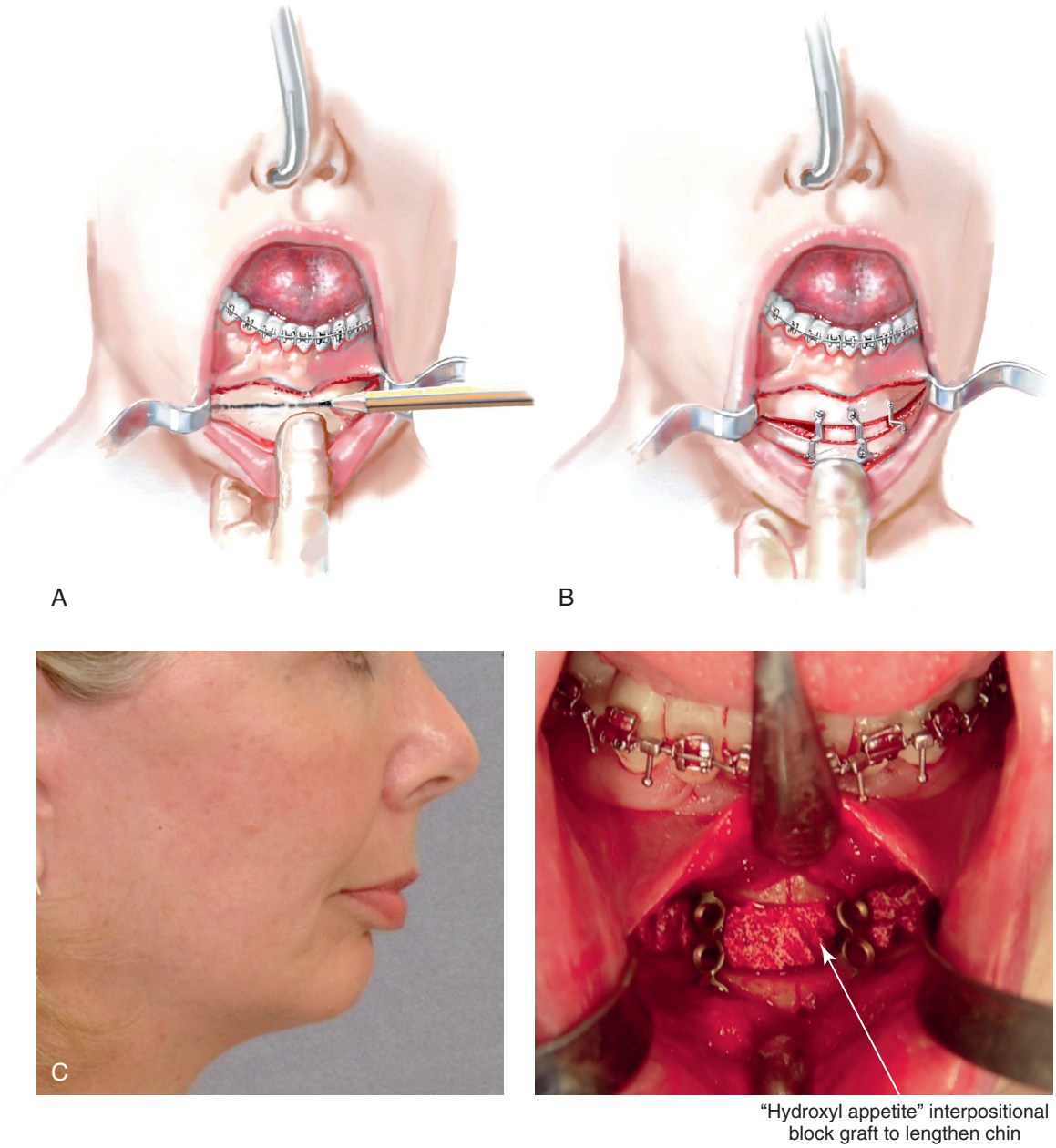
- The patient may shower without concern about moisture on the occlusive dressing that is covering the wound.
- The dressing remains intact for approximately 7 to 10 days.
- After the dressing is removed, no additional bandages are placed.

Alloplastic Graft (Porous Hydroxyapatite Bloc Implant)

When either a chin osteotomy with significant vertical lengthening or a Le Fort I osteotomy with significant horizontal advancement or vertical lengthening is carried out, an interpositional defect (i.e., dead space) is created. Despite rigid plate and screw fixation, inadequate bone contact across the osteotomy site may jeopardize successful healing (e.g., fibrous union) or leave the upper jaw or chin prone to skeletal relapse. When an interpositional graft is deemed necessary, I prefer to use either autogenous or allogenic anterior iliac crest. The disadvantage of an autogenous graft for this purpose relates to the potential for donor site morbidity. The use of porous bloc hydroxyapatite as an interpositional bone substitute in specific circumstances has been advocated by some.[11,18,27,30,87,93,94,98,95,97,129,134,139]

When bloc hydroxyapatite is used as an interpositional graft, histologic findings demonstrate fibrovascular and bone ingrowth into the interstices of the hydroxyapatite implants. Osteoid tissue is found in a diffuse fashion and interspersed between the calcified bone and the fibrous tissue. Osteoclastic activity is not demonstrated within the implanted bloc. Histologic examination of the bone–implant interface demonstrates no evidence of the resorption or remodeling of the bone. Medically packaged porous block hydroxyapatite is derived from specific marine corals that have a completely interconnected porous matrix with a pore size that averages 200 μm. The calcium carbonate skeleton is converted by the manufacturer to hydroxylated calcium phosphate. This mineral matrix is architecturally and chemically similar to the non-vascularized interstitial matrix of human cortical bone. Therefore, it provides a reasonable substitute scaffold for the potential ingrowth of fibrovascular tissue and bone. Unfortunately, the material has no intrinsic osteoinductive properties.

Coralline-derived porous bloc hydroxyapatite has been used successfully as an option when an interpositional graft is needed for chin-lengthening procedures (Fig. 18-6). Rosen and colleagues as well as other authors have published clinical reports of the use of this substance in the



• **Figure 18-6** In conjunction with an osseous genioplasty, if significant vertical lengthening is carried out, an interpositional graft (e.g., autograft, allograft, bloc hydroxyapatite) is crafted and placed in the gap. The graft fills the central gap in between the fixation plates. I do not find it necessary to place a graft in all of the lateral aspects of the gaps. An additional plate with screws is generally placed vertically in the midline across the osteotomy site directly over the graft. **A,** Illustration of a chin with vestibular incision exposure. The marking pencil indicates the proposed osteotomy. **B,** Illustration of osseous genioplasty with an interpositional graft. **C,** Profile view of a woman with a retrognathic mandible and a vertically short chin. Intraoperative view of the same patient indicating the completed osseous genioplasty with an interpositional hydroxyapatite graft.

Continued

• **Figure 18-6, cont'd D,** Profile views of the same patient shown before and after sagittal ramus osteotomies (horizontal advancement) and osseous genioplasty (vertical lengthening with interpositional hydroxyapatite graft). **E,** Oblique facial views before and after reconstruction (see Fig. 25-2).

above-mentioned manner with low rates of complications and rapid healing.[93-98] When used as an interpositional graft after Le Fort I osteotomy (as compared with at a chin osteotomy site), higher incidences of infection, sinus displacement, and extrusion have been reported and remain of concern.[139]

Allogenic (Human, Sterile, Freeze-Dried) Iliac (Corticocancellous Bloc) Graft

When either a chin osteotomy with significant vertical lengthening or a Le Fort I osteotomy with significant horizontal advancement or vertical lengthening is carried out, an interpositional defect (i.e., dead space) is created. Despite rigid plate and screw fixation, inadequate bone contact across the osteotomy site may jeopardize successful healing (e.g., fibrous union) or leave the upper jaw or chin prone to skeletal relapse. When an interpositional graft is deemed necessary, autogenous iliac bone offers theoretic wound-healing advantages, but it carries the potential for donor site morbidity. The use of allogenic iliac crest as an interpositional human bone substitute in these circumstances has been advocated and used successfully in clinical practice. With the use of current U.S. Food and Drug Administration and American Association of Tissue Banks standards and regulations, biomedical companies make sterile, freeze-dried, allogenic iliac crest bone available for use.

These sterile, freeze-dried allografts are processed from donated human tissue as a result of a gift from an individual or his or her family. The commercially available allografts

are generally processed with the use of patented and proprietary technology. The tissue is first disinfected and then terminally sterilized with the use of gamma radiation. The donor tissue should be screened and deemed suitable with reference to infectious disease test results, available donor medical history, and behavioral risks assessments. The physical assessment of the donor—including a review of relevant medical records and autopsy or coroner reports, if available—is preferred. Although few reports that critically evaluate the use of allogenic bone grafts specifically for orthognathic procedures are found in the literature, the successful use of these grafts for site-specific orthopedic procedures is well documented. In current clinical practice, when an interpositional graft is required at either the chin or the Le Fort I osteotomy site, I generally present both the autogenous and allogenic iliac crest graft options to the patient. If the patient is more than 50 years old or if he or she has hip, knee, or lumbar disc disease, I generally prefer the allogenic bone graft option. For a patient with a cleft maxilla that requires simultaneous fistula closure, I generally prefer autogenous bone. In most others circumstances, patient preference is a major factor.

Grafting Condyle and Ascending Ramus Defects

Autogenous Rib (Costochondral) Donor Site

When the condyle and portions of the ascending ramus have been removed (e.g., after tumor or trauma) or are congenitally absent (e.g., in a patient with hemifacial microsomia with Kaban type IIB malformation), there will be a limited number of reconstructive options to choose from, including an autogenous costochondral graft; an artificial total joint replacement; an autogenous vascularized fibular composite flap; or a sagittal split ramus osteotomy with proximal segment repositioning (see Chapters 27, 28, 35, and 36). For a patient who requires condyle and ascending ramus reconstruction, these options are not likely to give equivalent results. Clinical judgment and experience are required to assess the defect and the patient-specific factors (i.e., age, medical condition, quality of the recipient bed, and dental rehabilitation needs) before recommending the option that is best suited to the individual's reconstructive needs.

When a costochondral graft reconstruction is chosen, the harvesting of a portion of the sixth or seventh rib with a limited cartilaginous cap from the contralateral chest wall (of the recipient site) generally provides the best contour and morphology for the deficient mandible.

The use of an autogenous costochondral graft for the reconstruction of a deficient condyle and ascending ramus in an individual at or after skeletal maturity remains the author's preferred approach for patients with Kaban type IIB and type III mandibular malformation with hemifacial microsomia (see Chapter 28; Fig. 18-7). For a patient with a posttraumatic deformity (e.g., after condyle fracture), when the deficient condyle cannot be managed by the sagittal splitting the ramus of the mandible with proximal segment relocation into the glenoid fossa, then an autogenous costochondral graft is also generally the author's preferred approach (see Chapter 35). When the defect of the mandible or the glenoid fossa is more extensive and combined with soft-tissue hypoplasia or if the patient has undergone radiation therapy, then an autogenous vascularized crafted fibular composite flap is generally preferred (see Chapter 28). For an adult patient with a history of recurrent ankylosis, an artificial total joint replacement maybe the preferred recommendation.

Step-by-Step Approach

Preparation and Draping

- Nasotracheal intubation (i.e., RAE tube) is completed and secured in accordance with the patient's head and neck reconstruction needs.
- Ophthalmic ointment and corneal shields are placed for eye protection.
- The rib to be harvested is palpated and identified.
- Betadine solution is used for both chest wall and head and neck preparation.
- The patient is prepped below the rib cage, past the chest midline, superior to the nipple, and laterally to the midaxillary line.
- Draping is completed, with limited skin exposure left parallel to and on all sides of the planned incision.

Surgical Markings

- The planned incision (i.e., ≈4 cm) is located and marked with a surgical pen directly over the rib for harvesting before preparation and draping.

Local Anesthesia

- Xylocaine (1% with 1 : 100,000 epinephrine) is injected into the soft tissue below the planned incision and to the depth of the rib to be harvested.

Skin Incision and Flap Elevation

- With the use of a knife (no. 15 blade), the incision is carried through the skin, the subcutaneous tissue, the fascia, and the muscle directly over the rib to be harvested.
- Superior and inferior flaps are elevated to provide deep exposure of approximately 6 cm of bone and 2 cm of adjoining cartilage.

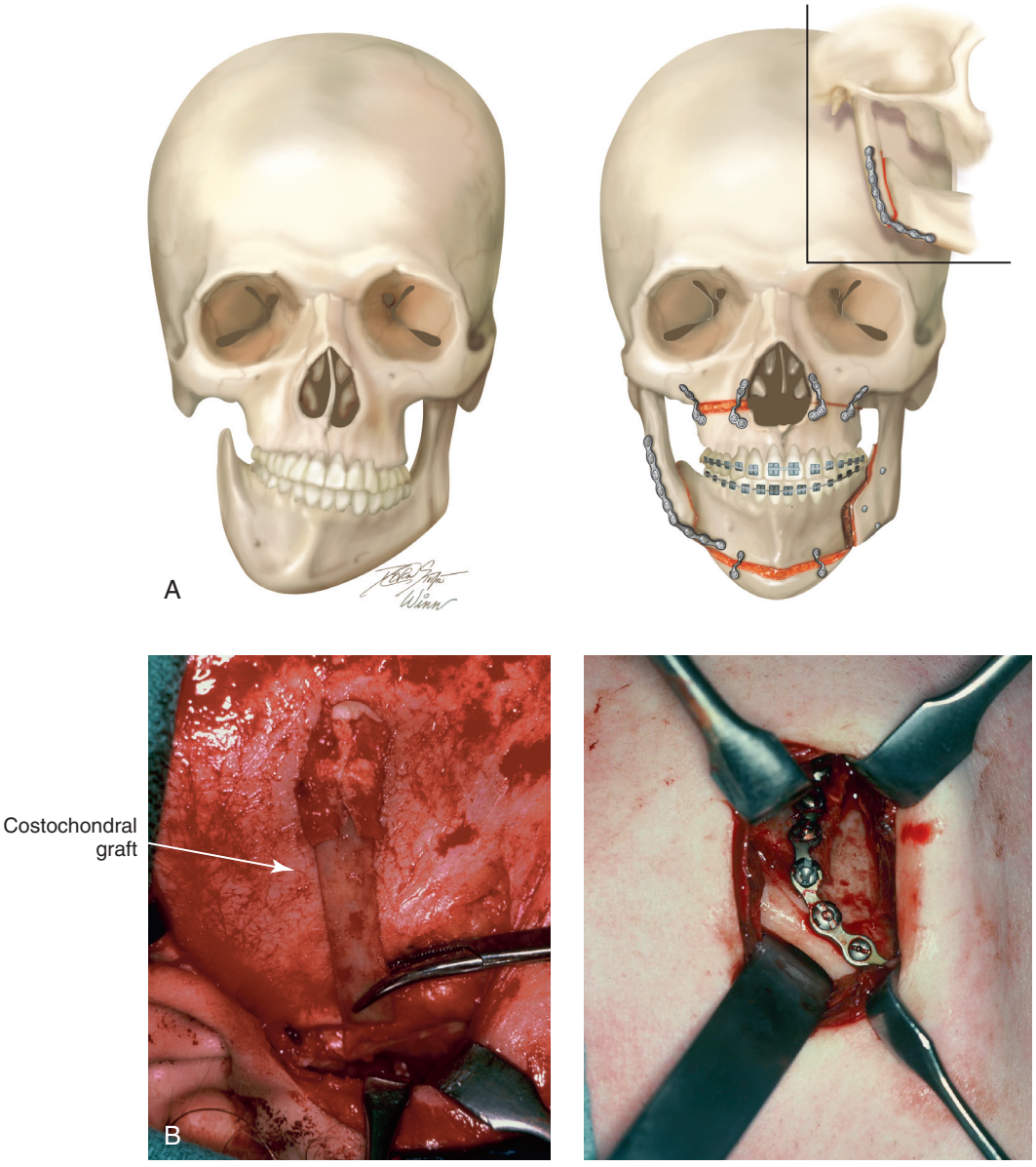
Graft Harvesting

- The periosteum is scored directly over the mid portion of the long axis of the rib to be harvested but not through bone or into the cartilaginous cap.
- Curved and straight elevators are used to dissect in the subperiosteal plane circumferentially around the rib to be harvested.

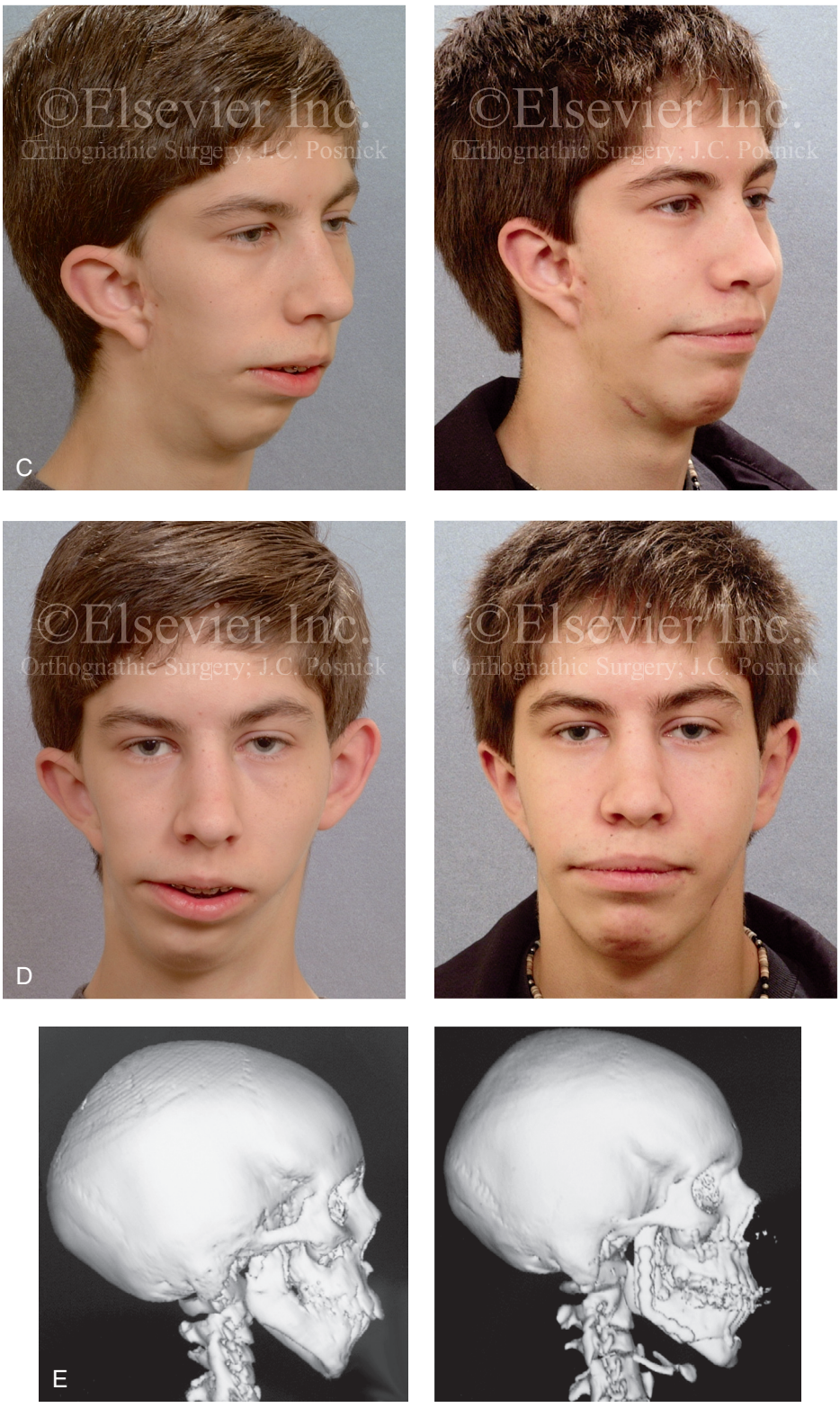
- A curved subperiosteal dissector (e.g., Doyen) is then inserted underneath and around the rib. This instrument is used to further strip and gain exposure in the subperiosteal plane, including underneath the cartilaginous cap.
- For protection, the retractor (e.g., Doyen) is placed deep and directly underneath the location for incising the cartilaginous cap.
- A knife (no. 15 blade) is then used to incise the full thickness of the cartilaginous cap, including not more than 1 cm of cartilage attached to the bone.
- The retractor (e.g., Doyen) is removed, and the rib cutter is inserted to cut and completely separate the rib according to the length of bone required for the purposes of reconstruction. The graft is removed.

Wound Closure

- The wound is checked for hemostasis and any exposure into the lung cavity; this is done by filling the wound with saline. The anesthesiologist introduces maximum end tidal volume and holds the area under pressure with the Valsalva maneuver. With the confirmation of no pneumothorax, wound closure is accomplished.
- The periosteal sleeve is approximated with interrupted suture ties (3-0 Vicryl).



• **Figure 18-7 A,** Illustrations of hemifacial microsomia (type IIB mandibular malformation) that show the presenting skeletal deformities and what the skull looks like after reconstruction is carried out. **B,** A patient has a costochondral graft inserted through a coronal (scalp) incision to reconstruct a condyle ascending ramus deficit. Stabilization of the graft occurs with an extended miniplate and screws placed through a Risdon (neck) incision. *Part A modified from an original illustration by Bill Winn.*



• **Figure 18-7, cont'd C,** Right oblique facial views of a teenaged boy with hemifacial microsomia (type IIB) before and after reconstruction. **D,** Frontal facial views before and after reconstruction. **E,** Profile computed tomography scan views of the same patient before and after reconstruction that demonstrate mandibular malformation and costochondral graft reconstruction.

- The deep and superficial fascia layers are closed with interrupted suture ties (3-0 Vicryl).
- Subdermal closure is performed with interrupted suture ties (4-0 Vicryl).
- The skin is closed with subcuticular running suture (5-0 resorbable).

Placement of Dressing

- The skin surface is cleaned with saline. It is then dried, and benzoin is applied.
- A limited number of Steri-Strips are placed directly over the closed wound.
- A small piece of cut gauze is placed directly over the Steri-Strips.
- An occlusive dressing (e.g., Tegaderm) is placed over the skin.

Postoperative Care

- A postoperative chest radiograph is used to confirm that there is no injury to the lungs.
- The patient may shower without concern about moisture on the occlusive dressing that is covering the wound.
- The dressing remains intact for approximately 7 to 10 days.
- When the dressing is removed, no additional bandages are placed.

Cartilage Grafting for Nasal Reconstruction

Over the last several decades of cosmetic rhinoplasty, the paradigm has shifted from aggressive reductive procedures to augmentation procedures. More attention is now focused on the use of grafts to augment deficiencies and the appropriateness of the graft material selected for each indication. When surgically altering the shape of the nose, two frequent reconstructive concerns are inadequate structural integrity and volume. These problems are generally best addressed with the use of cartilage grafts taken from a limited number of donor sources, such as the septum, the conchal bowl of the ear, or the rib cartilage. The choice of grafts and of the techniques used to place them remain both an art and science (see Chapter 38).[14,31]

Autogenous Nose (Septal) Cartilage Donor Site

Septal cartilage is generally reasonably straight and resilient; it provides structural support similar to that of the native nasocartilaginous framework. The septal cartilage can be contoured, carved, and sutured into an exact location. Significant resorption, infection, or warping is uncommon unless the cartilage is subjected to high-tension forces or crushing during preparation. Preferred uses for septal cartilage in nasal reconstruction often include caudal struts, lower lateral cartilage (LLC) crural grafts, spreader grafts, and tip grafts (e.g., shield grafts) to increase projection or definition (Fig. 18-8).[38,39]

A main disadvantage of the use of septal cartilage in nasal reconstruction is the limited amount that is often available. Anterior to the vomer and perpendicular plate of the ethmoid, septal cartilage can be harvested without concern, but always with the maintenance of the anterior structural support of the cartilaginous septum (i.e., adequate dorsal and caudal struts) to prevent dorsal or caudal collapse. In most individuals, unless the septal cartilage has been previously removed through submucous resection or is absent for other reasons (e.g., infection, congenital condition, ischemic necrosis), adequate cartilage can generally be harvested to serve as a caudal strut graft (i.e., to provide tip support); spreader grafts (i.e., to widen the nasal valves or to straighten the cartilaginous dorsum); LLC support (i.e., for the cleft or "pinched" nose); or as additional tip augmentation (see Chapters 29, 32, 33, 35, and 38).

Step-by-Step Approach (Fig. 18-9)

Preparation and Draping for Rhinoplasty

- Orotracheal intubation (i.e., RAE tube) is placed as required for the rhinoplasty procedure. The tube is taped to the chin to prevent dislodgement.
- Ophthalmic ointment and corneal shields are placed for eye protection.
- If the patient's hair is long, it is tied in a ponytail.
- Betadine solution is used for the preparation of the scalp, forehead, external ears, face, and neck. (Do not use soap, alcohol, Hibiclens, and so on.)

Draping is completed, with exposure of the forehead, the external ears, the face, and the neck down to the clavicles left in the operative field as per the routine for rhinoplasty.

Surgical Marking

The columella (stair-step) incision and the bilateral rim (marginal) intranasal incisions are located and marked with a surgical pen before prep and draping.

Local Anesthesia

- Xylocaine (1% with 1:100,000 epinephrine) is injected deep into the skin within the soft-tissue envelope of the nose as is routine for rhinoplasty.
- Xylocaine (1% with 1:100,000 epinephrine) is injected on each side of the septum, deep to the mucosa and superficial to the cartilage.
- Cocaine-soaked pledgets (5 cc of 4% solution) are placed in each nasal cavity.

Skin Incision and Flap Elevation

- The columella skin is incised, and the intranasal rim incisions are made.
- The nasal soft-tissue envelope is elevated with Stevens scissors as required for an open rhinoplasty procedure.

• **Figure 18-8 A,** Profile views of a teenaged girl before and after rhinoplasty with the use of a septal (caudal strut) cartilage graft to improve tip position and projection as described in Fig. 18-9. **B,** Oblique facial views before and after reconstruction.

Graft Harvesting

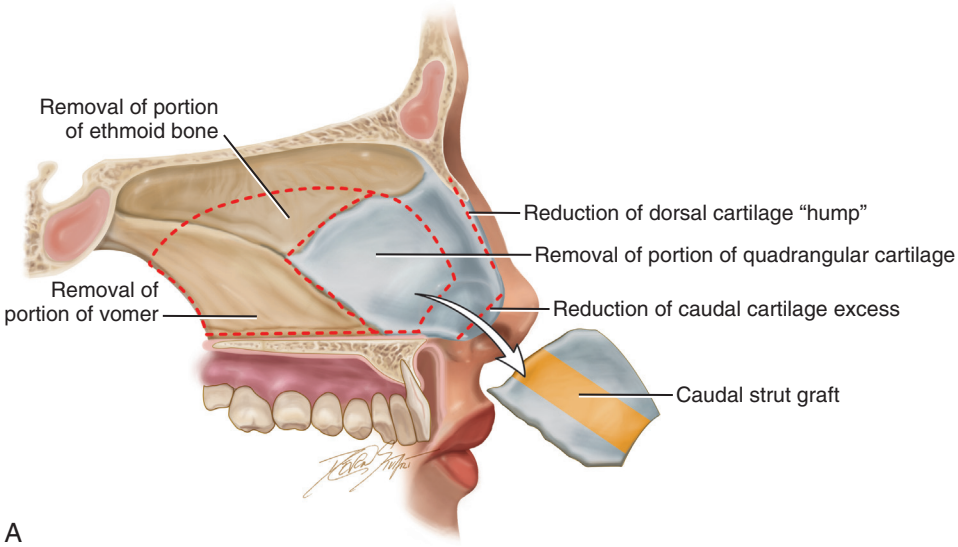
- Separate the lower lateral cartilages with Stevens scissors. Dissect down to and identify the anterior aspect of the caudal septal cartilage. Use a sharp periosteal elevator (e.g., Cottle) to initiate the submucosal dissection on each side of the caudal edge of the septal cartilage. Extend the submucosal dissection inferior to the maxilla and posterior to the vomer. Continue the subperiosteal dissection as required for the resection of the deviated bony septum.
- Use a knife (no. 15 blade) to initiate a caudal and dorsal septal incision through the quadrangular cartilage for the insertion of a swivel knife. The incision is located to maintain approximately 10 mm of caudal and 10 mm of dorsal septal cartilage.
- Insert the swivel knife, and incise the septal cartilage for graft harvesting and for submucous resection as indicated.
- Remove the incised septal cartilage graft.

Crafting of Graft

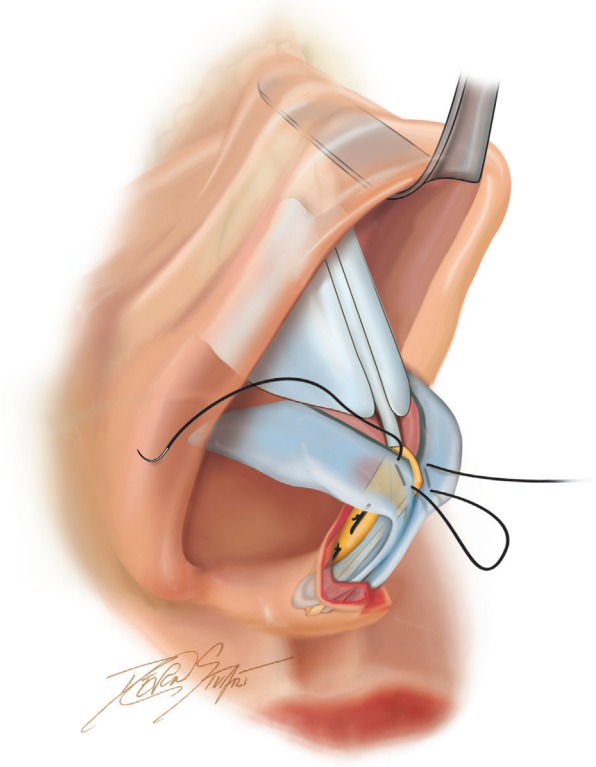
- Use a knife (no. 15 blade) to carve out and craft the required grafts (e.g., caudal strut, LLC reconstruction, spreader grafts, tip augmentation) from the full piece of harvested septal cartilage.

Wound Closure

- After the completion of the septorhinoplasty procedure, the closure of wounds is performed per routine rhinoplasty.

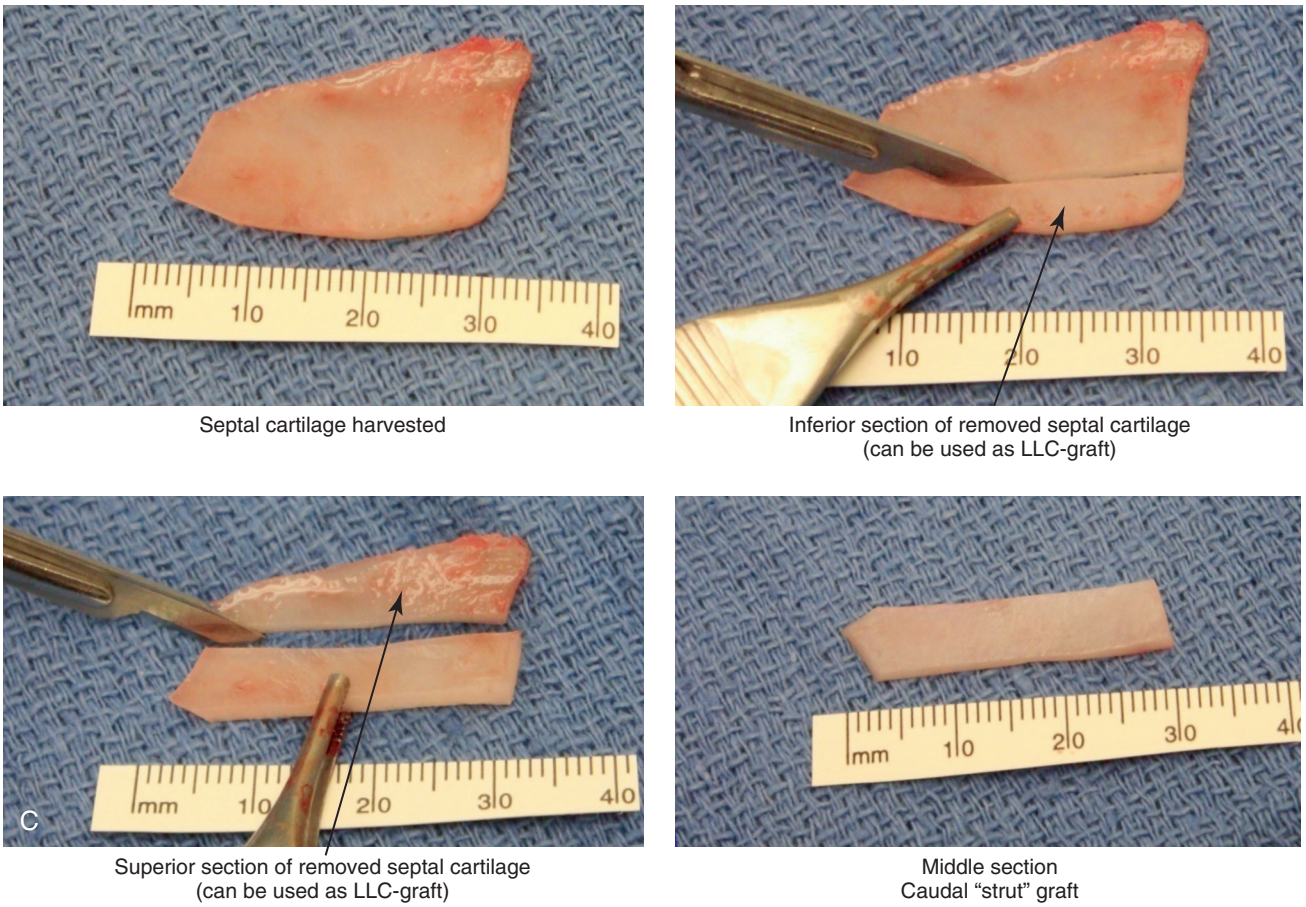


A



B Caudal strut graft sutured in place, lower lateral cartilages are then sutured together and over the top of the graft

• **Figure 18-9 A,** Illustrations that demonstrate the location of the harvesting of a septal cartilage graft and **B,** of the graft's placement as a caudal strut.



• **Figure 18-9, cont'd C,** Intraoperative views of removed septal cartilage that can be used for grafts to reconstruct the nose. The cartilage can be crafted as a caudal strut, spreader grafts, a lower lateral cartilage augmentation graft, and as tip onlay grafts.

- To approximate the columella skin (i.e., the stair-step incision), a single dermal suture is placed (5-0 Vicryl).
- Columella skin closure is performed with interrupted suture ties (6-0 nylon).
- Intranasal wound closure is performed with interrupted ties (5-0 chromic).

Placement of Dressing

- The placement of dressings is routine as per the septo-rhinoplasty procedure.
- Flexible splints (e.g., Reuter Type-Medtronic USA INC, Jacksonville, FL.) are generally placed on each side of the septum and secured together with a single suture tie (3-0 nylon).
- Antibiotic ointment (e.g., Bactroban) is generally injected into each nostril cavity.
- Packing (e.g., folded Telfa) is generally inserted equally into each nostril cavity.
- Tape (e.g., a Steri-Strip) is placed over dorsum of nose as per rhinoplasty procedure.
- A custom splint (e.g., Aquaplast) is molded over the dorsum of nose as per rhinoplasty.

Autogenous Ear (Conchal) Cartilage Donor Site

Conchal cartilage may be required for augmentation during secondary rhinoplasty when prior harvest, trauma, infection, or a genetic deficiency has rendered the nasal septal framework deficient and unavailable as a graft source.[76,79] In these cases, conchal cartilage may be the default option for non-structural nasal reconstruction applications. The conchal cartilage is pliable, resilient, flat, and thin. The conchal bowl may also have the natural contour to fit a specific nasal reconstructive need. Histologically, auricular cartilage is elastic hyaline. The flat base of the conchal bowl serves as the donor site and is harvested through a postauricular skin incision without leaving a noticeable aesthetic change in the external ear. The conchal bowl is composed of two components: the superior cymba and the inferior cavum. These are divided by the conchal extension of the helical root. The conchal bowl is bordered by the helical root and the external auditory meatus. Human anatomic dissection confirms the dimensions of the conchal bowl: it has a thickness of 1.9 mm to 4.4 mm; a maximum width of 1.9 cm to 2.9 cm; and a maximum height of 1.9 cm to

3.1 cm that can be harvested. During the harvesting of a conchal graft, the sidewalls are preserved to maintain the structural stability of the ear. A conchal graft can be useful for the reconstruction of the LLC (e.g., cleft nasal deformity, overresected iatrogenic deformity); spreader grafts (i.e., when minimal structural support is required); or nasal tip augmentation (i.e., when the underlying support is adequate). It is almost always a second or third donor site choice when nasal structural support is required, because it lacks sufficient strength to be used routinely as either a dorsum or caudal strut framework. In most cases, septal cartilage is preferred for these indications. For situations in which significant stretching of the overlying soft-tissue envelope is required to achieve tip projection (e.g., cocaine saddle deformity, bilateral cleft nasal deformity, Binder nasal deformity), a rib cartilage graft will best provide the needed framework support (see Chapters 29, 32, 33, 35, and 38).

Step-by-Step Approach

Preparation and Draping

- Orotracheal intubation (i.e., RAE tube) is placed as required for the rhinoplasty procedure. The RAE tube is taped to the chin to prevent dislodgement.
- Ophthalmic ointment and corneal shields are placed for eye protection.
- If the patient's hair is long, it is tied in a ponytail.
- Betadine solution is used for the preparation of the scalp, forehead, external ears, face, and neck. (Do not use soap, alcohol, Hibiclens, and so on.)
- Draping is completed, with exposure of the forehead, the external ears, the face, and the neck down to the clavicles left in the operative field as per the routine for rhinoplasty.

Surgical Marking

- The postauricular skin incision is located and marked with a surgical pen before preparation and draping.
- The incision is centered on the long axis of the ear from the superior helical rim to the lobe. Stop the incision approximately 1 cm short of the superior and inferior margins of the ear.

Local Anesthesia

- Xylocaine (1% with 1 : 100,000 epinephrine) is injected into the postauricular sulcus between the mastoid fascia and the ear cartilage.

Skin Incision and Flap Elevation

- With the use of a knife (no. 15 blade), the full thickness of the skin is incised along the surgical markings.
- Stevens scissors are used to elevate the medial postauricular flap. The incision is made on the surface of the postauricular cartilage and then down to the mastoid fascia.

Graft Harvesting

- The place at which the flat and curved portions of the conchal bowl meet is identified.
- The planned incision is marked to follow the natural curve of the conchal cartilage at the location at which the flat bowl begins.
- With a fresh knife (no. 15 blade), the full thickness of the conchal cartilage is incised at the surgical marking, without perforation through the anterior skin.
- With the use of a sharp periosteal elevator (e.g., Cottle), the incised cartilage is freed from the tightly attached overlying anterior skin along the flat portion of the conchal bowl.
- A decision about the volume of the conchal cartilage graft required is made.
- The conchal bowl is incised at the desired location to achieve graft requirements.
- Hemostasis and the integrity of the anterior auricular skin are confirmed.

Wound Closure

- Several interrupted sutures and then running and locking resorbable sutures are placed for skin closure (5-0 chromic). This prevents the need for postoperative suture removal.

Placement of Dressing

- A thick coat of antibiotic ointment is placed in the postauricular fold and over the anterior ear skin surface.
- A sheet of gauze (e.g., Xeroform) is placed in the postauricular groove and also on the anterior skin surface.
- Two or three pieces of dry "fluff" gauze are placed over the anterior surface of the ear.
- Gauze rolls (e.g., Kerlix) are used as a wrap to hold the fluff gauze and to apply gentle pressure. The gauze dressing is looped around the anterior neck and the forehead to secure it.
- An Ace wrap is loosely placed as an outer layer of the head and ear dressing.
- The whole dressing is removed without replacement 5 to 7 days later.

Postoperative Care

- After the ear dressing is removed, the patient is encouraged to shower and shampoo the hair, the external ears, the face, and the neck once or twice daily as per the rhinoplasty routine.
- The donor ear is dabbed dry (i.e., not rubbed) after washing.
- No vigorous sports activities are allowed during the early phase of healing (i.e., approximately 4 to 6 weeks) as per rhinoplasty.

Autogenous Rib (Cartilage Only) Donor Site (Video 10)

When significant structural support is required for either the caudal septum or dorsum of the nose within a tight soft-tissue envelope, neither conchal nor septal cartilage will generally be adequate. In these cases, autogenous rib

cartilage will frequently be the graft material of choice (Fig. 18-10) (see Chapters 29, 32, 33, 35, and 38).[37,59,68,84-86,108,109] Costal cartilage is available in abundance, it undergoes minimal postoperative resorption, and it is relatively easy to carve into a strut graft. Its perceived donor site harvesting difficulties often dissuade surgeons from choosing it; these include the need for a small scar on the anterior lower chest wall as well as concerns about pneumothorax and postoperative pain. With meticulous surgical technique, these potential disadvantages are generally overcome. However, somewhat unpredictable occasional graft warping that may jeopardize the long-term aesthetic results when used as either a dorsal or caudal strut remains a concern. Gibson's experimental studies provide the principles for understanding and managing cartilage warping.[31] He suggested the use of "balancing cross sectional carving along the long axis of the cartilage as a way to limit/prevent warping." The general approach when carving costal cartilage involves symmetrical removal from both sides and then using the central core of the cartilage for reconstruction and augmentation whenever feasible. In vitro studies suggest that the full distortion of the graft may be demonstrated within 30 minutes of carving. At a minimum, using Gibson's balanced carving principles and waiting 15 minutes before graft placement are likely to uncover warping tendencies in a majority of cases. We often use internal stabilization of the dorsal strut (and, less often, of the caudal strut) via the placement of a Kirschner wire (K-wire; no. 35

• **Figure 18-10** Facial views of a teenager who was born with unilateral cleft lip and palate before and after cleft jaw reconstruction followed by cleft rhinoplasty. The nasal reconstruction involved the use of a rib cartilage (caudal strut) graft as described in Figure 18-11. **A,** Profile facial views before and after reconstruction. **B,** Oblique facial views before and after reconstruction.

threaded) through the spine of the graft (see Chapters 29, 35, and 38).

Definitive rhinoplasty will be required for the reconstruction of the residual bilateral cleft lip nasal deformity at some point after the early teenage years. A "short" columella generally defines the presenting nasal deformity in the individual with bilateral cleft lip and palate (see Chapter 33). A crafted autogenous rib cartilage caudal strut provides the structural support to adequately stretch the columella skin and the soft-tissue envelope for favorable tip projection (see Fig. 18-10). The graft is stabilized to the base of the maxilla with a short, buried K-wire (no. 35 threaded). The LLCs are then sutured together over the top of the rib cartilage caudal strut to form the most superficial aspect of the tip (see Chapter 38).

For the reconstruction of the severe posttraumatic or congenital saddle nose deformity, rib cartilage often has advantages as compared with bone in that it restores at least a degree of springiness to the cartilaginous vault and tip; it is considered easier and less morbid to harvest; and it is not prone to resorption (see Chapter 35). A rib cartilage dorsal strut may extend from the radix to the nasal tip or just from the inferior aspect of the nasal bones to the tip. A K-wire may be inserted through the spine of the dorsal strut to prevent warping; this is done before graft inset. The dorsal strut is combined with a separate rib cartilage caudal strut, which extends from the base of the maxilla to the tip. A short buried K-wire secures the caudal strut to the base of the maxilla. The two grafts are then sutured together to form the new tip. The LLCs are sutured together and then over the top of the grafts to establish the most superficial aspect of the tip (see Chapter 38). The soft-tissue envelope is redraped over the reconstructed cartilaginous vault and tip.

For the correction of the cocaine-induced or iatrogenic saddle deformity of the nose, a rib cartilage graft is typically crafted into two struts (see Chapter 38). The first is a caudal strut that extends from the base of the maxilla to the new nasal tip. Assuming adequate height of the nasal bones (i.e., the osseous vault), the second graft (i.e., the dorsal strut) is inset deep and then flush with the bony dorsum. The dorsal strut extends to the new nasal tip. Each cartilage graft is secured in place at its point of insertion, and the grafts are then sutured to each other where they join at the tip. The LLCs are sutured together and then over the top of the grafts to form the most superficial aspect of the tip (see Fig. 18-9). The soft-tissue envelope is redraped over the reconstructed cartilaginous vault and tip (see Chapter 38).

Step-by-Step Approach (Fig. 18-11)

Preparation and Draping

- Orotracheal intubation (i.e., RAE tube) is placed as required for the rhinoplasty procedure. The RAE tube is taped to the chin to prevent dislodgement.
- The specific rib to be harvested is palpated and identified.
- Betadine solution is used for the preparation of both the hip and the head and neck. (Do not use soap, alcohol, Hibiclens, and so on.)
- The patient is prepped below the rib cage, past the chest midline, superior to the nipple, and laterally to the midaxillary line.
- Draping is completed, with limited skin exposure left parallel to and on all sides of the planned incision.

Surgical Markings

- The planned incision (i.e., ≈4 cm) is located and marked with a surgical pen directly over the rib cartilage to be harvested before preparation and draping.

Local Anesthesia

- Xylocaine (1% with 1 : 100,000 epinephrine) is injected into the soft tissues below the planned incision and to the depth of the rib cartilage to be harvested.

Skin Incision and Flap Elevation

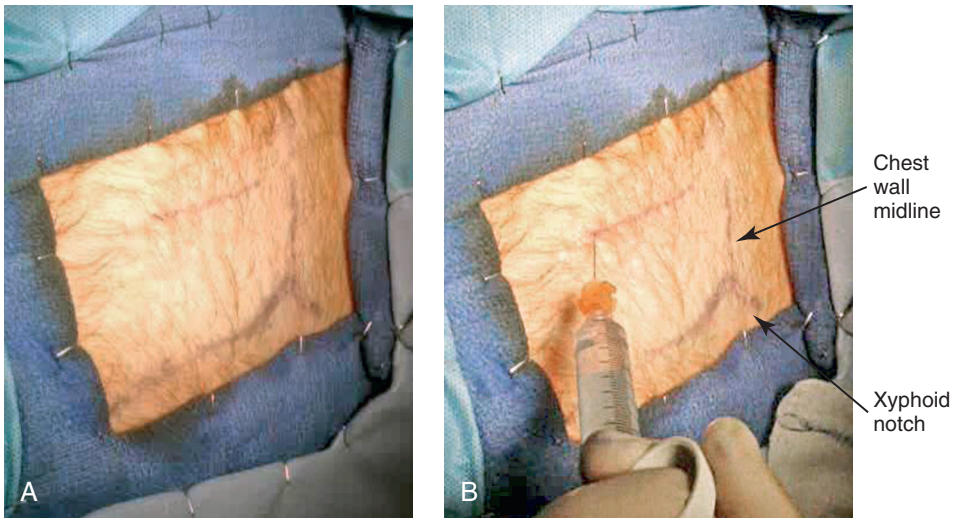
- With the use of a knife (no. 15 blade), the incision is carried through the skin, the subcutaneous tissue, and the fascia directly over the rib cartilage to be harvested.
- Superior and inferior flaps are elevated to provide access to the rib cartilage.
- This should provide exposure of approximately 4 cm of rib cartilage.

Graft Harvesting

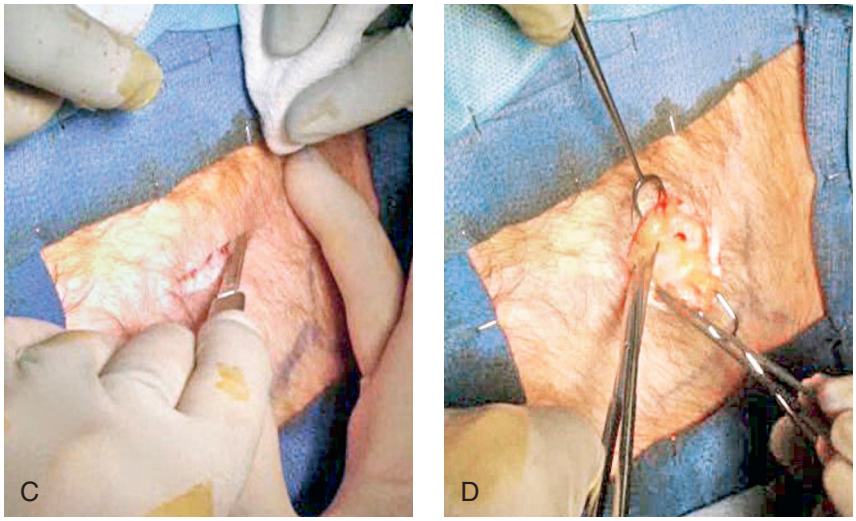
- The perichondrium is scored directly over the mid portion of the long axis of the rib cartilage to be harvested.
- Curved and straight elevators are used to dissect in the subperichondrial plane circumferentially around the cartilage to be harvested.
- A curved subperichondrial dissector (e.g., Doyen) is inserted underneath and around the rib cartilage. This instrument is used to gain further exposure in the subperichondrial plane.
- A retractor (e.g., Doyen) is placed deep and directly underneath the location for the incising of the cartilage graft. A knife (no. 15 blade) is then used to incise the full thickness of the cartilage.
- The retractor (e.g., Doyen) is then repositioned just below the other end of the cartilage at the location for incision. The knife (no. 15 blade) is used to incise the cartilage.
- The cartilage graft is removed.

Wound Closure

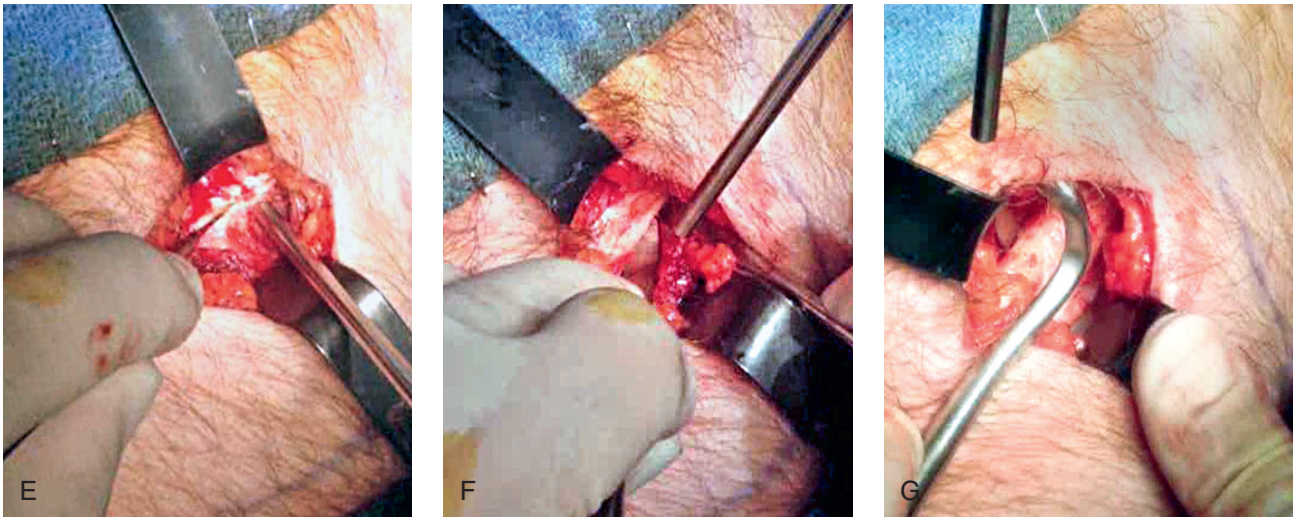
- The wound is checked for hemostasis and any exposure into the lung cavity; this is done by filling the wound with saline. The anesthesiologist introduces maximum end tidal volume and holds the area under pressure with the Valsalva maneuver. With the confirmation of no pneumothorax, wound closure is accomplished.



Incision marked | Local anesthesia injected



Skin incision | Scissors dissection down to cartilage



Perichondrium scored | Elevator used for subperichondrial dissection | Doyen used for subperichondrial dissection

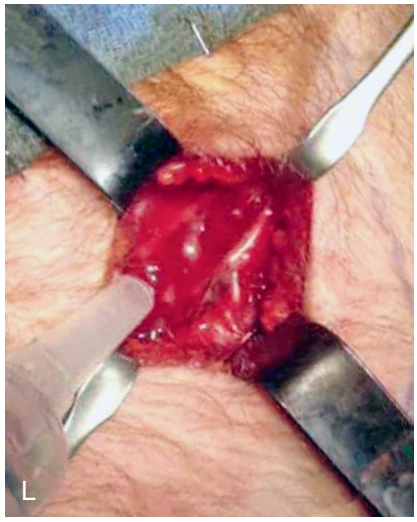
• **Figure 18-11** Intraoperative views of autogenous rib (cartilage only) donor site harvesting. **A,** The planned incision is marked directly over the rib cartilage to be harvested (≈4 cm incision length) before prepping and draping. **B,** Local anesthesia (Xylocaine 1% with epinephrine) is injected in the soft tissues below the skin and to the depth of the rib cartilage to be harvested. **C,** With a knife, the incision is carried through the skin and the subcutaneous tissue. **D,** Scissors dissection continues through the fascia directly over the rib cartilage. **E,** The perichondrium is scored with a knife directly over the mid portion of the long axis of the rib cartilage to be harvested. **F,** Curved and straight elevators are used to dissect in the subperichondrial plane circumferentially around the cartilage. **G,** A curved dissector (Doyen) is inserted underneath and around the rib cartilage. The Doyen is used to gain further exposure in the subperichondrial plane. *Continued*

Cutting proximal end of cartilage

Cutting distal end of cartilage

Cartilage graft

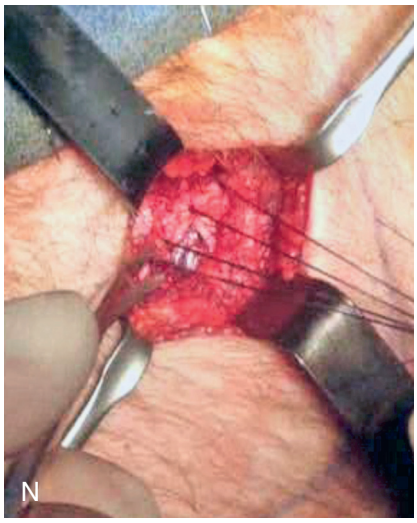
Defect checked for hemostasis



Water in wound prior to Valsalva maneuver

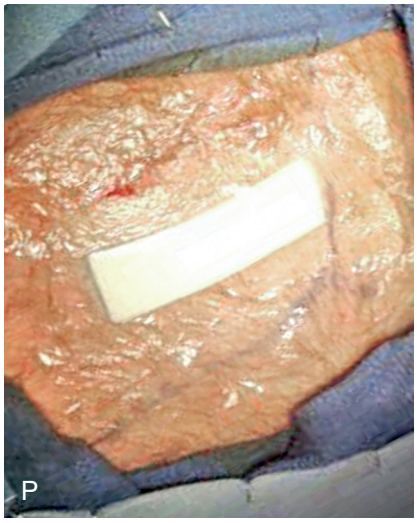
• **Figure 18-11, cont'd H,** A retractor is placed directly underneath the location for the full-thickness incision of the cartilage. A knife is then used to incise the full thickness of the cartilage on one end. **I,** A retractor is placed on the other end of the cartilage, and the knife is used to incise the cartilage. **J,** The cartilage graft is removed. **K,** After the removal of the cartilage graft, the defect is inspected for hemostasis. **L,** The wound is filled with saline, and the anesthesiologist introduces maximum end tidal volume and holds the area under pressure with the Valsalva maneuver. This confirms that no pneumothorax is present.

Perichondrial layer suture

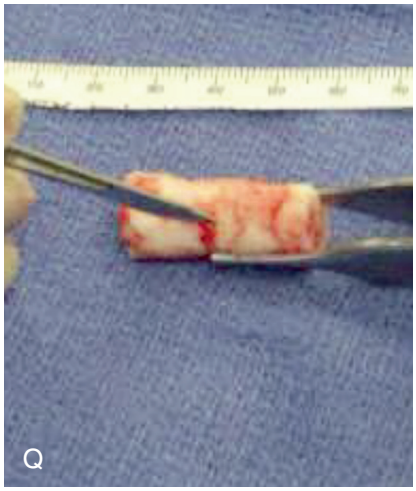


Fascia layer closure

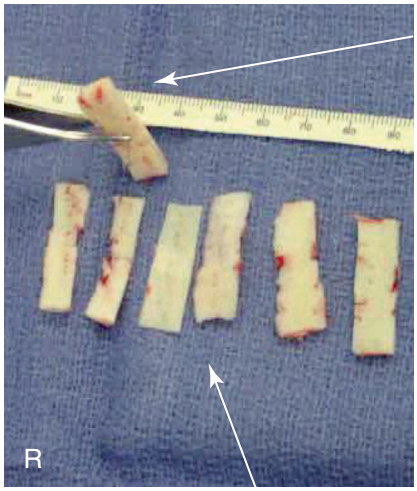
Skin closure



Occlusive dressing



Cartilage graft prior to crafting



Additional graft material crafted

• **Figure 18-11, cont'd M,** The perichondrial sleeve is approximated with interrupted suture ties (3-0 Vicryl). **N,** The deep and superficial fascia are closed with interrupted ties (3-0 Vicryl). **O,** Subdermal closure and then subcuticular closure are accomplished (5-0 Monocryl). **P,** Steri-Strips and an occlusive dressing (Tegaderm) are placed over the skin. **Q,** The harvested rib cartilage is crafted to suit the purposes of the recipient site. **R,** A crafted caudal strut graft can be seen. Additional grafts have also been crafted that can be used to reconstruct specific deficits (i.e., lower lateral cartilages, additional tip grafts, dorsal grafts).

- The perichondrial sleeve is approximated with interrupted suture ties (3-0 Vicryl).
- The deep and superficial fascia layers are closed with interrupted suture ties (3-0 Vicryl).
- Subdermal closure is with interrupted suture ties (4-0 Vicryl).
- The skin is closed with subcuticular running suture (5-0 resorbable).

Placement of Dressing

- The skin surface is cleaned with saline. It is then dried, and benzoin is applied.
- A limited number of Steri-Strips are placed directly over the closed wound.
- A small piece of cut gauze is placed directly over the Steri-Strips.
- An occlusive dressing (e.g., Tegaderm) is placed over the skin.

Postoperative Care

- The patient may shower without concern about moisture on the occlusive dressing that is covering the wound.
- The dressing remains intact for approximately 7 to 10 days.
- When the dressing is removed, no additional bandages are placed.

Alloplastic Grafts and Implants for the Augmentation of the Facial Skeleton

Porous Polyethylene Augmentation Implants

Specific patterns of skeletal dysmorphology may lead the individual to request and the surgeon to agree to augmentation and camouflage procedures for the sole purpose of enhancing facial aesthetics.[17,33,67,98,116,132,133,135,137] To avoid the need for graft harvesting and to limit concerns about the resorption of the graft over time, consideration of the use of alloplastic synthetic materials remains attractive. Alloplastic materials have been used to enhance and augment the facial skeleton over the years have included silicon, Biocoral, Biofax, Proplast, Teflon, Plasti-Pore, Seramic, polyamide-mesh, hydroxyapatite, Gore-Tex, methyl methacrylate, and porous polyethylene. These materials have been used with varying levels of success.[18,19,30,32,33,43-45,89,91,99,101,102,136] Their intrinsic biomedical properties generally fall short of ideal, but they may be adequate in specific clinical settings.

Of the currently available alloplastic synthetic implants that are commercially available, the materials that are seemingly most favorable for the augmentation of the facial skeleton are those made of porous polyethylene. Histologically, fibrous ingrowth into the polyethylene pores without capsule formation represents the key attribute of this particular material. The implants are placed in the subperiosteal plane and then preferably fixed in their new location to the underlying skeleton with titanium screws. Systemic antibiotics are administered perioperatively to limit the risk of infection. Technique-sensitive decisions regarding favorable sites for implant placement, preferred incision locations, extent of soft-tissue dissection, the size and shape of the implant selected, the crafting of the implant, the method of fixation, and postoperative wound management are all important factors for the limiting of morbidity and the achievement of desired results. Alloplastic implant extrusion, migration, infection, underlying bone resorption, and unsatisfactory aesthetic results all remain of concern.[142,140,141]

Augmenting the skeleton to camouflage facial dysmorphology with the use of porous polyethylene implants has been championed by Yaremchuk.[140-142] That author and others have demonstrated the effectiveness of this alloplastic graft approach for craniofacial reconstructive procedures and for purely facial aesthetic augmentation in specific clinical settings. The use of these implants in load-bearing locations as interpositional grafts or for purposes of nasal augmentation have consistently fallen short of the mark.

Conclusions

As part of maxillofacial surgery, grafting is a procedure that replaces missing structures with material from the patient's own body or with an artificial, synthetic, or natural substitute. When structures are deficient in the individual with a maxillofacial deformity, either additional bone or cartilage may be required. The grafting of interpositional defects or gaps of the mandible and maxilla is performed with autogenous bone, allogenic grafts, or alloplastic materials. The replacement of a condyle or an ascending ramus is generally done with autogenous costochondral graft, but vascularized fibular composite flaps and alloplastic joint replacements are additional options to consider. Nasal reconstruction is generally carried out with autogenous cartilage (e.g., septum, ear, rib) or, in some cases, autogenous bone. The use of alloplastic grafts for the augmentation of the facial skeleton is occasionally indicated. With adherence to basic biologic and aesthetic principles, an understanding of the individual's reconstructive and aesthetic needs, the development of a systematic treatment plan, and the maintenance of meticulous surgical technique, grafts can generally be used successfully to solve complex maxillofacial problems.

References

1. Abramowicz S, Katsnelson A, Forbes PW, Padwa BL: Anterior versus posterior approach to iliac crest for alveolar cleft bone grafting. *J Oral Maxillofac Surg* 70:211–215, 2012
2. Acocella A, Bertolai R, Nissan J, Sacco R: Clinical, histological and histomorphometrical study of maxillary sinus augmentation using cortico-cancellous fresh frozen chips. *J Craniomaxillofac Surg* 39:192–199, 2011
3. Albee F: *Bone graft surgery*, Philadelphia, 1915, Saunders, pp 40–85.
4. Albee F: The inlay bone graft versus Lane plates in the treatment of fractures. *Am J Surg* 29:77, 1915
5. Albee F: A statistical study of 539 cases of Pott's disease treated by bone graft. *Am J Orthop Surg* 14:134, 1916.
6. Arrington ED, Smith WJ, Chambers HG, et al: Complications of iliac crest bone graft harvesting. *Clin Orthop Relat Res* 329:300, 1996
7. Axhausen G: Die histologischen und klinischen gesetze der frien osteoplastik auf grund von teierversuchen. *Arch Klin Chir* 88:23, 1909.
8. Banwart JC, Asher MA, Hassanein RS: Iliac crest bone graft harvest donor site morbidity: A statistical evaluation. *Spine* 20:1055–1060, 1995.
9. Baqain Z, Anabtawi M, Karaky A, Malkawi Z: Morbidity from anterior iliac crest bone harvesting for secondary alveolar bone grafting: An outcome assessment study. *J Oral Maxillofac Surg* 67:570–575, 2009.
10. Barone A, Ricci M, Mangano F, Covani U: Morbidity associated with iliac crest harvesting in the treatment of maxillary mandibular atrophies: A 10-year analysis. *J Oral Maxillofac Surg* 69:2298–2304, 2011.
11. Bojescul JA, Polly DW, Jr, Kuklo TR, et al: Backfill for iliac-crest donor sites: A prospective randomized study of coralline hydroxyapatite. *Am J Orthop* 34:377–382, 2005.
12. Boyne PJ, James RA: Grafting of the maxillary sinus floor with autogenous marrow and bone. *J Oral Maxillofac Surg* 38:613–616, 1980.
13. Brunski JB, Puleo DA, Nanci A: Biomaterials and biomechanics of oral and maxillofacial implants: Current status and future developments. *Int J Oral Maxillofac Implants* 15:15, 2000.
14. Clark JM, Cook TA: Immediate reconstruction of extruded alloplastic nasal implants with irradiated homograft costal cartilage. *Laryngoscope* 112(6):968–974, 2002.
15. Cohen M, Figueroa AA, Haviv Y, et al: Iliac versus cranial bone for secondary grafting of residual alveolar clefts. *Plast Reconstr Surg* 87:423–428, 1991.
16. Consolo U, Politi M, Niconi PF, Salgarelli A: Prelievi d'osso da cresta iliaca. Revisione della casistica e valutazioni clinico-statistiche. *Minerva Stomatol* 39:133–137, 1990.
17. Converse JM: Restoration of facial contour by bone grafts introduced through the oral cavity. *Plast Reconstr Surg* 6:295, 1950.
18. Cottrell DA, Wolford LM: Long-term evaluation of the use of coralline hydroxyapatite in orthognathic surgery. *J Oral Maxillofac Surg* 56:935, 1998.
19. Davis PKB, Jones SM: The complications of silastic implants: Experience with 137 consecutive cases. *Br J Plast Surg* 24:405, 1971.
20. Dawson KH, Egbert MA, Myall RW: Pain following iliac crest bone grafting of alveolar clefts. *J Craniomaxillofac Surg* 24:151, 1996
21. De La Torre JI, Tenenhaus M, Gallagher PM, Sachs SA: Harvesting iliac bone graft: Decreasing the morbidity. *Cleft Palate Craniofac J* 36:388–390, 1999.
22. De Riu G, Meloni SM, Raho MT, et al: Delayed iliac abscess as complication of an iliac bone graft in an orthognathic case. *Int J Oral Maxillofac Surg* 37:1156–1158, 2008.
23. Dingman RO: The use of iliac bone on the repair of facial and cranial defects. *Plast Reconstr Surg* 3:24, 1950.
24. Doyen E: *Technique chirurgicale*, Paris, 1897, Masson et Cie, p 244.
25. Esteves JC, Borrasca AG, Aranega AM, et al: Histomorphometric analysis of the repair process of autogenous bone grafts fixed at rat calvaria with cyanoacrylate. *J Appl Oral Sci* 19:529–534, 2011
26. Eufinger H, Leppänen H: Iliac crest donor site morbidity following open and closed methods of bone harvest for alveolar cleft alveoplasty. *J Craniomaxillofac Surg* 28:31–38, 2000.
27. Finn RA, Bell WH, Brammer JA: Interpositional "grafting" with autogenous bone and coralline hydroxyapatite. *J Maxillofac Surg* 8:217–227, 1980.
28. Freihofer HP, Borstlap WA, Kuijpers-Jagtman AM: Timing and transplant materials for closure of alveolar clefts: A clinical comparison of 296 cases. *J Craniomaxillofac Surg* 21:143, 1993.
29. Gallie WE, Robertson DE: The transplantation of bone. *JAMA* 70:1134, 1918.
30. Ganguli A, Steward C, Butler SL, et al: Bacterial adhesion to bisphosphonate coated hydroxyapatite. *J Mater Sci Mater Med* 16:283–287, 2005.
31. Gibson T, Davis WB: The distortion of autogenous cartilage grafts: Its causes and prevention. *Br J Plast Surg* 10:257, 1958.
32. Godin MS, Waldman R, Johnson CM: The use of expanded polytetrafluoroethylene (Gore-Tex) in rhinoplasty. *Arch Otolaryngol Head Neck Surg* 121:1131, 1995.
33. Gosain AK, Persing JA: Report on the Symposium "Biomaterials in the Face: Benefits and Risks." *J Craniofac Surg* 10:1, 1999.
34. Gottsauner A, Hardt N: Technik and erfahrungen mit der Sinuslift-OP und enos-salen implantalen. *Z Zahnärztl Implantol* 9:184–187, 1993.
35. Goulet JA, Senunas LE, DeSilva GL, Greenfield ML: Autogenous iliac crest bone graft: Complications and functional assessment. *Clin Orthop Relat Res* 339:76–81, 1997.
36. Grossman MG, Ducey SA, Nadler SS, Levy AS: Meralgia paresthetica: Diagnosis and treatment. *J Am Acad Orthop Surg* 9:336–344, 2001.
37. Gunter JP, Clark CP, Friedman RM: Internal stabilization of autogenous rib cartilage grafts in rhinoplasty: A barrier to cartilage warping. *Plast Reconstr Surg* 100:162, 1997.
38. Gunter JP, Friedman RM: Lateral crural strut graft: Technique and clinical applications in rhinoplasty. *Plast Reconstr Surg* 99:943–952, 1997.
39. Gunter JP, Rohrich RJ: Augmentation rhinoplasty: Onlay grafting using shaped autogenous septal cartilage. *Plast Reconstr Surg* 86:39, 1990.
40. Hacker Von. Ersatz von Schaedeldefekten durch unter der kopfschwartz verscholbener oder un gelappte periostknochen. *Bietr Klin Chir* 37:499, 1903.
41. Hall HD, Posnick JC: Early results of secondary bone grafts in 106 alveolar clefts. *J Oral Maxillofac Surg* 41:289, 1983
42. Hill NM, Horne JG, Devane PA: Donor site morbidity in the iliac crest bone graft. *Aust N Z J Surg* 69:726, 1999
43. Hiraga Y: Complications of augmentation rhinoplasty in the Japanese. *Ann Plast Surg* 4:495, 1980.
44. Holmes RE: Bone regeneration within a coralline hydroxyapatite implant. *Plast Reconstr Surg* 63:626–633, 1979.
45. Holmes RE, Wardrop RW, Wolford LM: Hydroxylapatite as a bone graft substitute in orthognathic surgery: Histologic and histometric findings. *J Oral Maxillofac Surg* 46:661, 1988.
46. Hughes CW, Revington PJ: The proximal tibia donor site in cleft alveolar bone grafting: Experience of 75 consecutive cases. *J Craniomaxillofac Surg* 30:12, 2002.
47. Ilankovan V, Stronczek M, Telfer M, et al: A prospective study of trephined bone grafts of the tibial shaft and iliac crest. *Br J Oral Maxillofac Surg* 36:434–439, 1998.
48. Isaksson S: Evaluation of three bone grafting techniques for severely resorbed maxillae in conjunction with immediate endosseous implants. *Int J Oral Maxillofac Implants* 9:679–688, 1994.

49. Ivy R: Bone grafting for restoration of defects of the mandible. *Plast Reconstr Surg (1946)* 7:333, 1951.
50. Jackson IT, Adham H, Bite U, Marx R: Update on cranial bone grafts in craniofacial surgery. *Ann Plast Surg* 18:37–40, 1987.
51. James JD, Geist ET, Gross BD: Adynamic ileus as a complication of iliac bone removal. *J Oral Surg* 39:289, 1981.
52. Johannson B, Ohlsson A: Bone grafting and dental orthopaedics in primary and secondary cases of cleft lip and palate. *Acta Chir Scand* 112:112, 1961.
53. Joshi A, Kostakis GC: An investigation of post-operative morbidity following iliac crest graft harvesting. *Br Dent J* 196:167, 2004
54. Kager AN, Marks M, Bastrom T, Newton PO: Morbidity of iliac crest bone graft harvesting in adolescent deformity surgery. *J Pediatr Orthop* 26:132–134, 2006.
55. Kalk W, Raghoebar G, Jansma J, Boering G: Morbidity from iliac crest bone harvesting. *J Oral Maxillofac Surg* 54:1424, 1996.
56. Kappis A: Zur deckung von schaedeldefekten. *Centralblat Chir* 42:409, 1917.
57. Keller EE, Triplett WW: Iliac bone grating: Review of 160 consecutive cases. *J Oral Maxillofac Surg* 45:11, 1987
58. Kessler P, Thorwarth M, Bloch-Birkholz A, et al: Harvesting if bone from the iliac crest—a comparison of the anterior and the posterior sites. *Br J Oral Maxillofac Surg* 43:51–56, 2005.
59. Kim DW, Shah AR, Toriumi DM: Concentric and eccentric carved costal cartilage: A comparison of warping. *Arch Facial Plast Surg* 8(1):42–46, 2006.
60. Klapp R, Schroeder H: *Die unterkieferschussbruche*, Berlin, 1917, Hermann Meusser.
61. Kline Jr, RM, Wolfe SA: Complications associated with the harvesting of cranial bone grafts. *Plast Reconstr Surg* 95:5, 1995.
62. Koenig F: Der knoecherne ersatz schaedeldefekten. *Centralblat Chir* 17:497, 1890.
63. Koole R, Bosker H, Norman van der Dussen F: Late secondary autogenous bone grafting in cleft patients comparing mandibular and iliac crest grafts. *J Craniomaxillofac Surg* 17:28–30, 1989.
64. Kübler N, Reuther J, Kirschner T, et al: Osteoinductive, morphologic and biomechanical properties of autolyzed, antigen-extracted, allogenic bone. *J Oral Maxillofac Surg* 51:1346–1357, 1993.
65. Kübler NR: Osteoinduction und reparation. *Mund Kiefer Gesichts Chir* 1:2–25, 1997.
66. LaRossa D, Buchman S, Rothkopf DM, et al: A comparison of iliac and cranial bone in secondary grafting of alveolar clefts. *Plast Reconstr Surg* 96:789–797, 1995.
67. LaTrenta GS: *Atlas of aesthetic face and neck surgery*, Philadelphia, 2004, Saunders, pp 2–67.
68. Laurie JW, Kaban LB, Mulliken JB, Murray JE: Donor site morbidity after harvesting rib and iliac bone. *Plast Reconstr Surg* 73:933–938, 1984.
69. Lebedinsky J, Virenque M: *Prothese. Chirurgie Cranio-Maxillo-Faciale*, Paris, 1918, Librarie J B Bailliere et Fils, pp 218–223.
70. Lindermann A: *Bruhn's ergebnisse aus dem Düsseldorfer Lazareff*, Wiesbaden, 1916, Kieferschussverletzungen, p 243.
71. Lindemann A: Quoted by Dolomore WH. *Br Dent J* 38:16, 1917.
72. Marx RE, Morales MJ: Morbidity from bone harvest in major jaw reconstruction: A randomized trial comparing the lateral anterior and posterior approaches to the ilium. *J Oral Maxillofac Surg* 48:196–203, 1988.
73. Massey EW: Meralgia paraesthetica secondary to trauma of bone graft. *J Trauma* 20:342–343, 1980.
74. Mazock JB, Schow SR, Triplett RG: Posterior iliac crest bone harvest: Review of technique, complications and use of an epidural catheter for postoperative pain control. *J Oral Maxillofac Surg* 61:1497–1503, 2003.
75. Mischkowski RA, Selbach I, Neugebauer J, et al: Lateral femoral cutaneous nerve and iliac crest bone grafts—anatomical and clinical considerations. *Int J Oral Maxillofac Surg* 35:366–372, 2006.
76. Mowlavi A, Pham S, Wilhelmi B, et al: Anatomical characteristics of the conchal cartilage with suggested clinical applications in rhinoplasty surgery. *Aesthet Surg J* 30:523–526, 2010.
77. Mowlem R: Cancellous chips bone grafts: Report on 72 cases. *Lancet* 2:746, 1944.
78. Mueller W: Zur frage der temporaren schaedelresektion an stelle der trepanation. *Centralblat Chir* 17:65, 1890.
79. Murrell GL: Auricular cartilage grafts and nasal surgery. *Laryngoscope* 114(12):2092–2102, 2004.
80. Nkenke E, Weisbach V, Winckler E, et al: Morbidity of harvesting of bone grafts from the iliac crest for preprosthetic augmentation procedures: A prospective study. *Int J Oral Maxillofac Surg* 33:157–163, 2004.
81. Nocini PF, Bedogni A, Valsecchi S, et al: Fractures of the iliac crest following anterior and posterior bone graft harvesting. Review of the literature and case presentation. *Minerva Stomatol* 52:441, 2003
82. Nwoku AL, Al Atel A, Al Shlash S, et al: Retrospective analysis of secondary alveolar cleft grafts using iliac or chin bone. *J Craniofac Surg* 16:864–868, 2005.
83. Ollier L: Reserches experimentales sur les greffes osseuses. *J Physiol D Homme et des Animaux* 3:88, 1860.
84. Ortiz-Monasterio F, Olmedo A, Oscoy LO: The use of cartilage grafts in primary aesthetic rhinoplasty. *Plast Reconstr Surg* 67:597, 1981.
85. Peer L: *Transplantation of tissues, vol 1*, Baltimore, 1955, Williams & Wilkins, p 181.
86. Peer LA: Cartilage grafting. *Br J Plast Surg* 7:250, 1955.
87. Piecuch JF, Topazian RG, Skoly S, et al: Experimental ridge augmentation with porous hydroxyapatite implants. *J Dent Res* 62:148–154, 1983.
88. Porchet F, Jacques B: Unusual complications at iliac crest bone graft donor site: Experience with two cases. *Neurosurgery* 39:856–859, 1996.
89. Raghavan U, Jones NS, Romo R III: Immediate autogenous cartilage grafts in rhinoplasty after alloplastic implant rejection. *Arch Facial Plast Surg* 6:192–196, 2004.
90. Rawashdeh MA: Morbidity of iliac crest donor site following open bone harvesting in cleft lip and palate patients. *Int J Oral Maxillofac Surg* 37:223–227, 2008.
91. Rees TD, Krupp S, Wood-Smith D: Secondary rhinoplasty. *Plast Reconstr Surg* 46:332, 1970.
92. Robertson PA, Wray AC: Natural history of posterior iliac crest bone graft donation for spinal surgery: A prospective analysis of morbidity. *Spine* 26:1473, 2001
93. Rosen HM: Surgical correction of the vertically deficient chin. *Plast Reconstr Surg* 82:247–255, 1988.
94. Rosen HM: Porous block hydroxyapatite as an interpositional bone-graft substitute in orthognathic surgery. *Plast Reconstr Surg* 83:985, 1989.
95. Rosen HM: Definitive surgical correction of vertical maxillary deficiency. *Plast Reconstr Surg* 85:215–221, 1990.
96. Rosen HM: Response of porous hydroxyapatite to contiguous tissue infection. *Plast Reconstr Surg* 88:1076, 1991.
97. Rosen HM, Ackerman JL: Porous block hydroxyapatite in orthognathic surgery. *Angle Orthod* 61:185, 1991.
98. Rosen HM, McFarland MM: The biologic behavior of hydroxyapatite implanted into the maxillofacial skeleton. *Plast Reconstr Surg* 85:718–723, 1990.
99. Rubin JP, Yaremchuk MJ: Complications and toxicities of implantable biomaterials used in facial reconstructive and aesthetic surgery: A comprehensive review of the literature. *Plast Reconstr Surg* 100:1336, 1997.
100. Sadove AM, Nelson CL, Eppley BL, Nguyen B: An evaluation of calvarial and iliac donor sites in alveolar cleft grafting. *Cleft Palate J* 27:225–228, 1990.
101. Sailer HF: Experiences with the use of lyophilized bank cartilage for facial contour correction. *J Maxillofac Surg* 4:149, 1976.
102. Salyer KE, Ubinas EE, Snively SL: Porous, hydroxyapatite as an onlay graft in maxillofacial surgery. *Plastic Surgical Forum* 8:61, 1986.
103. Sasso RC, LeHuec JC, Shaffrey C; Spine Interbody Research Group: Iliac crest bone graft donor site pain after anterior lumbar interbody fusion: a prospective patient satisfaction outcome assessment. *J Spinal Disord Tech* 18:S77–S81, 2005

104. Schnee CL, Freese A, Weil RJ, Marcotte PJ: Analysis of harvest morbidity and radiographic outcome using autograft for anterior cervical fusion. *Spine (Phila Pa 1976)* 22:2222–2227, 1997.
105. Seiler JG, III, Johnson J: Iliac crest autogenous bone grafting: Donor site complications. *J South Orthop Assoc* 9:91, 2000
106. Seydel H: Eine neue methode, grosse knochendefekte des schaedels zu decken. *Centralblat Chir* 12:209, 1889.
107. Sheehan JH: Use of iliac bone in the facial and cranial repair. *Am J Surg* 52:55, 1941.
108. Sheen JH: Spreader graft: A method of reconstructing the roof of the middle nasal vault following rhinoplasty. *Plast Reconstr Surg* 73(2):230–239, 1984.
109. Sheen JH: The ideal dorsal graft: A continuing quest. *Plast Reconstr Surg* 102:2490, 1998.
110. Silber JS, Anderson DG, Daffner SD, et al: Donor site morbidity after anterior iliac crest bone harvest for single level anterior cervical discectomy and fusion. *Spine* 28:134, 2003
111. Skyoff W: Zur frage der knochenplastik am unterkeifer. Centralblatt f. *Chirurgie* 35:881, 1900.
112. Soehr O: Zur frage des schaedelplastik. *Beitr Klin Chir* 55:465, 1907.
113. Stevenson S: Biology of bone grafts. *Orthop Clin North Am* 30:543, 1999
114. Summers BN, Eisenstein SM: Donor site pain from the ilium. *J Bone Joint Surg Br* 71A:677, 1989
115. Swan MC, Goodacre TE: Morbidity at the iliac crest donor site following bone grafting of the cleft alveolus. *Br J Oral Maxillofac Surg* 44:129–133, 2006.
116. Terino EO: Alloplastic facial contouring by zonal principles of skeletal anatomy. *Clin Plast Surg* 19:487–510, 1992.
117. Tessier P: Autogenous bone grafts taken from the calvarium for facial and cranial applications. *Clin Plast Surg* 9:531, 1982.
118. Tessier P, Kawamoto H, Matthews D, et al: Autogenous bone grafts and bone substitutes—tools and techniques: I. A 20,000-case experience in maxillofacial and craniofacial surgery. *Plast Reconstr Surg* 116(5 Suppl):6S–24S, 2005.
119. Tessier P, Kawamoto H, Matthews D, et al: Complications of harvesting autogenous bone grafts: A group experience of 20,000 cases. *Plast Reconstr Surg* 116(5 Suppl):72S–73S, 2005.
120. Tessier P, Kawamoto H, Matthews D, et al: Taking bone grafts from the anterior and posterior ilium—tools and techniques: II. A 6800-case experience in maxillofacial and craniofacial surgery plastic and reconstructive surgery. *Plast Reconstr Surg* 116(5 Suppl):25S–37S, 2005.
121. Tessier P, Kawamoto H, Matthews D, et al: Taking long rib grafts for facial reconstruction—tools and techniques: III. A 2900-case experience in maxillofacial and craniofacial surgery plastic and reconstructive surgery. *Plast Reconstr Surg* 116(5 Suppl):38S–46S, 2005.
122. Tessier P, Kawamoto H, Matthews D, et al: Taking tibial grafts in the diaphysis and upper epiphysis—tools and techniques: IV. A 650-case experience in maxillofacial and craniofacial surgery plastic and reconstructive surgery. *Plast Reconstr Surg* 116(5 Suppl):47S–53S, 2005.
123. Tessier P, Kawamoto H, Matthews D, et al: Taking calvarial grafts, either split in situ or splitting of the parietal bone flap ex vivo—tools and techniques: V. A 9650-case experience in craniofacial and maxillofacial surgery. *Plast Reconstr Surg* 116(5 Suppl):54S–71S, 2005.
124. Tessier P, Kawamoto H, Matthews D, et al: Taking calvarial grafts—tools and techniques: VI. The splitting of a parietal bone "flap." *Plast Reconstr Surg* 116(5 Suppl):74S–88S, 2005.
125. Urist MR: Bone: Formation by autoinduction. *Science* 150:893–899, 1965.
126. Virenque M: *Chirurgie reparatrice maxillo-faciale*, Paris, 1940, Librarie Maloine, pp 131–136.
127. Virenque M: Quelques notions nouvelles en chirurgie reparatrice maxillo-faciale. *Rev Stomatol* 43:149, 1942.
128. Von Eiselsberg F: Zur behandlung von ernobenen schaedelknochen defekten. *Bielage Zentralblat Chir* 22:44, 1895.
129. Waite PD, Matukas VJ: Zygomatic augmentation with hydroxyapatite: A preliminary report. *J Oral Maxillofac Surg* 44:349, 1986.
130. Weikel A, Habal M: Meralgia paresthetica: A complication of iliac bone procurement. *Plast Reconstr Surg* 4:572, 1977.
131. Westrich GH, Geller DS, O'Malley MJ, et al: Anterior iliac crest bone graft harvesting using the corticocancellous reamer system. *J Orthop Trauma* 15:500, 2001
132. Whitaker LA: Aesthetic augmentation of the malar-midface structures. *Plast Reconstr Surg* 90:337–341, 1987.
133. Whitaker LA: Temporal and malar-zygomatic reduction and augmentation. *Clin Plast Surg* 18:55, 1991.
134. White E, Shors EC: Biomaterial aspects of Interpore-200 porous hydroxyapatite. *Dent Clin North Am* 30:49–67, 1986.
135. Wilkinson TS: Complications in aesthetic malar augmentation. *Plast Reconstr Surg* 71:643, 1983.
136. Williams JD, Romo T, III, Sclafani AP, Cho H: Porous high-density polyethylene implants in auricular reconstruction. *Arch Otolaryngol Head Neck Surg* 123:578, 1997.
137. Wolfe SA: Malar augmentation using autogenous materials. *Clin Plast Surg* 18:39, 1991.
138. Wolfe SA, Kawamoto HK: Taking the iliac-bone grafts. *J Bone Joint Surg Am* 60:411, 1978.
139. Wolford LH, Wardrop RW, Hartog J: Coralline porous hydroxyapatite as a bone graft substitute in orthognathic surgery. *J Oral Maxillofac Surg* 45:1034–1042, 1987.
140. Yaremchuk MJ: Infraorbital rim augmentation. *Plast Reconstr Surg* 107:1585–1590, 2001.
141. Yaremchuk MJ. Improving periorbital appearance in the "morphologically prone." *Plast Reconstr Surg* 114:980–987, 2004.
142. Yaremchuk MJ, Israeli D: Paranasal implants for correction of midface concavity. *Plast Reconstr Surg* 102:1676–1684, 1998.
143. Zijderveld SA, ten Bruggenkate CM, van Den Bergh JP, Schulten EA: Fractures of the iliac crest after split-thickness bone grafting for preprosthetic surgery: Report of 3 cases and review of the literature. *J Oral Maxillofac Surg* 62:781–786, 2004.

19

Primary Mandibular Deficiency Growth Patterns with or without Maxillary Arch Constriction

JEFFREY C. POSNICK, DMD, MD

- Functional Aspects
- Clinical Characteristics
- Dental Findings
- Radiographic Findings
- Treatment Indications and Objectives
- The Role of Growth Modification in the Preadolescent Child
- Orthodontic Camouflage Approach
- Definitive Reconstruction
- Postsurgical Orthodontic Maintenance and Detailing
- Complications, Informed Consent, and Patient Education
- Temporomandibular Disorders: The Effects of Orthodontics and Orthognathic Surgery
- Conclusions

The surgical correction of the underdeveloped mandible was first reported in the American literature by Vilray P. Blair in the *Journal of the American Medical Association* in July 1909.[7] Dr. Blair presented two cases of retrognathia in which a horizontal ramus osteotomy was performed on each side of the mandible. The operative technique was carried out through a preauricular stab incision; an aneurysm needle was introduced posteriorly to the neck of the condyle and directed medially around to the ascending ramus, where it then exited through the cheek in front of the anterior border of the ramus. A Gigli saw was then put in place and blindly used to complete an osteotomy. The same procedure was completed on the opposite side of the jaw, and then the mandible was advanced into the desired occlusion. Intermaxillary fixation was applied until a callus formed. As a result of the limited bone contact across the osteotomy sites, a strong relapse tendency required persistent counterforces with continued intermaxillary elastics. Extrusion of the incisors with periodontal sequelae and recurrent malocclusion was a frequent observation.

Mandibular deficiency is the most prevalent form of dentofacial deformity.[2,30,32,39,43] It is more common among Caucasians than Africans or Asians, which suggests that a hereditary component is a predominant part of the etiology. During the mid 1970s, the public health service of the U.S. Department of Health, Education, and Welfare conducted an assessment of the occlusions of children who were 6 to 11 years old and 12 to 17 years old. These collected data indicated that approximately 20% of the sample populations had an Angle Class II malocclusion. Unfortunately, no other means of evaluation were performed to separate those children with a Class II jaw disharmony from those with a more straightforward dental Class II malocclusion. According to a National Health and Nutrition Examination Survey, severe mandibular deficiency (defined as >7 mm of positive overjet) in the U.S. Caucasian population has a prevalence of approximately 10%.[43] This pattern of Class II excess overjet is less frequent among African-American and Mexican-American groups. It can be assumed that more than 7 mm of overjet indicates a jaw discrepancy of such magnitude that orthognathic surgery would be the preferred way to manage the problem. The majority of these patients will have dentofacial deformities that fall into the categories of a long face growth pattern, maxillomandibular deficiency, or primary mandibular deficiency with or without arch-width discrepancy.

This chapter will focus on the patient with a *mandibular deficiency* who presents without a significant component of either vertical or horizontal upper jaw deformity. These individuals frequently will have a degree of maxillary

arch-width constriction that also requires correction. Although this categorization of primary mandibular deficiency may appear arbitrary, it focuses on a distinct subgroup of patients who routinely present for clinical assessment and treatment.

Functional Aspects

- Articulation errors during speech and swallowing difficulties that result from the Class II malocclusion are expected (see Chapter 8).
- Difficulties with chewing and with lateral and protrusive mandibular movements as a result of the Class II malocclusion are also expected.
- Upper airway obstruction during sleep often exists as a result of the retropositioned tongue. Obstructive sleep apnea should be ruled out (see Chapter 26).
- Detrimental long-term effects on the dentition and the periodontium as a result of the crowding of the mandibular teeth in limited alveolar housing or from previous camouflage orthodontic treatments are often seen (see Chapters 5 and 6).
- A higher incidence of temporomandibular disorders that result from mandibular deficiency Class II malocclusion have been reported (see Chapters 9 and 36).

Clinical Characteristics

- The lower lip is curled and everted, and it is often wedged behind the incisors with an accentuated labiomental fold. The shorter the lower anterior facial height, the greater the tendency for an accentuated labiomental fold.
- The gonial angles of the mandible tend to be relatively square. The shorter the face and the deeper the bite, the more acute the angles.
- There is a retruded and weak chin with a convex profile.
- There is a deficient mandible with the illusion of a large nose.
- There is a short chin-to-neck length and an obtuse angle, often with a double chin (even in a thin individual).
- The upper lip appears short, curled, and protrusive.

Dental Findings

- There are varied degrees of increased overjet with a Class II molar and canine relationship.
- The crowding of the teeth within the lower arch (i.e., the smaller the mandible, the greater the crowding) needs to be evaluated, and so does lower incisor flaring. Mandibular dental compensations often mask the true skeletal discrepancy.
- There are varied degrees of dental compensation in the upper jaw with either procumbency (i.e., "buck teeth") or maxillary incisors that are retroclined.
- The anterior deep bite (i.e., excessive overbite) may involve impingement of the anterior maxillary palatal tissues by the mandibular incisors.
- There is an excessive curve of Spee in the mandible (i.e., the deeper the anterior bite, the greater the excess curve).
- A reduced or negative curve of Spee is found in the maxillary arch.
- Dental crowding may also be seen in the maxilla (i.e., the more constricted the maxilla, the greater the crowding).
- Excessive wear facets, cusp fractures, and significant restorations in the posterior dentition are frequent findings in adults.
- Accelerated loss of the periodontium associated with the maxillary incisors (due to traumatic occlusion or previous orthodontics); the maxillary molars (due to arch constriction or previous orthodontics); and the mandibular incisors (due to supraeruption and anterior flaring from previous orthodontics) may be seen in adults.

Radiographic Findings

- Horizontal retrusion is measurable at the B-point of the mandible.
- The A-point–nasion–B-point angle is generally increased as a result of the retrusive lower jaw.
- Excessive procumbency of the mandibular incisors is frequently measurable.
- A vertically short and retrusive chin is generally measurable.
- The maxillary incisors are often procumbent, but they may be retropositioned as a result of previous extractions and camouflage orthodontics.

Treatment Indications and Objectives

The individual's immediate aesthetic concerns often center on 1) unfavorable lower-lip contours (e.g., a curled and everted lower lip with an accentuated labiomental fold) 2) a lack of prominence of the chin (e.g., a weak profile) and 3) an unattractive neck (e.g., a soft-tissue double-chin tendency).[27,38,39,42,48,47,50,59] Functional issues are also to be expected. For example, when the anterior bite is "deep," it will cause irritation of the gingival tissues of the palate just behind the maxillary incisors. Partial obstruction of the upper airway as a result of a retropositioned tongue is also common (i.e., heavy snoring, restless sleep, daytime fatigue, and documented or undiagnosed sleep apnea; see Chapter 26).[11,62] Complaints of an uncomfortable "dual" bite, chewing difficulties, masticatory muscle discomfort, and audible speech articulation errors are frequent. The individual with a Class II deep bite may have a greater tendency toward clicking (i.e., anterior disc displacement) in each temporomandibular joint.[25,51] Interestingly, there is little scientific evidence to support the notion that the signs of clicking will into more significant temporomandibular joint pathology (see Chapter 9).

When the mandible is deficient, with significant overjet, and when the anterior facial height is short, the lower lip tends to "get caught" behind the maxillary incisors. During growth, if the lower lip stays behind the upper teeth, then the upper teeth will adapt by tipping forward, thus creating a Class II, Division 1 malocclusion with varied degrees of maxillary incisor spacing. When the mandibular deficiency and the protrusive lower incisors are combined with procumbent maxillary incisors, the individual tends to be concerned with both the lack of chin prominence and the protruding maxillary anterior teeth (i.e., a toothy smile). Alternatively, if the lower lip remains in front of the maxillary incisors, there is an adaptive tendency for the maxillary incisors to tip lingually (i.e. Class II, Division 2).

The Role of Growth Modification in the Preadolescent Child

When the preadolescent child with mandibular deficiency is identified, a growth modification approach to treatment is frequently considered by the evaluating orthodontist.[13,33,32] Cozza and colleagues completed a systematic review of the literature to assess the efficacy of functional appliance use for enhancing mandibular growth in subjects with Class II malocclusions.[14] With the use of the Medline database, the survey covered the period from January 1966 to January 2005. A review of 704 articles confirmed only 22 that qualified for the final analysis. Of these 22 articles, only four randomized clinical trials were retrieved and found to be worthy of inclusion. *Interestingly, none of these four trials reported a clinically significant change in mandibular length to be induced by functional appliances in the patients with Class II deformities.* According to Proffit, the only concrete indications for the treatment of the preadolescent mixed dentition child with a mandibular deficiency who presents without an anterior open bite are the following[44]:

- Significant trauma to the palatal tissue (i.e., the lower incisors are injuring the palatal mucosa) or other indications of potential irreversible tissue damage
- Special concerns about the effects of the condition on the child's psychological well-being and the strong belief that the recommended treatment will alter this in a positive way
- Skeletal maturation that is well ahead of dental development so that the adolescent growth spurt occurs before the mixed dentition ends

Despite the review by Cozza and colleagues and Proffit's stated limited indications for early mixed dentition treatment in the child with a mandibular deficiency, a growth modification approach is often carried out by the evaluating orthodontist. The goals are frequently stated to be the restraining of forward maxillary growth while encouraging the mandible to grow downward and forward. In accordance with this rationale, mixed dentition growth modification treatment frequently incorporates the following steps:

1. *The alignment of the upper incisors* (i.e., the correction of lingual tipping with a Class II, Division 2 malocclusion) to prevent any interference with the forward movement of the mandible.
2. *Attempts to impede the biologic programmed forward growth of the maxilla and to facilitate the forward growth of the mandible.* Extraoral (posterior vectored) compressive forces are typically applied to the maxilla through a headgear device.
3. *Attempts to stimulate the mandibular posterior teeth to hypererupt while preventing the incisors from doing so.* During this process, it is hoped that the deep overbite will improve. An intra-oral functional appliance is often designed to facilitate these events.

Orthodontic Camouflage Approach

When the orthodontist proceeds with growth modification maneuvers and a less than favorable biologic response is realized, it is apparently tempting to proceed with a camouflage (dental compensation) approach (Figs. 19-1 through 19-5).[34,35,37,36,42] This would be carried out to "neutralize" the occlusion rather than to position the teeth solidly into the alveolar process in combination with orthognathic surgery. Current evidence-based medical standards recommend that the treating orthodontist have a "timeout" (i.e., a sit-down discussion and reassessment with the parents and the patient) that includes a review of the records (e.g., photos, models, radiographs) and a discussion of the biologic effects on the airway, the facial aesthetics, the occlusal stability, and the periodontium that may result from the various treatment options. A referral to an experienced orthognathic surgeon for the discussion of possible future surgical aspects is highly recommended.

An orthodontic camouflage approach for the individual with mandibular deficiency may take the form of maxillary premolar extractions so that the upper incisors can be retracted. This is generally combined with the flaring of the mandibular incisors. When this is carried out in the individual with a mandibular deficiency, the short-term objective of a neutralized occlusion may be achieved, but the following are potential sequelae: 1) unfavorable long-term facial aging 2) the risk for obstructive sleep apnea later in life 3) long-term periodontal deterioration 4) lower anterior crowding with recurrent malocclusion and 5) an inconsistent occlusion (i.e., a dual bite) (see Figs. 19-1 through 19-5). The orthodontist may feel favorable toward a camouflage approach on the basis of the belief that the patient can always have a "chin implant" to improve his or her facial aesthetics. Unfortunately, this approach will rarely achieve a pleasing aesthetic outcome. A chin implant tends to result in 1) an accentuated labiomental fold 2) an overly prominent pogonion and 3) a residual obtuse neck–jaw angle

• **Figure 19-1** A 20-year-old man with a primary mandibular deficiency growth pattern requested a surgical consultation for a weak chin. During his early teenage years, the patient underwent growth modification in an attempt to stimulate the forward projection of the mandible. This was followed by an orthodontic camouflage approach that included maxillary first bicuspid extractions to retract the anterior teeth. In addition, the mandibular anterior dentition was flared forward. His history was significant for heavy snoring, restless sleeping, and a degree of daytime fatigue, all of which are suggestive of obstructive sleep apnea. Examination confirmed a retrognathic mandible with a molar Class II deep-bite malocclusion. The mandibular incisors were crowded and procumbent. The family had hoped that a chin implant would be effective to manage the aesthetic effects. A sleep study was recommended, and an orthognathic approach with redo orthodontic treatment including lower bicuspid extractions was suggested as the preferred method to improve the airway, to achieve long-term dental health, and to enhance facial aesthetics. **A,** Frontal facial and occlusal views. **B,** Profile facial view and lateral cephalometric radiograph.

(Fig. 19-4). Furthermore, it does nothing for any baseline masticatory and airway dysfunction (see Chapter 37).

Definitive Reconstruction

Timing of Orthognathic Surgery

Stahl and colleagues carried out a longitudinal study to compare the biologic jaw growth changes of untreated subjects with Class II, Division 1 malocclusions as compared with those subjects with a normal Class I occlusion.[55] Each subject was followed from the prepubertal stage through the postpubertal stage of development. The study provides evidence of a similar growth pattern in both Class II and Class I subjects. The absolute amount of mandibular lengthening at the pubertal peak was significantly smaller in the Class II subjects than in those with normal occlusion. A significant deficiency was measured in overall mandibular length (i.e., Condyle-Gonion [Co-Gn]) in the Class II malocclusion subjects at the completion of growth. Interestingly, the

• **Figure 19-2** A 21-year-old woman with a primary mandibular deficiency growth pattern requested a surgical consultation for a weak chin. During her early teenage years, the patient underwent growth modification in an attempt to stimulate the forward projection of the mandible. This was followed by an orthodontic camouflage approach. The mandibular anterior dentition was flared forward. Her history was significant for restless sleeping and a degree of daytime fatigue, which are suggestive of obstructive sleep apnea. Examination confirmed a retrognathic mandible with a Class II malocclusion. The mandibular incisors were crowded and procumbent. The family had hoped that a chin implant would be effective to manage the aesthetic effects. A sleep study confirmed obstructive sleep apnea (respiratory disturbance index = 18/hour). An orthognathic approach with redo orthodontic treatment including lower bicuspid extractions was recommended as the preferred method to improve the airway, to achieve long-term dental health, and to enhance facial aesthetics. **A,** Frontal facial and occlusal views. **B,** Profile facial view and lateral cephalometric radiograph.

authors' findings show that a Class II dentofacial disharmony does not have a tendency to self-correct with growth.

There are numerous reports in the literature of surgeons carrying out successful orthognathic corrections in patients with mandibular deficiencies who are between the ages of 13 and 15 years. This is before the age at which growth maturity is typically considered to be complete. Interestingly, after the patient with a mandibular deficiency has undergone successful surgical correction, there are no reports late mandibular catch up growth.[5,20,23,29,45,52,61]

A review of available research data and clinical reports indicated that, when orthognathic surgery was undertaken in the patient with a mandibular deficiency as early as the completion of the adolescent growth spurt, further significant forward growth of the mandible almost never occurred.[31,55] Further horizontal growth of the maxilla at this age is also not expected. In theory, limited vertical growth of both the mandible and maxilla may continue; however, if it does, it tends to be proportionate. Proceeding with definitive orthognathic surgery in the patient with a

• **Figure 19-3** A 20-year-old man with a primary mandibular deficiency growth pattern requested a surgical consultation for a weak chin. During his the early teenage years, the patient underwent growth modification in an attempt to stimulate the forward projection of the mandible. This was followed by an orthodontic camouflage approach. The mandibular anterior dentition was flared forward. The patient's history was significant for heavy snoring, restless sleeping, and a degree of daytime fatigue, all of which are suggestive of obstructive sleep apnea. Examination confirmed a retrognathic mandible with clockwise rotation of the maxillomandibular complex. The mandibular incisors were crowded and procumbent. He had hoped that a chin implant would be effective to manage the aesthetic effects. An orthognathic approach with redo orthodontic treatment including lower bicuspid extractions was recommended as the preferred method to improve the airway, to achieve long-term dental health, and to enhance facial aesthetics. **A,** Frontal facial and occlusal views. **B,** Profile facial view and lateral cephalometric radiograph.

mandibular deficiency as early as the age of 13 or 14 years—but only after the eruption and orthodontic alignment of the teeth—is generally safe from a growth perspective.

Periodontal Considerations before Orthodontic Treatment

The position of the mandibular incisors in the alveolar housing has important periodontal consequences (see Chapter 6). In the retrognathic patient, the more the mandibular incisors are orthodontically tipped forward, the greater the chance of labial bone loss and gingival recession. There should be a healthy band of attached gingiva at the beginning of treatment and flaring of the incisors is to be avoided. This is a frequent concern in borderline crowding cases, when the final decision is made to not extract and then further labial tipping of the incisors occurs.

Even after successful gingival grafting, if further incisor flaring is allowed to occur in the presence of deficient labial bone, negative periodontal sequelae are to be expected. For patients with borderline crowding, if the incisor crowns are

• **Figure 19-4** A 29-year-old women arrived to discuss chin aesthetics. During the mixed dentition, she was recognized as having mandibular deficiency and a constricted maxillary arch width. At that time, she underwent growth modification techniques that included headgear. She also underwent rapid palatal expansion followed by compensatory orthodontic mechanics to neutralize the occlusion, but this also involved proclination of the incisors. A retrusive profile and a dual bite remained. The patient had recently undergone the placement of a chin implant without fixation. Aesthetic modification of the anterior maxillary dentition was also completed. The patient was displeased with the cosmetic results of a deepened labiomental fold, a mobile chin implant, and a "button" appearance during lip closure. There is a dual bite with Class II malocclusion when the teeth are in centric relation. **A,** Oblique and frontal facial views. **B,** Profile facial view and lateral cephalometric radiograph 6 months after chin implant placement.

morphologically large, the judicious use of interproximal reduction may create the needed space to uncrowd the arch and to simultaneously upright the teeth (see Chapter 17).

Orthodontic Preparation for Orthognathic Surgery

The clinicians (i.e., the orthodontist and surgeon), the patient, and the patient's family should agree about the treatment approach from the outset. It makes no sense for the orthodontist to tell the patient that he or she will first try to avoid surgery. The orthodontic objectives are diametrically opposite, depending on plans for either full correction (i.e., surgery and orthodontics) or camouflage (i.e., orthodontics alone). When a definitive correction is planned for the individual with mandibular deficiency, lower extractions to uncrowd the arch should always be considered. The need for maxillary extractions is not typical. The labial

• **Figure 19-5** A 43-year-old woman arrived to discuss the aesthetics of her weak chin and premature facial aging. During her late mixed dentition, she was recognized as having mandibular deficiency with a Class II malocclusion. At that time, she underwent growth modification techniques followed by compensatory orthodontic mechanics. This included upper first bicuspid extractions, the retraction of the anterior maxillary teeth, and the proclination of the mandibular incisors, all in an attempt to neutralize the occlusion. A retrusive profile and a dual bite remained. The weak profile of her adolescence had progressed into premature facial aging. She also had a lifelong history of obstructed nasal breathing, restless sleeping, and excessive daytime fatigue, all of which are suggestive of obstructive sleep apnea. An orthognathic approach with redo orthodontic treatment including lower bicuspid extractions was recommended as the preferred method to improve the airway, to achieve long-term dental health, and to enhance facial aesthetics. **A,** Frontal facial and occlusal views. **B,** Profile facial view and lateral cephalometric radiograph.

region of the mandibular incisors should be assessed for the need for gingival augmentation before the initiation of treatment.

For the patient with mandibular deficiency who has agreed to a combined treatment approach, an important orthodontic objective is to place the incisors more completely within the alveolus. Incisor placement will determine the eventual horizontal and vertical positioning of the mandible in relation to the maxilla as well as the long-term periodontal health.

The orthodontic leveling of an excessive curve of Spee in the lower arch is accomplished through a combination of the intrusion of the incisors and the extrusion of the mid-arch posterior teeth. With an excessive curve of Spee, premolar extrusion gives the major effect with only a small amount of incisor intrusion to be expected. This is initiated before surgery, but it is most efficiently fully accomplished after the mandible has been surgically repositioned (i.e., forward and down). Alternatively, if extensive intrusion of the anterior teeth is desired to correct the deep curve, this

should be done before the mandibular advancement surgery. The correction of a deep curve of Spee via incisor intrusion will result in less clockwise rotation of the mandible in association with surgical advancement. This can be leveraged for enhanced profile aesthetics.

The mechanics of the orthodontic leveling of the curve of Spee will generally result in the lengthening of the arch. The arch expansion primarily occurs at the incisors (i.e., labial flaring) and, to a lesser degree, at the molars. Space is required for both leveling and for aligning. For this reason, pre-alignment extractions to create space will be required if the severity of the curve of Spee is significant and if the available alveolar bone is limited. With a combination of incisor crowding and a deep curve of Spee, extraction therapy is always preferred. If the upper incisors are extremely flared, maxillary premolar extraction to gain space for retraction is also preferred; however, this is rarely the case.

When the deficient mandible is surgically advanced and vertically lengthened to achieve a more ideal overbite and overjet, the relative widening of the mandibular arch in the molar region occurs. Maxillary arch expansion may then be required to correct posterior crossbites. If more than several millimeters is required, then simultaneous surgical arch expansion of the maxilla through segmental Le Fort I osteotomy is the preferred approach (see Chapter 17).

Key aspects of presurgical orthodontics in the individual with mandibular deficiency include the following:

- Recognizing the need for extractions in the mandible and incorporating this information into the treatment plan from the beginning
- Recognizing the need for the surgical expansion of the upper arch (consider incorporating Le Fort I segmental osteotomies to widen the arch into the treatment plan from the beginning; see Chapter 17)
- Aligning the teeth independently within each arch with the objective of a compatible postsurgical occlusion
- Establishing the preferred sagittal and vertical positions of the incisors in the alveolar bone so that the jaw reconstruction that follows will result in enhanced facial aesthetics and improved head and neck function.

Additional orthodontic considerations for the individual with mandibular deficiency include the following:

- To manage an excessive curve of Spee in the lower arch, a majority of the leveling will be done after jaw surgery via the extrusion of the premolars.
- If the mandibular arch is fully leveled before surgery, then the bicuspid extraction spaces should also be closed before surgery.
- If a degree of lower arch leveling is to be accomplished after jaw surgery, then it is desirable to leave some of the bicuspid extraction space to be closed postoperatively.
- Passive surgical arch wires to be used for stabilization during surgery should be full-dimensional and rectangular.
- With effective plate and screw fixation at each osteotomy site, continuous intermaxillary fixation after surgery is not warranted. Nevertheless, the orthodontic placement of stable hooks throughout the arch wires is useful both at the time of surgery and to accomplish supportive vectored elastics postoperatively.

Immediate Presurgical Planning

The immediate presurgical planning takes into account collaborative clinical efforts between the orthodontist, the surgeon, the patient, and the patient's family that will have been ongoing since the initial consultation visits. Depending on the condition of the dentition and its supporting structures, the restorative dentist and the periodontist may also have been intricately involved.

Patient-specific medical and dental records are reviewed, which include (but are not limited to) the following: radiographs (e.g. computed tomography scans; panoramic, lateral, and posterior anterior cephalometric and periapical scans); photographs (facial and occlusal); and dental models.

Immediate presurgical records (i.e., within 2 to 6 weeks of surgery) will most importantly include facial measurements taken during the direct visual examination; alginate impressions of the maxilla and mandible; an accurate centric relation bite; and an accurate face-bow registration.

First, broad-stroke decisions are made regarding preferred vector changes for each jaw; only then are the precise millimeter distances and angular changes that are required in each jaw to achieve the desired result determined (see Chapter 12).[3,4,17,42] Analytic model planning is carried out on the articulated dental casts with use of the Erickson Model Table. Splints are constructed to assist with the achievement of the precise occlusion and the preferred facial aesthetics in the operating room (see Chapter 13).

During surgical planning for the individual with a primary mandibular deficiency growth pattern, it is known that bilateral sagittal split ramus osteotomies with advancement into a corrected overbite and overjet position will be required (Figs. 19-6 through 19-9).[6,21] An important consideration is whether or not surgical repositioning of the upper jaw (i.e., Le Fort I osteotomy) will also be useful to correct arch-width discrepancies; to further adjust the horizontal and vertical position of the maxilla; and to alter the maxillary plane (i.e., pitch orientation) and cant (i.e., roll orientation) (Figs. 19-10 through 19-15).[3,4,15,18,41,40,46,49,60] An assessment of the baseline chin morphology is also important. Anticipating the location of the pogonion after jaw repositioning and considering the advantage of an osseous genioplasty to achieve an enhanced horizontal

Text continued on p. 682

©Elsevier Inc.
Orthognathic Surgery; J.C. Posnick

• **Figure 19-6** A 15-year-old boy with mandibular deficiency and microgenia underwent a combined orthodontic and surgical approach. The procedures included bilateral sagittal split ramus osteotomies (horizontal advancement) and an osseous genioplasty (horizontal advancement). One year later, he underwent a septorhinoplasty procedure to open the airway and to enhance nasal aesthetics. He is shown before and 5 years after reconstruction. **A,** Frontal view in repose before and 5 years after reconstruction. **B,** Frontal views with smile before and 5 years after reconstruction.

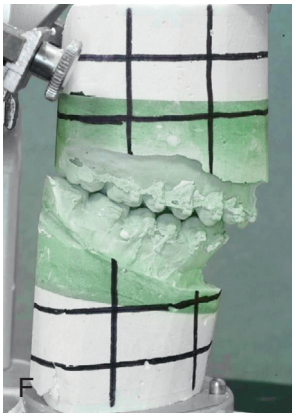
Continued

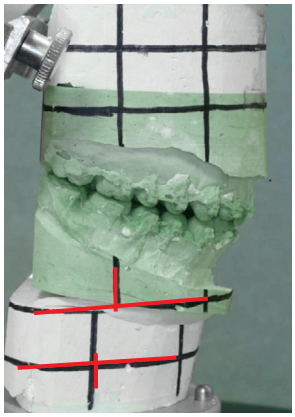
©Elsevier Inc.
Orthognathic Surgery; J.C. Posnick

• **Figure 19-6, cont'd** **C,** Oblique facial views before and 5 years after reconstruction. **D,** Profile views before and 5 years after reconstruction.

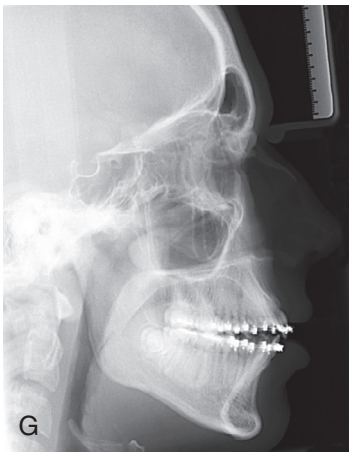
Prior to surgery (centric occlusion)

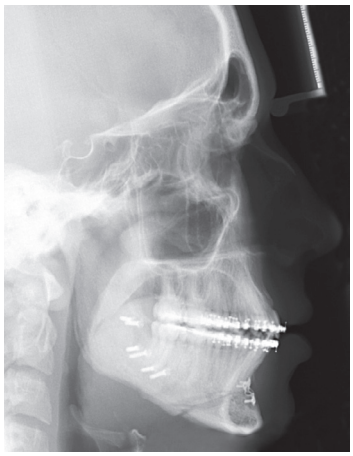
After treatment





7 mm mandibular advancement



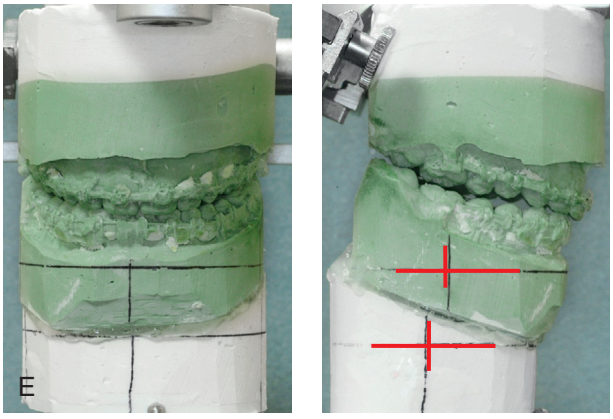


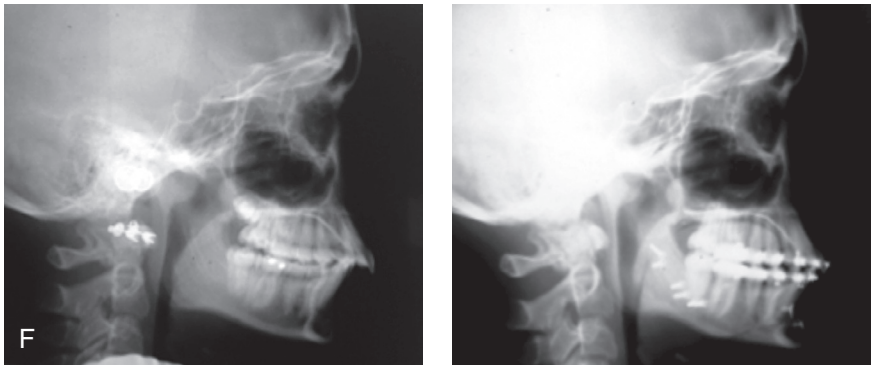
• **Figure 19-6, cont'd** **E,** Occlusal views with orthodontics in progress (centric occlusal) and 5 years after treatment. **F,** Articulated dental casts before (centric relation) and after model planning. **G,** Lateral cephalometric radiographs before and after reconstruction.

• **Figure 19-7** A 15-year-old girl with mandibular deficiency and microgenia underwent a combined orthodontic and surgical approach. The surgery included bilateral sagittal split ramus osteotomies (horizontal advancement) and an osseous genioplasty (horizontal advancement). **A,** Frontal view in repose before and after reconstruction. **B,** Oblique facial views before and after reconstruction. **C,** Profile views before and after reconstruction.

Prior to treatment

After treatment





• **Figure 19-7, cont'd** **D,** Occlusal views before and after the completion of treatment. **E,** Articulated dental casts that indicate analytic model planning. **F,** Lateral cephalometric radiographs before and after treatment.

• **Figure 19-8** A woman in her late 20s was referred by an orthodontist for surgical evaluation. During her high school years, the patient underwent maxillary first bicuspid extractions with orthodontic retraction to neutralize the occlusion in the presence of mandibular deficiency. She now hoped to improve her profile aesthetics and to achieve long-term dental health. The patient agreed to an orthodontic and surgical approach. Orthodontic (dental) decompensation required opening the maxillary bicuspid extraction spaces for dental implants and crowns. The mandibular arch was also decompensated. The patient then underwent surgery that included bilateral sagittal split ramus osteotomies (horizontal advancement); osseous genioplasty (horizontal advancement); and an anterior approach to the soft tissues of the neck (cervical flap elevation, neck defatting, and vertical platysma muscle plication). **A,** Frontal views in response before and after reconstruction. **B,** Frontal views with smile before and after reconstruction.

©Elsevier Inc.
Orthognathic Surgery; J.C. Posnick

• **Figure 19-8, cont'd** **C,** Oblique facial views before and after reconstruction. **D,** Profile views before and after reconstruction.

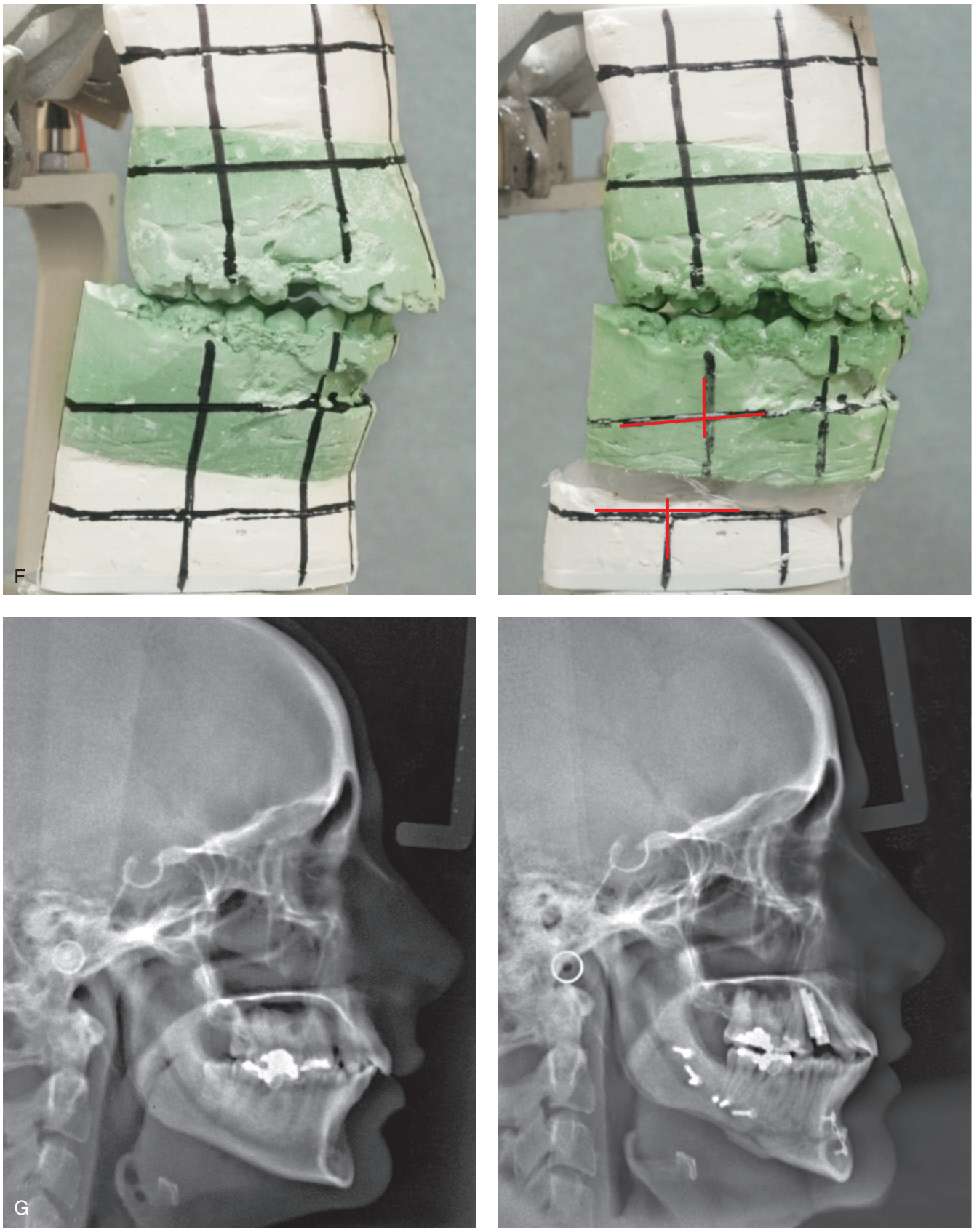
Continued

Prior to retreatment with upper bicuspid extractions as a teenager

Pre surgery with re-opening of upper bicuspid spaces for implants

After treatment with maxillary bicuspid
Implants in place

• **Figure 19-8, cont'd E,** Occlusal views before retreatment, with orthodontic decompression in progress, and then after treatment.



• **Figure 19-8, cont'd** **F,** Articulated dental casts indicating analytic model planning. **G,** Lateral cephalometric radiographs before and after reconstruction.

• **Figure 19-9** A woman in her late 20s was referred by an orthodontist for surgical evaluation. During her teenage years, she underwent camouflage orthodontics to neutralize the occlusion, including maxillary first bicuspid extractions with space closure. She arrived for evaluation to improve the occlusion and to achieve enhanced profile aesthetics. She agreed to an orthodontic and surgical approach. Orthodontic (dental) decompensation required the proclination of the maxillary anterior dentition and mandibular first bicuspid extractions with the retraction of the anterior dentition. This was followed by surgery that included bilateral sagittal split osteotomies of the mandible (horizontal advancement); osseous genioplasty (horizontal advancement); and an anterior approach to the soft tissues of the neck (cervical flap elevation and neck defatting). **A,** Frontal views in repose before and after reconstruction. **B,** Oblique facial views before and after reconstruction. **C,** Profile views before and after reconstruction.

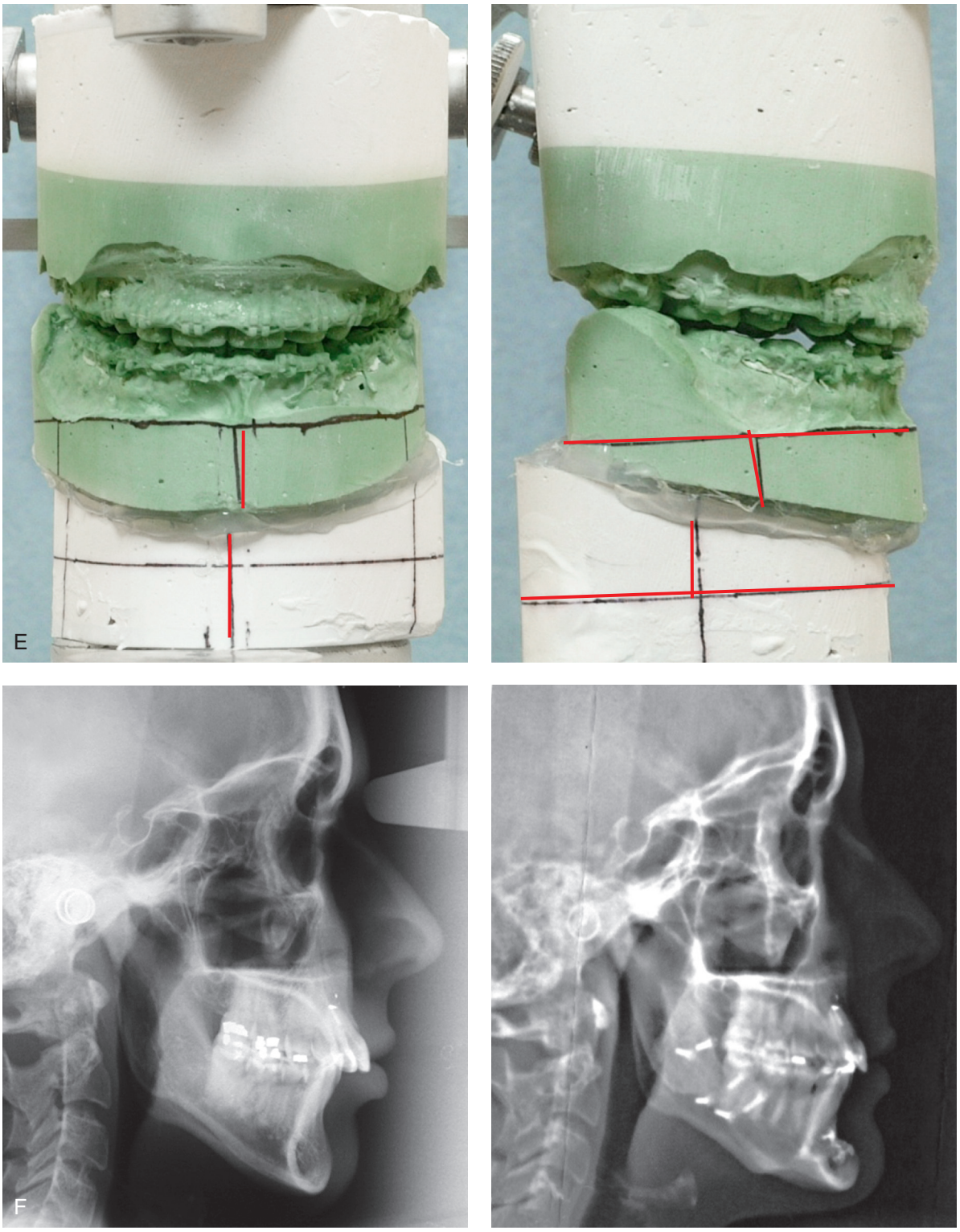
Upper bicuspids removed years earlier. Now undergoing retreatment with mandibular bicuspid extractions

Pre surgery

After treatment

• **Figure 19-9, cont'd D,** Occlusal views early during orthodontic retreatment, before surgery, and after reconstruction.

Continued



• **Figure 19-9, cont'd E,** Articulated dental casts indicating analytic model planning. **F,** Lateral cephalometric radiographs before and after treatment.

©Elsevier Inc.
Orthognathic Surgery; J.C. Posnick

• **Figure 19-10** A teenaged girl was referred by her orthodontist for surgical assessment. She had a primary mandibular deficiency and maxillary arch constriction. She also had a Class II anterior open-bite malocclusion. A comprehensive orthodontic and surgical approach was selected. No extractions were required. Her surgery included a Le Fort I osteotomy in two segments (transverse widening, horizontal advancement, and counterclockwise rotation); bilateral sagittal split osteotomies of the mandible (horizontal advancement and counterclockwise rotation); and osseous genioplasty (horizontal advancement). **A,** Frontal views in repose before and after treatment. **B,** Frontal views with smile before and after treatment.

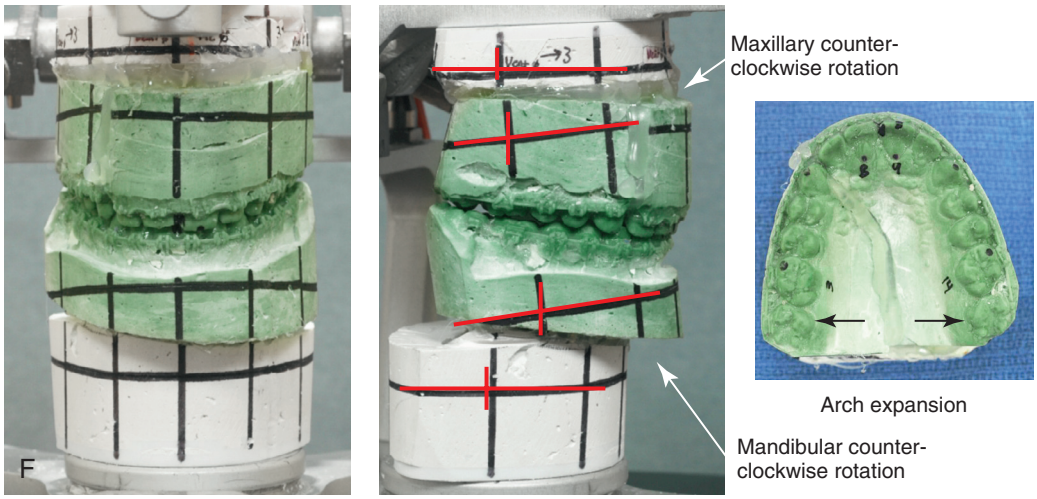
Continued

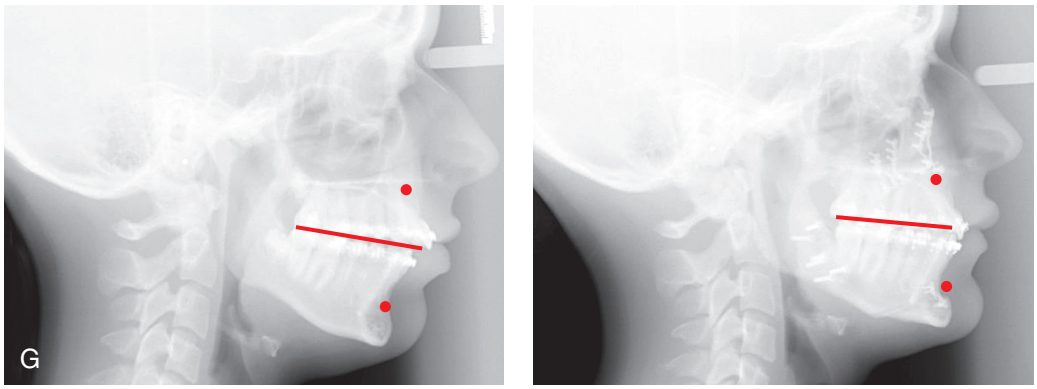
©Elsevier Inc.
Orthognathic Surgery; J.C. Posnick

• **Figure 19-10, cont'd C,** Oblique facial views before and after treatment. **D,** Profile views before and after treatment.

Pre surgery

After treatment



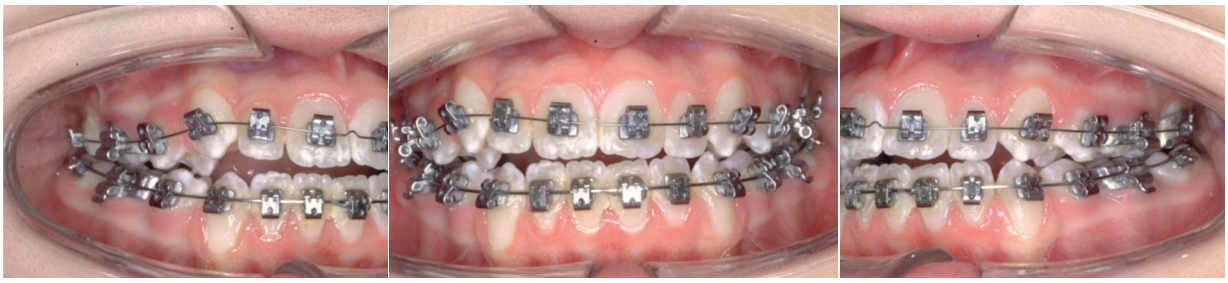


• **Figure 19-10, cont'd** **E,** Occlusal views during orthodontic decompensation and after treatment. **F,** Articulated dental casts that indicate analytic model planning. **G,** Lateral cephalometric radiographs before and after reconstruction.

• **Figure 19-11** A 16-year-old girl was referred for surgical assessment only after orthodontic treatment was well under way. She had undergone four bicuspid extractions and compensating orthodontics, but these were unsuccessful with regard to the neutralization of the occlusion. She was then recognized as having mandibular deficiency with a significant overjet, an anterior open bite, and obstructed nasal breathing. A degree of gingival recession on the labial aspect of the lower canines was appreciated. The maxillary incisors were procumbent but without space for further retraction. Reconstruction included a Le Fort I osteotomy in segments (horizontal advancement and transverse widening); bilateral sagittal split ramus osteotomies (horizontal advancement and counterclockwise rotation); osseous genioplasty (horizontal advancement); and septoplasty and inferior turbinate reduction. The patient's gingival recession which was present prior to surgery is being monitored. **A,** Frontal views in repose before and after treatment. **B,** Frontal views with smile before and after treatment.

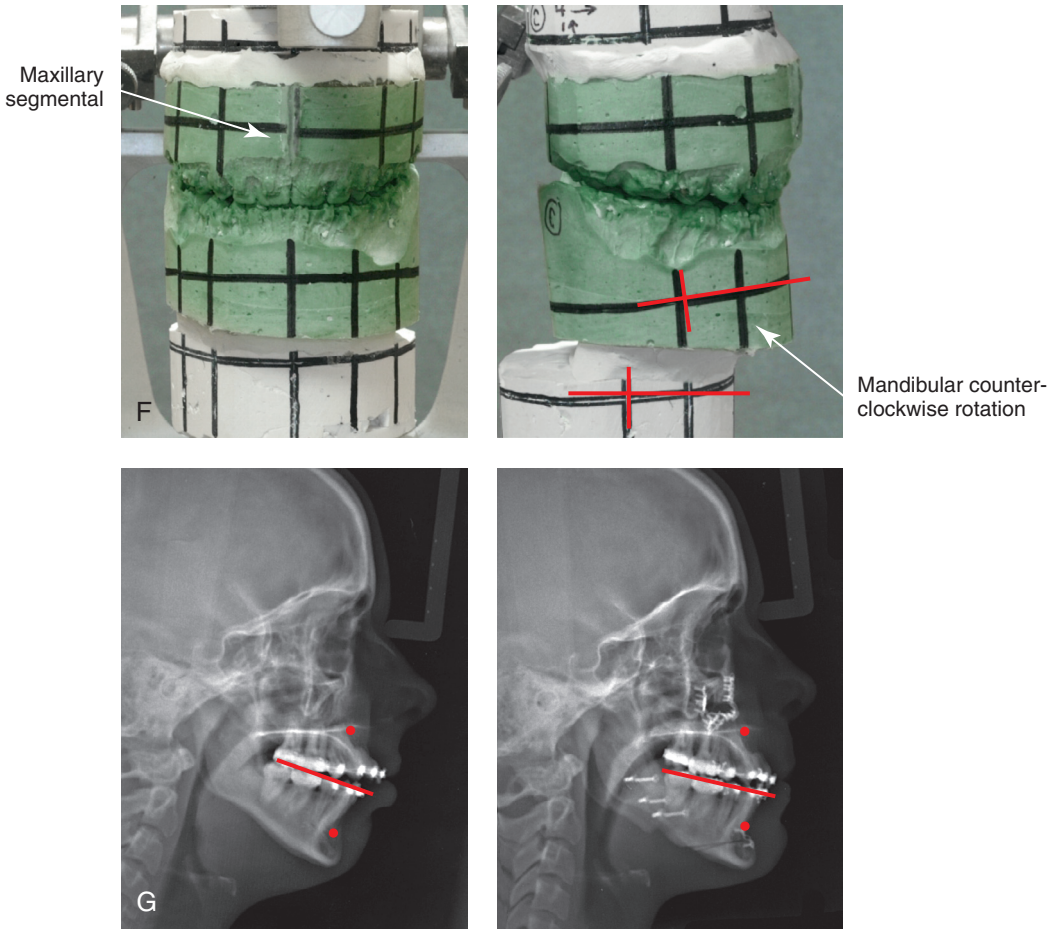
• **Figure 19-11, cont'd** **C,** Oblique facial views before and after treatment. **D,** Profile views before and after treatment.

Continued



Pre surgery with 4 bicuspid extractions prior to referral

After treatment



• **Figure 19-11, cont'd** **E,** Occlusal views before surgery and after reconstruction. **F,** Articulated dental casts that indicate analytic model planning. **G,** Lateral cephalometric radiographs before and after treatment.

• **Figure 19-12** A 30-year-old woman was referred for surgical assessment only after orthodontic treatment was well under way. When the patient was between 9 and 15 years old, orthodontic growth modification and then a camouflage approach were carried out, but these were not successful. The current orthodontic treatment was flaring the maxillary anteriors even further forward. The patient was referred to this surgeon and agreed to a combined surgical and orthodontic approach. Orthodontic retraction of the maxillary anteriors was then initiated. With sufficient orthodontic (dental) decompensation, surgery was carried out, including bilateral sagittal split osteotomies of the mandible (horizontal advancement and counterclockwise rotation); maxillary Le Fort I osteotomy in segments (arch expansion, horizontal advancement, and minimal counterclockwise rotation); osseous genioplasty (horizontal advancement); and an anterior approach to the soft tissues of the neck (cervical flap elevation, neck defatting, and vertical platysma muscle plication). **A,** Frontal views in repose before and after treatment. **B,** Frontal views with smile before and after treatment.

Continued

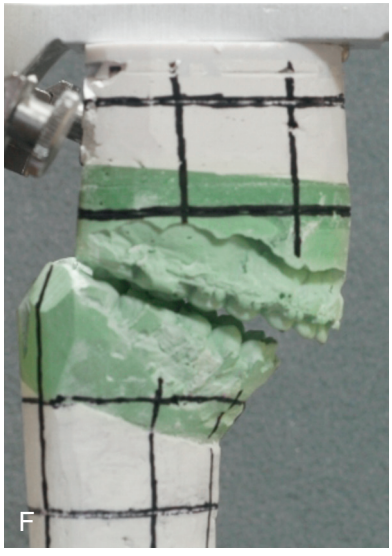
©Elsevier Inc.
Orthognathic Surgery; J.C. Posnick

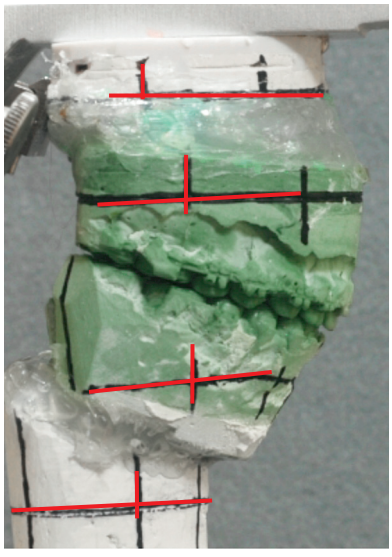
• **Figure 19-12, cont'd C,** Oblique facial views before and after treatment. **D,** Profile views before and after treatment.

Prior to retreatment

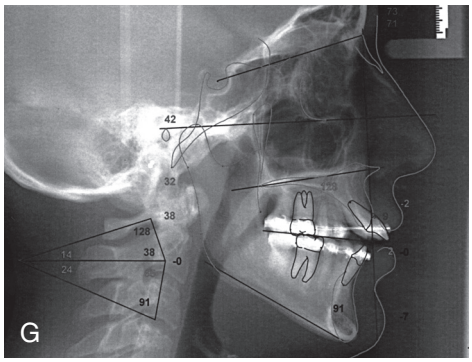
Orthodontics in progress. Spacing between canines and bicuspids requires closure prior to surgery

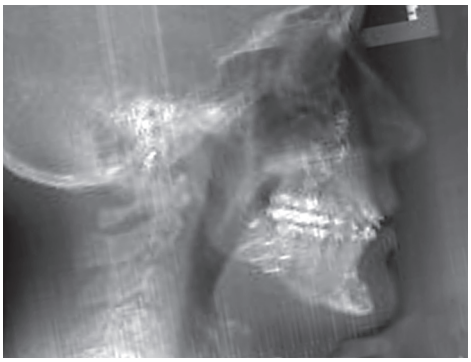
After treatment





Arch expansion





• **Figure 19-12, cont'd E,** Occlusal views before retreatment, with orthodontics in progress, and after the completion of treatment. Note the early gingival recession of the maxillary canine and bicuspid teeth as a result of orthodontic mechanics. **F,** Articulated dental casts that indicate analytic model planning. **G,** Lateral cephalometric radiographs before and after treatment.

©Elsevier Inc.
Orthognathic Surgery; J.C. Posnick

• **Figure 19-13** A 15-year-old girl was referred for surgical evaluation only after orthodontic treatment was well under way. She has primary mandibular deficiency with maxillary arch construction. There is a Class II negative overjet malocclusion despite four bicuspid extractions. The patient agreed to an orthodontic and surgical approach. After orthodontic (dental) decompensation, she underwent surgery that included a maxillary Le Fort I osteotomy in segments (horizontal advancement, counterclockwise rotation, transverse expansion, and cant correction); bilateral sagittal split osteotomies of the mandible (horizontal advancement, counterclockwise rotation, and asymmetry correction); and osseous genioplasty (horizontal advancement). **A,** Frontal views in repose before and 3 years after treatment. **B,** Frontal views with smile before and 3 years after treatment.

©Elsevier Inc.
Orthognathic Surgery; J.C. Posnick

• **Figure 19-13, cont'd C,** Oblique facial views before and 3 years after treatment. **D,** Profile views before and 3 years after treatment.

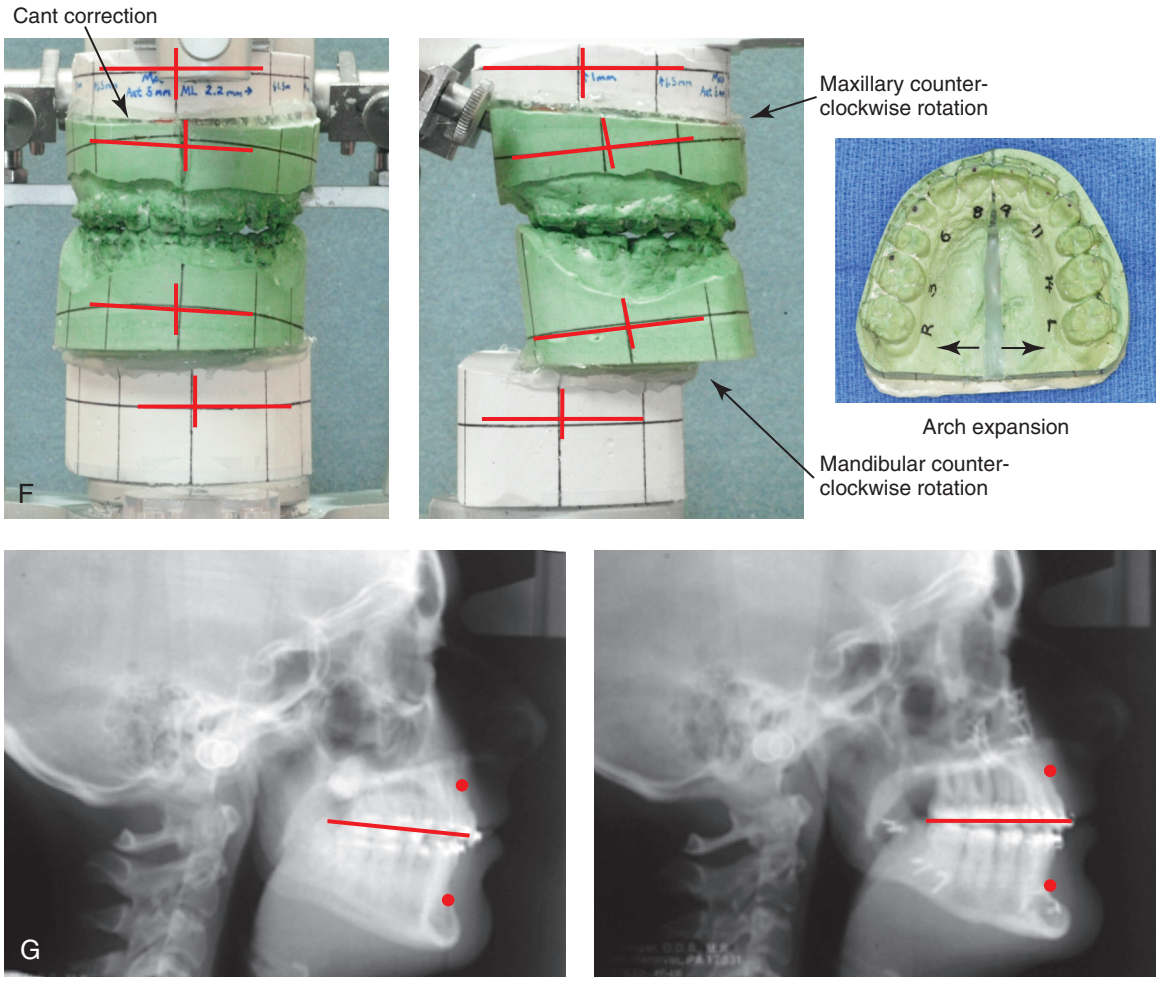
Continued

Prior to treatment

Pre surgery with 4 bicuspid extractions

After treatment

• **Figure 19-13, cont'd E,** Occlusal views before treatment, with orthodontics in progress, and 3 years after treatment.



• **Figure 19-13, cont'd F,** Articulated dental casts that indicate analytic model planning. **G,** Lateral cephalometric radiographs before and after treatment.

• **Figure 19-14** A 16-year-old girl was referred for surgical evaluation only after orthodontic treatment was well under way. Lower bicuspid extractions with the retraction of the anterior dentition were recommended for full decompensation. A computed tomography scan was obtained and confirmed congenital dysplasia of the condyle–glenoid fossa complex; however, there was a functional temporomandibular joint complex on each side. There was no suggestion of condylar resorption now or in the past. The reconstruction included a maxillary Le Fort I osteotomy in segments (arch expansion and horizontal advancement); bilateral sagittal split ramus osteotomies (horizontal advancement); osseous genioplasty (horizontal advancement); and septoplasty and inferior turbinate reduction. The patient is shown before and after treatment. **A,** Frontal views in repose before and after treatment. **B,** Frontal views with smile before and after treatment.

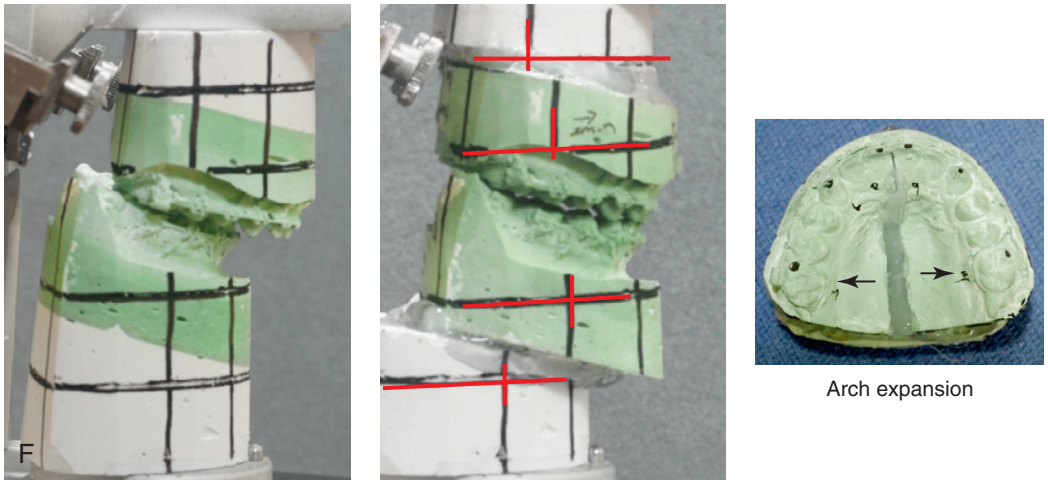
©Elsevier Inc.
Orthognathic Surgery; J.C. Posnick

• **Figure 19-14, cont'd C,** Oblique facial views before and after treatment. **D,** Profile views before and after treatment.

Continued

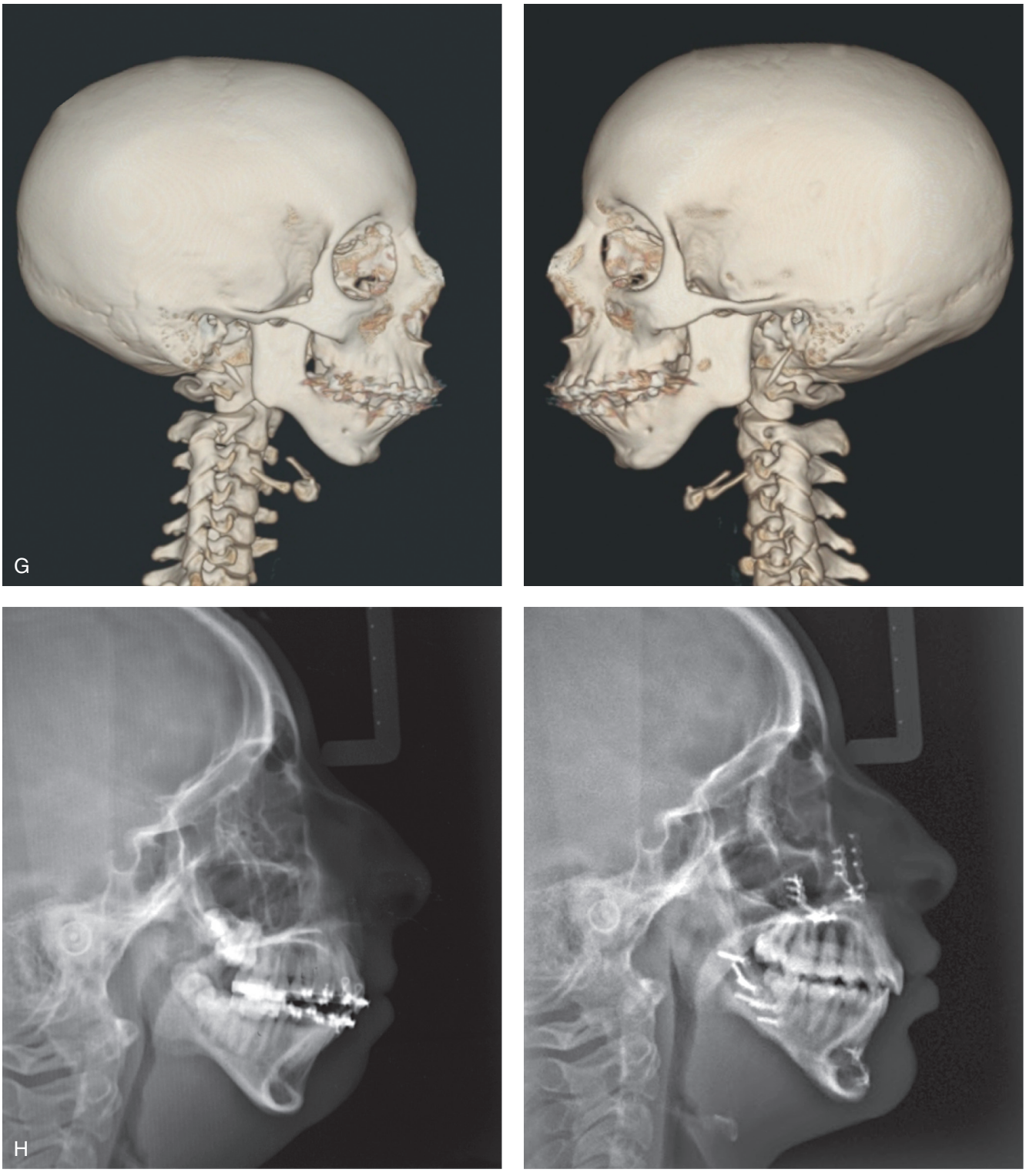
During retreatment with mandibular bicuspid extractions

After treatment



Arch expansion

• **Figure 19-14, cont'd** **E,** Occlusal views with orthodontics in progress and after treatment. **F,** Articulated dental casts that indicate analytic model planning.



• **Figure 19-14, cont'd G,** Computed tomography scan views before reconstruction that indicate congenital dysplasia of the condyle–glenoid fossa complex. **H,** Lateral cephalometric radiographs before and after treatment.

• **Figure 19-15** A 15-year-old girl was referred for surgical assessment only after orthodontic treatment was well under way. She had severe mandibular deficiency and microgenia, and she had already undergone four bicuspid extractions and a full course of orthodontic treatment. An anterior open bite with procumbent maxillary incisors remained. Evaluation was carried out, including a computed tomography scan, a sleep study, and speech and otolaryngology consultations. The computed tomography scan confirmed congenital dysplasia of the condyle–glenoid fossa complex without condylar resorption. Orthodontics were reinitiated, and surgical reconstruction was chosen. The surgical procedures included a maxillary Le Fort I osteotomy (horizontal advancement, vertical shortening, and counterclockwise rotation); bilateral sagittal split ramus osteotomies (horizontal advancement and counterclockwise rotation); and osseous genioplasty (horizontal advancement). A mild anterior open bite has recurred, but this had been stable for several years. A review of the immediate and late postoperative cephalometric radiographs confirmed a consistent sella–nasion–B-point angle without radiographic evidence of condylar resorption (i.e., no skeletal relapse could be documented). Dental relapse in the anterior dentition was felt to be the cause of recurrent malocclusion. **A,** Frontal views in repose before and after treatment. **B,** Frontal views with smile before and after treatment.

• **Figure 19-15, cont'd C,** Oblique facial views before and after treatment. **D,** Profile views before and after treatment.

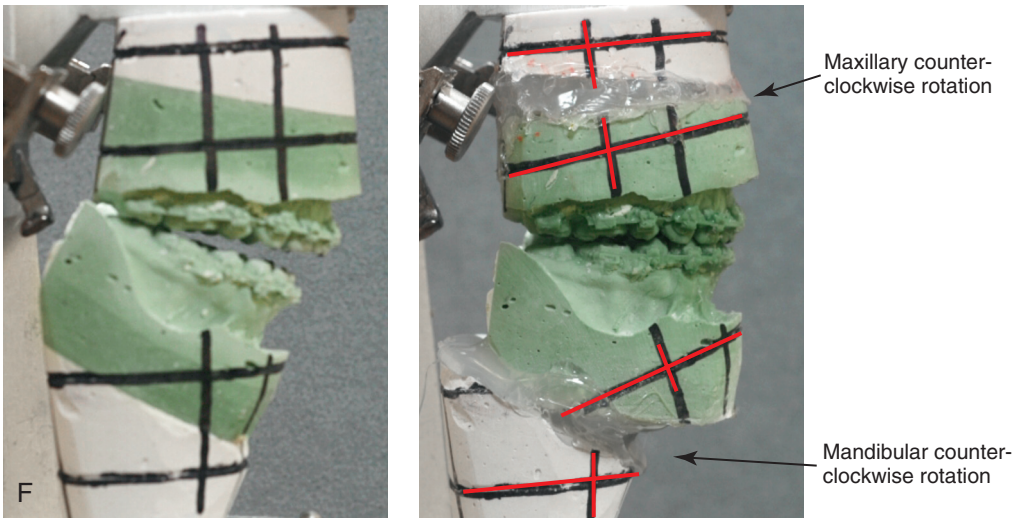
Continued

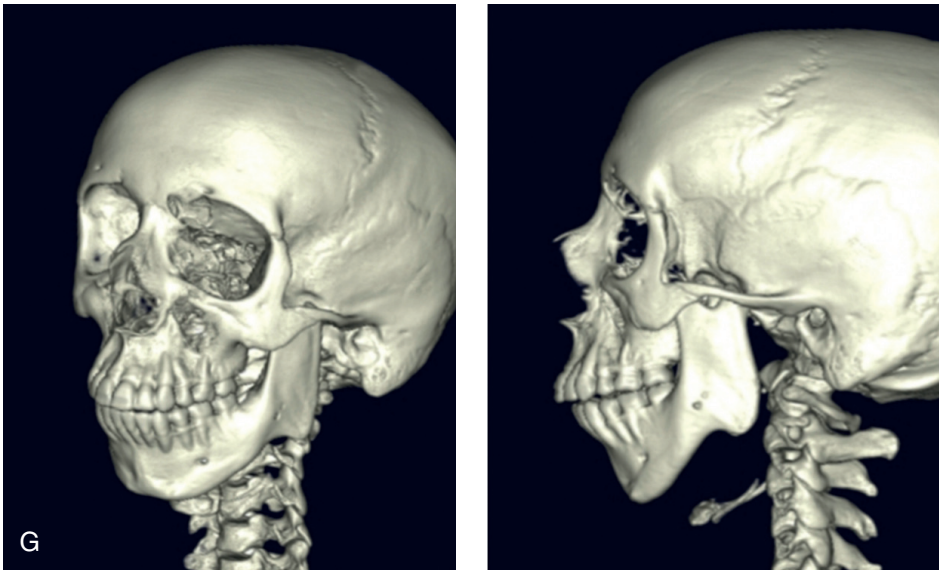
Prior to retreatment with four bicuspid extractions

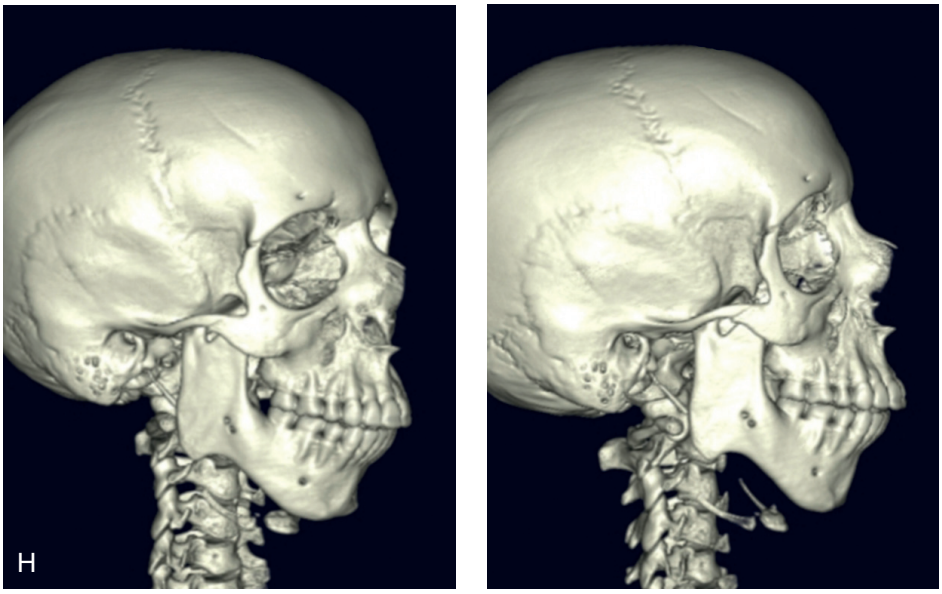
Pre surgery

After treatment

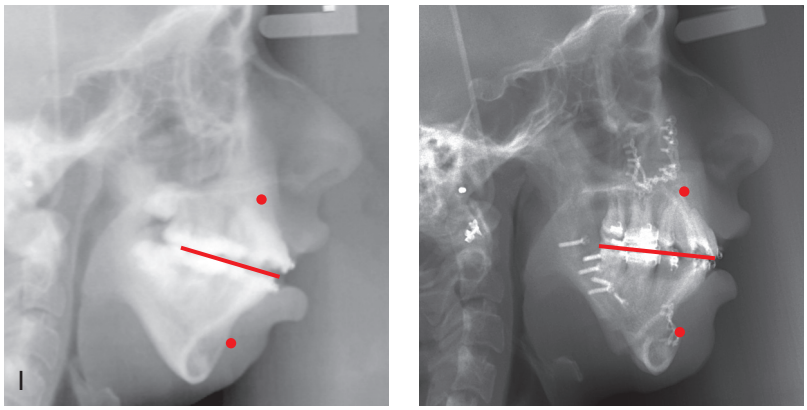
• **Figure 19-15, cont'd E,** Occlusal views before retreatment (after four bicuspid extractions), before surgery, and then after treatment.







• **Figure 19-15, cont'd F,** Articulated dental casts that indicate analytic model planning. **G** and **H,** Computed tomography scans of the maxillomandibular complex that indicate congenital condyle glenoid fossa dysplasia. *Continued*



• **Figure 19-15, cont'd I,** Lateral cephalometric radiographs before and after treatment.

and vertical position of the chin are important (see Chapter 37).

Basic Surgical Approach

In addition to lower jaw advancement (i.e., sagittal ramus osteotomies), maxillary surgery to correct arch-width discrepancies may be required for the individual with primary mandibular deficiency. This is generally accomplished by segmental maxillary osteotomies. Through the Le Fort I down-fracture, any needed improvement in the upper-lip-to-upper-tooth relationship can also be simultaneously carried out via the alteration of the vertical height. Any deficiencies in the horizontal position (i.e., projection) of the maxilla are also managed. The correction of a maxillary midline discrepancy (i.e., yaw), the aesthetic advantages of maxillary plane alteration (i.e., pitch), cant correction (i.e., roll), and intranasal procedures (i.e., septoplasty and inferior turbinate reduction) can also be simultaneously accomplished through the Le Fort I osteotomy (see Chapter 12).

In cases of only borderline maxillary deformity, limiting the surgery to the lower jaw is generally possible to manage the malocclusion and to address aesthetic aspects.

- When the decision is made to limit surgery to the mandible and with only a minimum arch-width discrepancy, the option of narrowing the mandible (i.e., a midline symphyseal split) may be considered.
- When only borderline maxillary vertical deficiency coexists (i.e., not enough to warrant a Le Fort I osteotomy), additional surgical vertical lengthening of the chin may be incorporated to achieve an aesthetic advantage.
- When mandibular deficiency is combined with a less than ideal clockwise rotated and vertically deficient maxilla, the surgically advanced mandible (without simultaneous Le Fort I osteotomy) may not achieve the preferred profile aesthetics (i.e., there will be a suboptimal A-point to B-point relationship in profile). In borderline cases, a vertical lengthening and a limited horizontal advancement osseous genioplasty may provide the necessary degree of aesthetic compensation.

COMMENT: When there are only borderline indications for maxillary surgery, the ability to fine-tune facial aesthetics with a compensating osseous genioplasty may exist (see Chapter 37).[48] Nevertheless, it is important to understand that a percentage of patients who present with primary mandibular deficiency will also have degrees of maxillary dysmorphology that are best served by double jaw surgery.

Postsurgical Orthodontic Maintenance and Detailing

If the patient has a presurgical habit of posturing the mandible forward (i.e., a dual bite), there may also be a tendency to do so during the immediate postoperative healing phase. Intermittent elastics are used during this time 1) to retrain the masticatory muscles, 2) to provide movement in the temporomandibular joints, 3) to increase patient comfort, and 4) to confirm the stability of the occlusion.

If segmental maxillary osteotomies were also carried out, a prefabricated splint will have been secured to the maxillary occlusal surfaces during surgery. The orthodontist sees the patient within 24 hours of splint removal (approximately 5 weeks after surgery) and replaces the maxillary sectional arch wires with a rigid continuous arch wire. The maxillary teeth are ligated together to maintain the arch form. The use of a transpalatal appliance or a palatal plate may also be used to stabilize and maintain the arch form if significant transverse expansion was carried

out.[12] Close orthodontic monitoring for skeletal and dental shifts during the first 6 months after surgery is essential (see Chapter 17).[1,8,9,10,16,22,26,33,54,56,63]

The postsurgical orthodontic objectives should be to bring the teeth into ideal occlusion and to maintain the surgical result. In the patient in whom the curve of Spee is to be fully leveled postoperatively, the use of vertical elastics to close the mid-arch open bites and for the full closure of the bicuspid extraction sites will also be priorities. This process can be initiated immediately after surgery.

Complications, Informed Consent, and Patient Education

The orthodontist is frequently asked to evaluate a preadolescent child who is presenting with primary mandibular deficiency. The clinician may attempt to alter nature's events through growth modification maneuvers.[53] Clinical studies now confirm limited success with a growth modification approach if the goal is to "normalize" the deficient lower jaw. The camouflage orthodontic approach that often follows may neutralize the occlusion, but it will not improve baseline upper airway dysfunction, and it will not enhance the appearance of a retrusive profile. Furthermore, it may have detrimental effects on the periodontium (see Figs. 19-1 through 19-5). The onus is on the clinician to communicate the shortcomings of these isolated orthodontic maneuvers to the patient and the family before beginning treatment.

After a combined surgical and orthodontic approach is agreed to, the clinician's ability to communicate the potential risks, complications, and benefits of the recommended coordinated plan to the patient and the family is essential (see Chapter 17).[19,24] The patient's and the family's informed decision to proceed provides the basis for shared responsibility of the outcome (see Chapter 7).

Temporomandibular Disorders: The Effects of Orthodontics and Orthognathic Surgery

Temporomandibular disorders (TMDs) include various symptoms and signs of the temporomandibular joint, the masticatory muscles, and related structures. The symptoms and signs may include a spectrum of referred head and neck pain; joint noise (e.g., popping, clicking, crepitus); reduced or altered mandibular movements (e.g., muscle spasm or disc displacement with or without reduction); condylar head erosion; and pain on direct palpation of either temporomandibular joint or of the masticatory muscles.

Occlusal features more likely to be associated with symptomatic TMDs include a centric relation to centric occlusion slide of more than 2 mm; overjets of more than 4 mm; and the presence of a skeletal anterior open bite. It is likely for these reasons that White and colleagues found that TMDs were significantly greater among patients with Class II skeletal deformities as compared with those patients with Class III deformities.[58] Westermark and colleagues studied a consecutive series of Class II patients to understand the effects of orthognathic surgery on the signs and symptoms of TMD.[57] Preoperatively, 43% of the patients reported subjective symptoms of TMD; postoperatively, 28% reported such symptoms. This difference suggests an overall beneficial effect of orthognathic surgery for many individuals with Class II mandibular deficiencies.[28] Interestingly, clinical studies indicate that patients with mandibular retrusion and TMD do not improve as much from an orthognathic correction as patients with a Class III skeletal pattern do (see Chapter 9).

Conclusions

In the American and Northern European populations, mandibular deficiency is the most frequently seen developmental jaw deformity. This results in an Angle Class II malocclusion with an excessive overjet and often with a deep overbite. The mandible and the chin region will be deficient, and the maxilla may also be deformed. The basic facial dysmorphology in an individual with primary mandibular deficiency is that of diminished vertical and horizontal strength in the lower third of the face. There will be lack of support for the overlying soft tissues that results in an overly obtuse neck–chin angle, with double-chin formation and early jowling. The tongue will be retropositioned within a small oral cavity and have negative effects on the airway. The laxity of the soft-tissue envelope only worsens throughout the normal aging process.

The correction of this frequently seen facial imbalance requires the orthodontic removal of dental compensations, often with extractions. Orthognathic surgery is performed for the achievement of a stable occlusion, long-term periodontal health, improvements in the upper airway, and the enhancement of facial aesthetics.

References

1. Abeloos J, De Clercq C, Neyt L: Skeletal stability following miniplate fixation after bilateral sagittal split osteotomy for mandibular advancement. *J Oral Maxillofac Surg* 91:624, 1993.
2. Angle EH: *Treatment of malocclusion of the teeth: Angle's system*, ed 7, Philadelphia, 1907, S.S. White.
3. Arnett GW, Bergman RT: Facial keys to orthodontic diagnosis and treatment planning—part I. *Am J Orthod Dentofacial Orthop* 103:299–312, 1993.
4. Arnett GW, Bergman RT: Facial keys to orthodontic diagnosis and treatment planning—part II. *Am J Orthod*

Dentofacial Orthop 103:395–411, 1993.
5. Bell WH, Proffit WR, White RP: *Surgical correction of dentofacial deformities*, Philadelphia, 1980, W.B. Saunders Company, pp 684–843.
6. Bell WH, Schendel SA: Biologic basis for modification of the sagittal split ramus osteotomy. *J Oral Surg* 35:362, 1977.
7. Blair VP: *Surgery of the mouth and jaws*, St. Louis, 1914, Mosby.
8. Blomqvist JE, Ahlborg G, Isaksson S, et al: A comparison of skeletal stability after mandibular advancement and use of two rigid fixation techniques. *J Oral Maxillofac Surg* 55:568, 1997.
9. Bouwman JP, Kerstens HC, Tuinzing DB: Condylar resorption in orthognathic surgery. The role of intermaxillary fixation. *Oral Surg Oral Med Oral Pathol* 78:138, 1994.
10. Broadbend TR, Woolf RM: Our experience with sagittal split osteotomy for retrognathia. *Plast Reconstr Surg* 60:860, 1977.
11. Burstein FD, Cohen SR, Scott PH, et al: Surgical therapy for severe refractory sleep apnea in infants and children: application of the airway zone concept. *Plast Reconstr Surg* 96:34, 1995.
12. Chamberland S, Proffit WL: Short-term a nd long-term stability of surgically assisted rapid palatal expansion revisited. *Am J Orthod Dentofacial Orthop* 139:815–822, 2011.
13. Coben SE: Growth and Class II treatment. *Am J Orthod* 52:5–26, 1966.
14. Cozza P, Baccetti T, et al: Mandibular changes produced by functional appliances in Class II malocclusion: A systematic review. *Am J Orthod Dentofacial Orthop* 129:599e1–599e12, 2006.
15. Epker BN: Occlusal plane alteration in orthognathic surgery [discussion]. *J Oral Maxillofac Surg* 51:740–741, 1993.
16. Epker BN, Wessberg GA: Mechanisms of early skeletal relapse following surgical advancement of the mandible. *Br J Oral Surg* 20:175–182, 1982.
17. Ewing M, Ross RB: Soft tissue response to mandibular advancement and genioplasty. *Am J Orthod Dentofacial Orthop* 101:550–555, 1992.
18. Finn RA: Biomechanical considerations in the surgical correction of mandibular deficiency. *J Oral Surg* 38:257–264, 1980.
19. Friehofer H, Petresevic D: Late results after advancing the mandible by sagittal splitting of the rami. *J Maxillofac Surg* 3:250–257, 1975.
20. Friehofer HP: Results of osteotomies of the facial skeleton in adolescence. *J Maxillofac Surg* 5:267–297, 1977.
21. Gallo W, Moss M, Gaul J: Modification of the sagittal split ramus osteotomy for retrognathia. *J Oral Surg* 34:178–179, 1976.
22. Guernse YL: Stability of treatment results in Class II malocclusion corrected by full mandibular advancement surgery. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 37:668, 1974.
23. Huang CS, Bruce-Ross R: Surgical advancement of the retrognathic mandible in growing children. *Am J Orthod* 82:89–103, 1982.
24. Jackson L: Outpatient orthognathic surgery: review of 205 cases [discussion]. *J Oral Maxillofac Surg* 55:563, 1997.
25. Jacobs JS: Mandibular retrusion, temporomandibular joint derangement, and orthognathic surgery planning [discussion]. *Plast Reconstr Surg* 90:230, 1992.
26. Jäger A, Kubein-Meesenburg D, Luhr HG: Longitudinal study of combined orthodontic and surgical treatment of Class II malocclusion with deep overbite. *Int J Adult Orthod Orthognath Surg* 6:29, 1991.
27. Johnston LE, Jr: Level of agreement in clinicians' perceptions of Class II malocclusions [discussion]. *J Oral Maxillofac Surg* 52:572, 1994.
28. Magnusson T, Ahlborg G, Svartz K: Function of the masticatory system in 20 patients with mandibular hypo- or hyperplasia after correction by a sagittal split osteotomy. *Int J Oral Maxillofac Surg* 19:289, 1990.
29. McNeil RW, West RA: Severe mandibular retrognathism: orthodontic versus surgical orthodontic treatment. *Am J Orthod* 72:176–182, 1977.
30. Newman GV: Prevalence of malocclusion in children six to fourteen years of age and treatment in preventable cases. *J Am Dent Assoc* 52:566–575, 1965.
31. Ngan PW, Byczek E, Scheick J: Longitudinal evaluation of growth changes in Class II Division 1 subjects. *Semin Orthod* 3:222–231, 1997.
32. O'Brian K: Is early treatment for Class II malocclusion effective? Results from a randomized clinical trial. *Am J Orthod Dentofacial Orthop* 120(Suppl 1):564–565, 2006.
33. O'Brian K, Wright J, Conboy F, et al: Effectiveness of early orthodontic treatment with the twin-block appliance: a multi-center, randomized, controlled trial. Part 2: Psychosocial effects. *Am J Orthod Dentofacial Orthop* 124:488–495, 2003.
34. Obwegeser HL: The surgical correction of mandibular prognathism and retrognathia with consideration of genioplasty: part 1. *Oral Surg* 10:677, 1957.
35. Obwegeser HL: The indications for surgical correction of mandibular deformity by the sagittal splitting technique. *Br J Oral Surg* 1:157–171, 1964.
36. Obwegeser HL: Variations of a standard approach for correction of the bird-face deformity. *J Craniomaxillofac Surg* 16:247–265, 1988.
37. Obwegeser HL, Hadjianghelou O: Two ways to correct bird-face deformity. *Oral Surg Oral Med Oral Pathol* 64:507–518, 1987.
38. Otuyemi OD, Jones SP: Long-term evaluation of treated Class II Division 1 malocclusions utilizing the PAR index. *Br J Orthod* 22:171–178, 1995.
39. Phillips C, Bailey LJ, Sieber RP: Level of agreement in clinicians' perceptions of Class II malocclusions. *J Oral Maxillofac Surg* 52:565, 1994.
40. Posnick J, Fantuzzo J, Orchin J: Deliberate operative rotation of the maxillo-mandibular complex to alter the A-point to B-point relationship for enhanced facial esthetics. *J Oral Maxillofac Surg* 64:1687–1695, 2006.
41. Posnick JC: Occlusal plane rotation: aesthetic enhancement in mandibular micrognathia [discussion]. *Plast Reconstr Surg* 91:1241, 1993.
42. Posnick JC, editor: *Mandibular deficiency with or without maxillary deformity. Craniofacial and maxillofacial surgery in children and young adults*, Philadelphia, 2000, W.B. Saunders Company, pp 981–1003.
43. Proffit WR, Fields HW, Jr, Moray LJ: Prevalence of malocclusion and orthodontic treatment need in the United States: estimates from the NHANES III survey. *Int J Adult Orthodon Orthognath Surg* 13:97, 1998.
44. Proffit WR, Phillips C, Douvartzidis N: A comparison of outcomes of orthodontic and surgical-orthodontic treatment of Class II malocclusion in adults. *Am J Orthod Dentofacial Orthop* 101:556–565, 1992.
45. Reichenback E: Uber den zeitpunk kieferchirurgisch-orthod-padischer, Eingriffe, Stsch. *Zahn-Mund-Kierferheilkd* 47:302, 1966.
46. Reyneke JP, Evans WG: Surgical manipulation of the occlusal plane. *Int J Adult Orthodon Orthognath Surg* 5:99–110, 1990.
47. Rosen H: Aesthetics in facial skeletal surgery. *Perspect Plast Surg* 6:1, 1993.
48. Rosen HM: Surgical correction of the vertically deficient chin. *Plast Reconstr Surg* 82:247, 1988.
49. Rosen HM: Occlusal plane rotation: aesthetic enhancement in mandibular micrognathia. *Plast Reconstr Surg* 91:1231, 1993.
50. Rowe NL: The etiology, clinical features and treatment of mandibular deformity. *Br Dent J* 108:45–112, 1960.
51. Schellhas KP, Piper MA, Bessette RW, et al: Mandibular retrusion, temporomandibular joint derangement, and orthognathic surgery planning. *Plast Reconstr Surg* 90:218, 1992.
52. Schendel SA, Wolford LM, Epker BN: Mandibular deficiency syndrome. III. Surgical advancement of the deficient mandible in growing children: treatment results in twelve patients. *Oral Surg Oral Med Oral Pathol* 45:364–377, 1978.
53. Simmons KE, Turvey TA, Phillips C, Proffit WR: Surgical orthodontic correction of mandibular deficiency: five-year follow-up. *Int J Adult Orthodon Orthognath Surg* 7:67–80, 1992.

54. Snow MD, Turvey TA, Walker D, et al: Surgical mandibular advancement in adolescents: postsurgical growth related to stability. *Int J Adult Orthodon Orthognath Surg* 6:143, 1991.
55. Stahl F, Baccetti T, Franchi L, McNamara JA: Longitudinal growth changes in untreated subjects with Class II Division 1 malocclusion. *Am J Orthod Dentofacial Orthop* 134:125–137, 2008.
56. Van Sickels JE: A comparative study of bicortical screw and suspension wires versus bicortical screws in large mandibular advancements. *J Oral Maxillofac Surg* 49:1293, 1991.
57. Westermark A, Shayeghi F, Thor A: Temporomandibular dysfunction in 1516 patients before and after orthognathic surgery. *Int J Adult Orthodon Orthognath Surg* 16:145–151, 2001.
58. White CS, Dolwick MF: Prevalence and variance of temporomandibular dysfunction in orthognathic surgery patients. *Int J Adult Orthodon Orthognath Surg* 7:7–14, 1992.
59. Wolfe SA, Berkowitz S: The mandible. In Wolfe SA, Berkowitz S, editors: *Plastic surgery of the facial skeleton*, Boston, 1989, Little, Brown, p 149.
60. Wolford LM, Chemello PD, Hilliard FW: Occlusal plane alteration in orthognathic surgery. *J Oral Maxillofac Surg* 51:730, 1993.
61. Woldford LM, Schendel SA, Epker BN: Surgical orthodontic correction of mandibular deficiency in growing children (long term treatment result). *J Maxillofac Surg* 7:61–72, 1979.
62. Yu LF, Pogrel MA, Ajayi M: Pharyngeal airway changes associated with mandibular advancement. *J Oral Maxillofac Surg* 52:40, 1994.
63. Zarrinkelk HM, Throckmorton GS, Ellis E, III, et al: A longitudinal study of changes in masticatory performance of patients undergoing orthognathic surgery. *J Oral Maxillofac Surg* 53:777, 1995.

20

Maxillary Deficiency with Relative Mandibular Excess Growth Patterns

JEFFREY C. POSNICK, DMD, MD

- Functional Aspects
- Clinical Characteristics
- Dental Characteristics
- Radiographic Findings
- The Role of Growth Modification in the Preadolescent Patient
- Orthodontic Camouflage Approach
- Surgical Camouflage Approach
- Definite Reconstruction
- Postsurgical Orthodontic Maintenance and Detailing
- Complications, Informed Consent, and Patient Education
- Malocclusion after Orthodontics and Orthognathic Surgery
- Temporomandibular Disorders: The Effects of Orthodontics and Orthognathic Surgery
- Suboptimal Facial Aesthetics after Orthognathic Surgery
- Conclusions

Developmental Class III skeletal problems generally result from maxillary deficiency in combination with relative mandibular excess. Through trial and error, it is now recognized that either growth modification in the preadolescent patient or camouflage treatment in the teenager or adult as a method of managing this type of dentofacial deformity is at best relatively ineffective. In addition, even mild degrees of a Class III facial appearance are aesthetically noticeable as unattractive by the general population.[76] Therefore, the perceived need for treatment (i.e., de facto orthognathic surgery) for the Class III patient—whether by the public, the evaluating clinicians, the affected individual, or the patient's family and friends—will be high.[13,72,78,125]

There is a strong family tendency for a Class III skeletal pattern. The so-called "Habsburg jaw" within the royal European family is well documented.[58,104,106,122,124] The frequency of Class III skeletal patterns among white people of European or North American ancestry is reported to have a prevalence in the low single digits. There is a somewhat higher prevalence of Class III skeletal patterns among Africans than among Caucasians.[27] Data from Japan, Korea, and China confirm a more frequent Class III pattern among Asians than among any other subgroup (i.e., >4%).[28,117] Incidence rates of Class III malocclusion of as high as 13% have been reported in specific regions of Asia.[119] Lew and colleagues examined occlusal parameters in 1050 Chinese schoolchildren who were between 12 and 14 years old.[48] They found an incidence of 12.6% for Class III malocclusion, 58.8% for Class I occlusion, and 21.5% for Class II malocclusion. Samman and colleagues investigated 300 Chinese individuals residing in Hong Kong who were known to have dentofacial deformities and who had been referred to an "orthognathic surgery clinic."[91] After excluding those individuals with cleft lip and palate, 222 study patients remained. Forty-seven percent of these individuals (n = 104) were found to have a Class III skeletal malocclusion.

Despite hereditary tendencies, the development of a Class III jaw disharmony is believed to be multifactorial and complex.[8,33,39,57] Alternatively, it has been documented that environmental factors have a significant effect on vertical facial growth (see Chapter 21). The combination of an individual's hereditary tendency toward a Class III skeletal pattern and a longstanding, forced mouth breathing, open-jaw posture during growth explains the occasional occurrence of a long face growth pattern (i.e., excess vertical growth) with Class III negative overjet malocclusion.[29] An outlier for a Class III skeletal pattern is the impaired midface growth that is seen in many individuals from all backgrounds with repaired cleft palates (see Chapters 32, 33, and 34). Another commonly seen clinical pattern is the individual with maxillary vertical deficiency and a prominent pogonion (i.e., pseudoprognathism) as a result of "over closure" (see Chapter 23).[97,99,109]

An additional source of data regarding the prevalence of individuals with skeletal Class III is the National Health and Nutrition Examination Survey III, a large-scale epidemiologic survey of the U.S. population that was carried out during the 1990s (see Chapter 3).[77] The data from this study confirm that Class III problems increase from early childhood through adolescence.[1,18] This can be explained by the expected late growth spurt in the mandible as compared with the maxilla. The study also confirmed that the presence of 2 mm or more of negative overjet is about twice as likely to occur among African Americans than among Caucasian Americans or Mexican Americans. Data from a U.S. public health survey of youths between the ages of 12 and 17 years indicates that about 14% have a mandibular first molar that is mesial in relation to the maxillary first molar.[42]

With regard to treatment, by the late 1800s, both Edward Angle, an orthodontist, and Vilray Blair, a surgeon, recognized that orthodontic treatment alone could not solve the Class III skeletal problem.[15] Angle suggested that the only possible way to correct a true mandibular protrusion was with a combination of orthodontics and surgery.[2] By the late 1800s, the initial orthognathic procedures were carried out to "set-back" the prominent mandible (see Chapter 2).

Functional Aspects

- Predictable articulation errors in speech result from a Class III malocclusion (see Chapter 8).
- Chronic obstructed nasal breathing is commonly seen in the presence of maxillary deficiency (see Chapter 10).
- Characteristic difficulties with swallowing and chewing as a result of a Class III malocclusion are expected (see Chapter 8).
- Wide lip separation (often with upper lip hypotonia) and mentalis strain required for lip seal are commonly seen in the presence of a significant negative overjet.
- Detrimental long-term effects on the dentition and the periodontium that result from a Class III malocclusion (secondary occlusal trauma), dental crowding, or previous camouflage orthodontics are often seen (see Chapter 6).
- The incidence of temporomandibular disorder (TMD) may be higher among these patients than among individuals with normal jaw harmony and occlusion (see Chapter 9).

Clinical Characteristics

Profile View

- Long chin–neck length and acute angle with a visually accentuated inferior border of the mandible
- Prominent chin with a reduced labiomental fold
- Protrusive and thick lower lip and vermillion
- Sunken-appearing midface (A negative vector relationship between the ocular globe and the lower eyelid as a result of anterior maxillary deficiency is a frequent finding.)
- Deflated, short, hypotonic appearance of the upper lip, with a thin vermillion
- Acute nasolabial angle
- Columella of the nose oriented more horizontally

Frontal View

- Sunken-appearing cheeks (i.e., the impression of flat cheek bones)
- Middle one third of the face appears flat and long
- Increased scleral show (i.e., pulling down of the lower lids)
- Narrow alar base with paranasal flattening
- Flat and relatively short upper lip with reduced vermillion exposure
- Normal to deficient maxillary tooth to upper lip relationship frequently seen
- Mandible appears prominent
- Reduced labiomental fold

Dental Characteristics

- Minimal attached gingiva over the labial aspects of the mandibular anterior teeth is frequently seen (see Chapter 6).
- Mandibular incisors are usually upright or lingually inclined.
- Interdental spacing with flared mandibular incisors may also be seen, especially in the presence of an anterior open bite with lip incompetence.
- Class III malocclusion (molar and canine) with anterior open bite, negative overjet, and posterior crossbites is frequent.
- Narrow maxillary arch width is seen as excess negative space in the buccal corridors.
- Dental crowding with flared maxillary incisors is frequent.

Radiographic Findings

- Maxillary horizontal deficiency
- Concave anterior maxilla consistent with a negative vector relationship of the ocular globe to the lower eyelid
- Flat, counterclockwise-rotated maxillary plane
- Procumbent maxillary incisors
- Flat base of the nose with retrusive pyriform rims
- Altered lower anterior facial height (either short or long)
- B-point is anterior to or in front of A-point in profile (i.e., natural head position)
- Negative overjet to the occlusion
- Prognathic-appearing mandible
- Lingually inclined mandibular incisors frequently found
- Chin morphology varies, but it is generally dysmorphic. Frequent patterns of chin dysmorphology include vertical excess with either a prominent or flat pogonion.

The Role of Growth Modification in the Preadolescent Patient

Since the mid 1800s, clinicians have developed and used devices to attempt to correct and prevent a worsening Class III skeletal pattern during childhood. Despite this extensive history, little long-term success has been documented involving the use of these devices. Techniques have included use of a *chin cup* to "hold back" the mandible; a *face mask* to "grow" the maxilla; and, more recently, the use of *skeletal anchorage devices and elastics* in an attempt to do both.

Most studies have found no difference in the ultimate size of the mandible between children with Class III conditions who were treated with a *chin cup* and those who were not.[105] Wylhelm-Nold and colleagues presented some evidence that the use of the chin cup in preschool-aged children was able to decrease mandibular anterior growth if good compliance was achieved.[123] Unfortunately, this did not prevent the mandible from continuing an excess growth pattern later during childhood, after the chin cup treatment was stopped. In fact, a rebound accelerated growth effect was documented. What chin cup therapy may accomplish is a change in the growth vector of the mandible, with more downward and backward (clockwise) rotation.[37] These secondary changes have their own negative effects on facial morphology and occlusion (i.e., the law of unintended consequences).

With the realization that the presenting maxillary deficiency should be the primary focus of the correction for most skeletal Class III individuals, growth modification moved toward methods of stimulating the upper jaw. The *Delaire face mask* has been used extensively to pull the maxilla forward.[24] The work of Baccetti and McNamara confirmed that use of the Delaire face mask for the individual with maxillary deficiency is more likely to have effect when it is used before the patient is 8 years old.[6,21,92] After this age, tooth movement (dental compensation) and mandibular clockwise rotation will be the likely consequences of this form of treatment rather than the hoped for horizontal maxillary projection. In addition, many children experience recurrence of the deformity after face mask therapy is discontinued. It is unlikely that face mask (for the maxilla) and chin cup (for the mandible) interventions will be effective in the long term for preventing the need for orthognathic surgery in all but mild cases. An accepted guideline is that more than 2 to 4 mm of reverse overjet in the preadolescent child indicates that surgery will eventually be needed if both facial and dental correction are sought.

DeClerck and colleagues used orthopedic traction on the maxilla and compression on the mandible with temporary mini-plate *skeletal anchorage and elastics* in a limited number (n = 3) of 10- to 11-year-old children presenting with skeletal Class III malocclusions.[25] Pure bone-borne orthopedic forces were applied with intermaxillary elastics on the skeletally anchored mini-plates. This approach was shown to either enhance midface growth or to hold back the mandible; in any case, the intent was to produce a corrected overjet. Although the authors were encouraged, many unknowns remained, including the ideal age for this type of orthopedic traction; the effect of the direction of force on the rotation of the palatal and mandibular planes; the long-term effects on the temporomandibular joints; and the need for retention to prevent catch-up growth of the mandible or relapse of the maxilla afterward. DeClerck's ongoing prospective clinical trial of this promising work on a larger sample of patients (as well as the work of other independent researchers) will help to answer these questions and to determine the true effectiveness of a growth-modification approach that involves temporary skeletal anchorage and elastics.[21,92]

Orthodontic Camouflage Approach

The decision to proceed with an orthodontic camouflage approach frequently implies that orthopedic growth modification mechanisms to overcome the Class III pattern were either ineffective earlier during the patient's life or that the patient presents to the orthodontist at an age at which such mechanisms are not feasible. In the adolescent or adult, when a significant reverse overjet remains, the possibility of achieving even the limited parameter of a neutralized occlusion with the use of only orthodontic maneuvers is questionable. In fact, doing so may require the further displacement of the teeth outside of the supporting alveolar bone as a means of compensation for the underlying jaw discrepancy. The extraction of the mandibular first premolars in combination with Class III elastics and extraoral forces on the maxilla (i.e., the proclination of the maxillary incisors) will improve the occlusion for the Class III patient; however, treatment generally falls short of expectations and rarely produces acceptable facial aesthetics. Even minimal retraction of the lower incisors often magnifies the chin's prominence, which is likely a primary reason why the patient sought treatment. Furthermore, camouflage orthodontics will not succeed in altering any combined vertical skeletal problems. In fact, the orthodontic mechanisms that are used to reposition the dentition often extrude the posterior teeth, thus further accentuating the vertical facial disproportion.

Individuals who present to the orthodontist with a Class III skeletal pattern will usually have dental compensation that developed as an adaptive biologic mechanism during growth. Typically, the upper incisors are at least somewhat proclined relative to the maxillary basal bone. The lower incisors are often overly upright relative to the basal bone of the mandible. If an orthodontic camouflage approach is chosen, then the upper incisors are proclined, and the lower incisors are retropositioned even further.

It is not uncommon for the orthognathic surgeon to see an adult in consultation who is emotionally exhausted after having undergone failed growth modification followed by disappointment after additional years of camouflage orthodontics. The individual may present with 1) significant residual malocclusion (i.e., effects on speech, swallowing,

and chewing) 2) deteriorating periodontal health (i.e., crest bone loss and gingival recession associated with lower anterior and upper posterior teeth), 3) often with chronic upper airway obstruction (i.e. deviated septum, hypertrophic inferior turbinates, and tight nasal apertures) and 4) self-esteem and body image issues related to the facial disproportion and disappointment with failed treatment (Figs. 20-1 through Fig. 20-4).

All too often, the orthodontic camouflage approach was centered on the achievement of a neutralized occlusion as the primary objective, without full disclosure to the patient and his or her family. If the goals of treatment are to achieve long-term ideal dental health, improved facial aesthetics and an open upper airway, few treatment plans for the Class III patient would include a camouflage approach. Clear contraindications to an orthodontic camouflage approach include the following:

- Moderate or severe Class III patterns (i.e., >4 mm of reverse overjet)
- Moderate to severe vertical skeletal discrepancies
- Significant dental crowding in the lower jaw and significant anterior dental protrusion in the upper jaw
- A concern that the planned orthodontic mechanics may cause periodontal deterioration
- A desire for enhanced facial aesthetics
- A need to open the upper airway

Surgical Camouflage Approach

Some orthodontists have been buoyed in their efforts to neutralize the occlusion in the individual with a skeletal Class III condition on the basis of a belief that limited cosmetic surgery can improve the facial appearance and thus avoid major orthognathic procedures. Attempts at surgical camouflage for the patients with a Class III skeletal pattern may include a combination of reduction genioplasty; onlay grafting to the midface; upper lip augmentation; lower lip reduction; and neck and chin soft-tissue procedures. In general, these camouflage surgical techniques are ineffective, irreversible, and disappointing.

Often, a prominent chin and apparent flat cheek bones are perceived by the patient to be the aesthetic issues. Although a reduction genioplasty seems to be a logical approach, the surgical reduction of the prominent chin as an isolated procedure in the absence of orthognathic surgery rarely satisfies the facial aesthetic concerns. "Setting back the chin" generally leads to drooping (i.e., ptosis) of the associated soft tissues; irregularities of the mentalis muscles when in function; and a double-chin appearance of the neck. *Submental lipectomy* may then be considered in an attempt to improve the chin ptosis and the appearance of the neck, but with only limited effects. *Onlay grafts or implants* involving the anterior maxilla have been used in an attempt to camouflage the midface deficiency. These perinasal implants run the risk of looking and feeling unnatural. *Augmentation of the zygomas and the infraorbital rims* is generally misguided and will produce an unnatural and "operated" look. *Upper lip augmentation* and *lower lip reduction* procedures in the Class III patient also tend to look and feel unnatural.

To be fair to the patient and his or her family, the limitations of compensating orthodontics followed by surgical camouflage should be disclosed before the initiation of treatment.

Definitive Reconstruction

Timing of Orthognathic Surgery

Skeletally immature individuals with true mandibular excess can be expected to outgrow any early orthodontic or surgical corrections and later require further treatment.[7,17,83,90] Indirect methods of assessing growth maturity (e.g., a hand–wrist film to determine bone age) are not accurate enough to be used in isolation for planning the timing of surgery. A reasonable method to assess growth maturation is through the analysis of *serial lateral cephalometric radiographs*. These radiographs can be used in two ways: for a cervical vertebral analysis or to make sequential direct mandibular measurements. Surgery is delayed until the deceleration of growth can be documented. The use of a *quantitative condylar head bone scan* can also be helpful to evaluate the growth of the mandible in the adolescent, but both false-positives and false-negatives may occur. If the Class III condition is largely the result of maxillary deficiency, gauging the preferred timing of surgery is more straightforward, because the late postsurgical growth of the mandible is less likely (see Chapter 17).

Alexander and colleagues completed a study of craniofacial growth changes in Caucasians with untreated Class III malocclusions by using longitudinal cephalometric records (n = 103).[1] In girls, the mandibular growth spurt generally occurred between the ages of 10 and 12 years. In boys, the growth spurt was usually between the ages of 12 and 15 years. In some of the Class III subjects, limited degrees of mandibular growth relative to that of maxillary growth were documented to continue after the adolescent spurt. After the age of 13 years in girls and 15 years in boys and until closer to the age of 17 years, annual increments of mandibular growth were often between 1.5 mm and 2.0 mm, whereas increments of midface length dropped to less than 1 mm. The authors concluded that the differential growth behavior of the two jaws among individuals with Class III conditions may result in recurrent jaw disharmony and malocclusion when orthodontic or surgical treatment is undertaken before or during puberty.

Pre-Orthodontic Periodontal Checklist (see Chapter 6)

- Assess for active gingivitis or periodontitis and the need for treatment.

Text continued on p. 694

• **Figure 20-1** A 34-year-old male arrived for the evaluation of a longstanding jaw deformity with malocclusion, chronic obstructive nasal breathing, and periodontal issues. Since his early childhood years, the patient was known to have a maxillary deficiency with a relative mandibular excess growth pattern. The family elected a camouflage orthodontic approach that included four bicuspid extractions and the introduction of dental compensation in an attempt to neutralize the occlusion when the patient was 10 to 15 years old. He is a forced mouth breather as a result of increased nasal airway resistance. The patient underwent septoplasty when he was 20 years old, but significant improvement of the airway did not occur. His general dentist confirmed labial bone loss and gingival recession of the mandibular anterior and maxillary posterior dentition. He was referred to this surgeon, and a comprehensive orthognathic and dental rehabilitation approach was selected. Consultation with a periodontist, an orthodontist, and an ear, nose, and throat specialist was carried out. The patient was confirmed to have residual deviation of the septum and enlarged inferior turbinates, which explained the continual difficulty that he had breathing through his nose. A degree of root resorption of the anterior dentition was confirmed. The need for gingival grafting to attain improved root coverage and to generate a wider band of attachment was recommended for tooth nos. 3, 11, 14, 19, 20, 23 through 26, 29, and 30. This will be followed by the orthodontic removal of dental compensation and then orthognathic and intranasal procedures to improve long-term dental health, to enhance facial aesthetics, and to open the upper airway. **A,** Frontal facial and occlusal views before retreatment. **B,** Profile facial and lateral cephalometric radiograph before retreatment. **C,** Panorex radiograph before retreatment.

• **Figure 20-2** A 26-year-old man arrived for surgical evaluation. Since the mixed dentition, he had been recognized as having a developmental jaw deformity characterized by maxillary deficiency in combination with relative mandibular excess. Camouflage orthodontic treatment that included four bicuspid extractions was carried out when the patient was between 12 and 16 years old. A residual Class III negative overjet with an anterior open-bite malocclusion remains. The patient has never been able to breathe well through the nose as a result of septal deviation and inferior turbinate enlargement. Secondary occlusal trauma resulted in the need for root canal therapy and crowns in all four posterior quadrants. There is labial bone loss and gingival recession associated with the anterior mandibular teeth. A comprehensive dental rehabilitative and reconstructive approach will require periodontal evaluation and treatment followed by orthodontic decompensation and then by jaw and intranasal surgery. **A,** Frontal facial and occlusal views before retreatment. **B,** Profile facial view and lateral cephalometric radiograph before retreatment. **C,** Panorex radiograph before retreatment.

• **Figure 20-3** A 30-year-old man arrived for the evaluation of a longstanding jaw deformity with malocclusion, chronic obstructive nasal breathing, and an awareness of periodontal issues. Since his early childhood years, he was known to have a jaw deformity with malocclusion, including an anterior open bite. When he was between 8 and 12 years old, he underwent orthodontic camouflage treatment in an attempt to close the open bite; this included full bracketing and the use of heavy anterior elastics. By the time that he graduated from high school, he was conscious of a significant recurrent anterior open bite with dental crowding. Throughout his college years, he was aware of gingival recession on the labial aspect of many of the anterior teeth of the maxilla more so than the mandible. He was sent by his general dentist for an orthodontic evaluation and then to this surgeon for an opinion. The patient was referred for periodontal evaluation with confirmation of significant labial bone loss of the anterior dentition (more so on the maxilla than the mandible) and gingival recession. A comprehensive approach was recommended; this would include gingival grafting and four bicuspid extractions, with orthodontic retraction of the anterior dentition into the solid alveolar bone. This would be followed by jaw reconstruction, finishing orthodontics, and periodontal surveillance. **A,** Frontal facial and occlusal views before retreatment. **B,** Profile facial view and lateral cephalometric radiograph before retreatment. **C,** Panorex radiograph before retreatment.

• **Figure 20-4** A 29-year-old man arrived for the evaluation of a longstanding developmental jaw deformity characterized by maxillary deficiency with relative mandibular excess. There is malocclusion and chronic obstructive nasal breathing. By the time he was 13 years old, he was referred by his pediatric dentist to an orthodontist for the evaluation of a Class III negative overjet malocclusion. He then underwent a 2-year course of compensatory orthodontics in an attempt to neutralize the occlusion. He was left with an Angle Class III negative overjet and early signs of gingival recession. The patient also has a history of stress-induced asthma and chronic obstructed nasal breathing. He uses Flonase, Singulair, and other medications in an effort to open the nasal airway, and he is also being seen by an allergist. For his gingival recession, he was referred to a periodontist, and connective tissue grafting was carried out for the lower anterior dentition on several of the maxillary posterior teeth. An orthodontic consultation confirmed crowding, the proclination of the maxillary anterior dentition, and uprighted mandibular anterior teeth. Evaluation of the upper airway documented a deviated septum and hypertrophic inferior turbinates. An orthodontic and surgical approach was recommended to limit further secondary dental trauma, to stabilize the periodontium, and to correct the occlusion. **A,** Frontal facial and occlusal views before retreatment. **B,** Profile facial and lateral cephalometric radiographs before retreatment. **C,** Panorex radiograph before re-treatment.

- Assess the current level of oral hygiene and the need for a home care program.
- Assess for the effects of occlusal trauma and malocclusion on the periodontium.
- Assess the level of attached gingiva and the need for augmentation.
- Confirm the adequacy of the alveolar bone and the need to create space with the use of extractions.
- Assess the need for restorations to remove decay, to replace deficient enamel, and to achieve a healthy gingival interface.

Orthodontic Preparation for Orthognathic Surgery

1. Before orthodontic decompensation, assess the level of attached gingiva, the position of the labial and lingual plates, and the alveolar crest heights. Consider the effects of any planned orthodontic maneuvers on the periodontium.
2. Make decisions about the tooth (root) size to alveolar volume discrepancies in each arch and the need for extractions to establish and preserve periodontal health.
3. Create a plan (i.e., separate surgical and orthodontic objectives) to achieve preferred anteroposterior and vertical incisor positions in the maxilla and the mandible.
4. Understand how the orthodontic objectives become more limited when they are combined with the planned surgical procedures (e.g., maxillary segmentation, bimaxillary osteotomies) to then achieve the final desired arch form, midline positioning, occlusion, and facial symmetry and proportions.
5. Make decisions about orthodontic mechanics and anchorage requirements to achieve the chosen objectives.
6. At the end of the presurgical orthodontic phase (i.e., 4 to 6 weeks before surgery), full-dimensional stabilizing (i.e., passive) arch wires should be in place. The placement of surgical hooks can be done just before surgery.

Leveling the Mandibular Arch

When an accentuated curve of Spee is present in the lower arch of a patient with Class III malocclusion, it is managed through a combination of intrusion of the incisors and extrusion of the premolars or molars.[20,49] In preparation for surgery, repositioning the lower incisors forward is generally advantageous for the Class III skeletal pattern. Therefore, lower arch extractions are rarely indicated. If limited labial bone is present, a periodontal evaluation is recommended before the incisors are orthodontically moved forward. If gingival recession has occurred or if the gingiva is genetically inadequate, augmentation grafting procedures are indicated (see Chapter 6).

Leveling of the Maxillary Arch

If the upper arch is narrow, then orthodontic leveling is performed in segments in anticipation of surgical management.[8,14,16,22,71,88,95,114] If the curve of Spee is excessive as a result of vertical skeletal dysplasia, then leveling is also done in segments in preparation for surgical correction (see Chapter 17).

In the presence of maxillary deficiency, limited space to uncrowd the upper teeth may leave the roots tipped outside of the solid alveolar housing. In this circumstance, maxillary first bicuspid extractions with space closure before surgery is preferred. With only borderline crowding, extractions are often not carried out. Limited dental space deficiency can often be managed through the judicious use of interproximal reduction. In addition, the aesthetic consequences of mildly proclined incisors (i.e., A-point to B-point discrepancy in profile) can be improved via the clockwise rotation of the maxillomandibular complex at the time of surgery (see Chapters 12 and 13).

Immediate Presurgical Planning

The immediate presurgical planning takes into account collaborative clinical efforts between the orthodontist, the surgeon, the patient, and the patient's family that will have been ongoing since the initial consultation visits.[32,74,75,84,93,98] Depending on the condition of the dentition and the supporting structures, the restorative dentist and the periodontist may also have been intricately involved.

Patient-specific medical and dental records are reviewed, including radiographs (e.g., computed tomography scans; panoramic, lateral, and posteroanterior cephalometric and periapical radiographs); photographs (e.g., facial, occlusal); and dental models.

Immediate presurgical records (i.e., within 2 to 6 weeks of surgery) will most importantly include facial measurements taken during direct visual examination; alginate impressions of the maxilla and the mandible; a centric relation bite registration; and a face-bow registration.

First, broad-stroke clinical decisions are made involving the preferred vector changes for each jaw; only then are the precise millimeter distances and angular changes required for each jaw to achieve the desired results determined (see Chapter 12). Analytic model planning is carried out on the articulated dental casts with use of the Erickson Model Table. Splints are constructed to assist with the achievement of the precise occlusion and the preferred facial aesthetics in the operating room (see Chapter 13).

During surgical planning, consideration should be given to the following eight aspects (see Chapter 12):

1. *Assess the degree of horizontal advancement required in the midface as measured at the maxillary incisal edge.*

   A nose that appears to be excessively large in a patient with a skeletal Class III condition favors the horizontal advancement of the maxilla. Repositioning and projecting the upper jaw forward will diminish the apparent size of the nose. An obtuse nasolabial angle with a hypotonic upper lip also favors maxillary

horizontal advancement; doing so will improve the nasolabial angle and provide fullness to the upper lip. A constricted nasal aperture and a depressed base of the nose also benefit from maxillary advancement. A fully projected midface fills out the pyriform rims and provides needed width to the alar base. For these same reasons, the individual who appears to have flat upper cheeks with pulled-down lower eyelids (i.e., scleral show) benefits from Le Fort I osteotomy with advancement and clockwise rotation.

2. *Assess the degree of vertical change required in the midface as measured at the maxillary incisal edge.*

   Individuals with maxillary deficiency and relative mandibular excess generally present with varying degrees of either vertical midface excess or deficiency. A decision regarding the preferred aesthetic vertical position of the maxillary incisors should be made. Anterior vertical lengthening and clockwise rotation are frequent preferred vector changes used to achieve enhanced midface aesthetics in the patient with a Class III growth pattern.
3. *Assess the potential advantages of clockwise rotation of the maxillary plane to achieve preferred A-point to B-point positioning (i.e., pitch orientation).*

   Clockwise rotation of the maxillomandibular complex is generally required to enhance a positive vector relationship between the ocular globe and the lower eyelid.
4. *Clarify any maxillary midline to facial midline discrepancies and their planned correction (i.e. yaw orientation).*

   When doing so, consideration is given to the maxillary yaw orientation and the anticipated yaw effects on facial symmetry.
5. *Identify any canting (i.e., roll orientation) of the maxilla and its planned correction.*

   If more than 4 degrees of cant are present, this will be noticeable to even the untrained eye at conversational distance (see Chapter 12).
6. *Identify the advantages of segmentation of the upper jaw to achieve the preferred maxillary arch form and occlusion.*

   Determine the preferred location of the interdental osteotomies to best achieve the patient's objectives. This will have been discussed with the treating orthodontist before the initiation of treatment.
7. *Assess the baseline chin morphology.*

   Anticipate the location of the pogonion after jaw repositioning. Consider the advantages of osseous genioplasty and the preferred ultimate horizontal and vertical position of chin.
8. *Assess the baseline pyriform rim, floor of nose, and anterior nasal spine morphology.*

   Anticipate the location of these structures after maxillary repositioning and the advantages of recontouring them to achieve the ultimate objectives.

Basic Surgical Approach

Regardless of the cause of the Class III skeletal pattern, the common aesthetic impression in profile is of a flat or concave midface with a long and forward-projecting chin.[89] The classic dentofacial deformity of maxillary deficiency with relative mandibular excess will require at a minimum a Le Fort I osteotomy with horizontal advancement.[30,31,38,62,85,86,112] The need for additional vector changes in the maxilla should always be considered (e.g., transverse widening, vertical adjustment, maxillary midline to facial midline correction, cant correction, clockwise maxillary plane rotation).[121]

The Le Fort I osteotomy (often performed in segments) with three-dimensional repositioning is generally combined with sagittal split ramus osteotomies to place the distal mandible into ideal occlusion at the new maxillary location.[9-12,50-52,63-66,70,87,94,110,113] In most cases, the full overjet deficiency is corrected in the maxilla. Clockwise rotation of the maxilla and the mandible will generally leave the mandibular incisor unchanged in the sagittal (i.e., horizontal) plane. However, the pogonion will have moved posteriorly.

An osteotomy of the chin with horizontal or vertical change is also frequently desirable.[36] If chronic nasal obstruction is present, then procedures to improve the airway in accordance with the physical examination (i.e., septoplasty, inferior turbinate reduction) may be simultaneously accomplished (Figs. 20-5 through Fig. 20-20).[73,75]

In general, well thought out maxillary surgical maneuvers (i.e., horizontal advancement, clockwise rotation) will also correct the presenting negative vector relationship between the ocular globe and the lower eyelid. In doing so, the overlying cheek and periorbital soft tissues are immediately enhanced. This also limits the unattractive eyelid and midface aging effects that would otherwise occur (i.e., soft-tissue descent, rounding of the palpebral fissure, lengthening of the lower lid, loss of cheek region soft-tissue prominence; see Chapter 25).

When limiting the surgery to one jaw (either through mandibular set-back or Le Fort I advancement), the immediate correction of the occlusion may be possible, but a suboptimal outcome is likely. A one-jaw approach is rarely preferred from the overall perspective of facial aesthetics, occlusal stability, and upper airway management. A Le Fort I osteotomy in combination with mandibular ramus osteotomies that incorporates clockwise rotation of the maxillomandibular complex is generally needed to achieve midface fullness, to improve the projection of the nasal tip and, to enhance the upper lip to tooth aesthetics (see Figs. 20-5 through 20-20).

Demetriades and colleagues confirmed the clinical importance of considering the airway when formulating a treatment plan for the correction of a skeletal Class III pattern.[26] They reported on 26 patients who presented with developmental dentofacial deformities that were

Text continued on p. 753

©Elsevier Inc.
Orthognathic Surgery; J.C. Posnick

• **Figure 20-5** A 17-year-old high school student was referred by his orthodontist for surgical evaluation. A non-extraction orthodontic approach had previously been carried out, without success. The Class III negative overjet anterior open-bite malocclusion required redo orthodontics and jaw surgery. Because the patient had a lifelong history of nasal obstruction and consistent physical findings (i.e. septal deviation and inferior turbinate hypertrophy), intranasal procedures were also planned. When orthodontic decompensation was complete, the patient's surgery included maxillary Le Fort I osteotomy in segments (arch expansion, horizontal advancement, vertical lengthening, and clockwise rotation); sagittal split ramus osteotomies (limited corrections); osseous genioplasty (vertical reduction and minimal horizontal advancement); and septoplasty and inferior turbinate reduction. **A,** Frontal views in repose before and after treatment. **B,** Frontal views with smile before and after treatment.

©Elsevier Inc.
Orthognathic Surgery; J.C. Posnick

• **Figure 20-5, cont'd C,** Oblique facial views before and after treatment. **D,** Profile views before and after treatment.

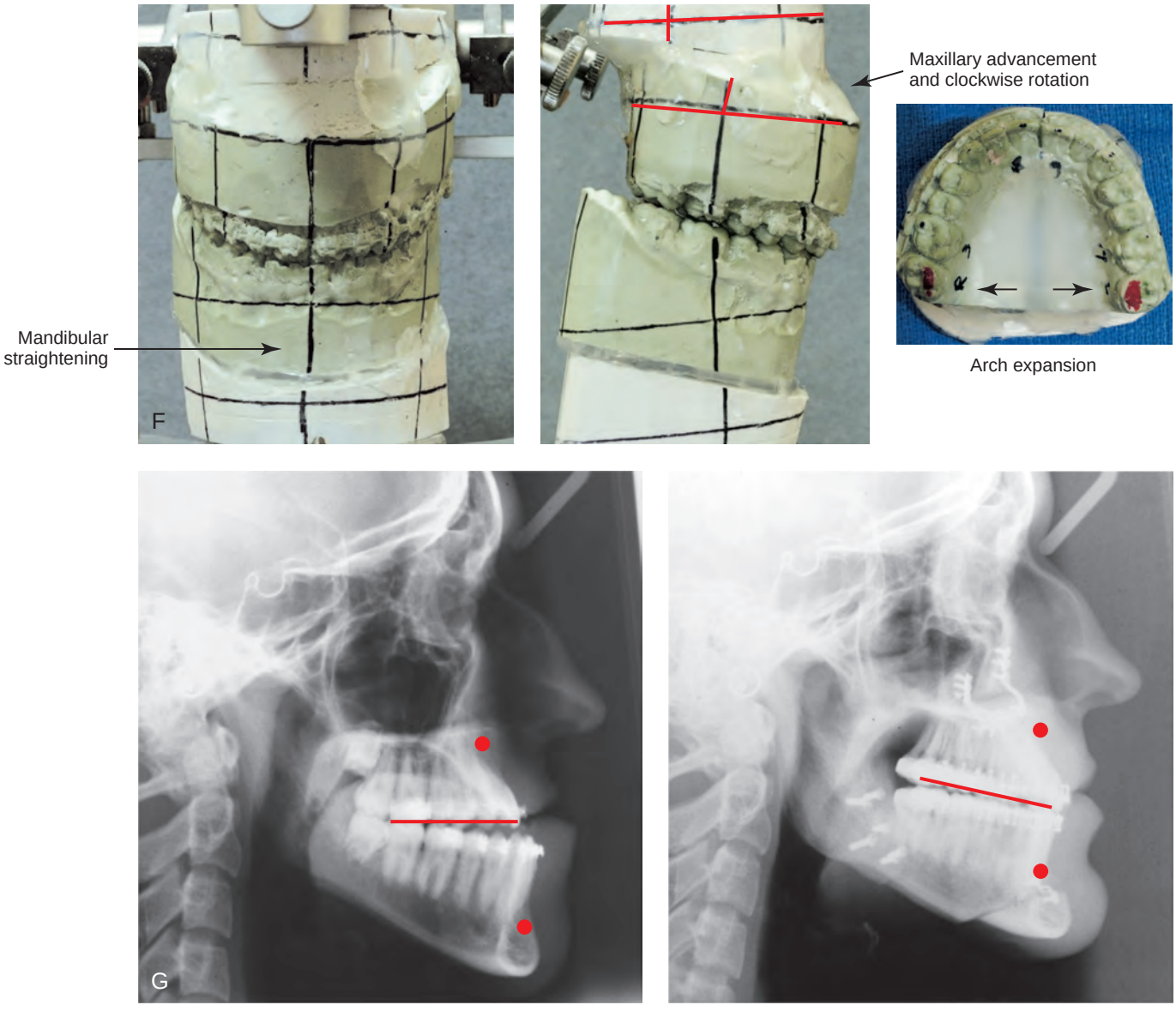
Continued

Prior to retreatment

Pre surgery

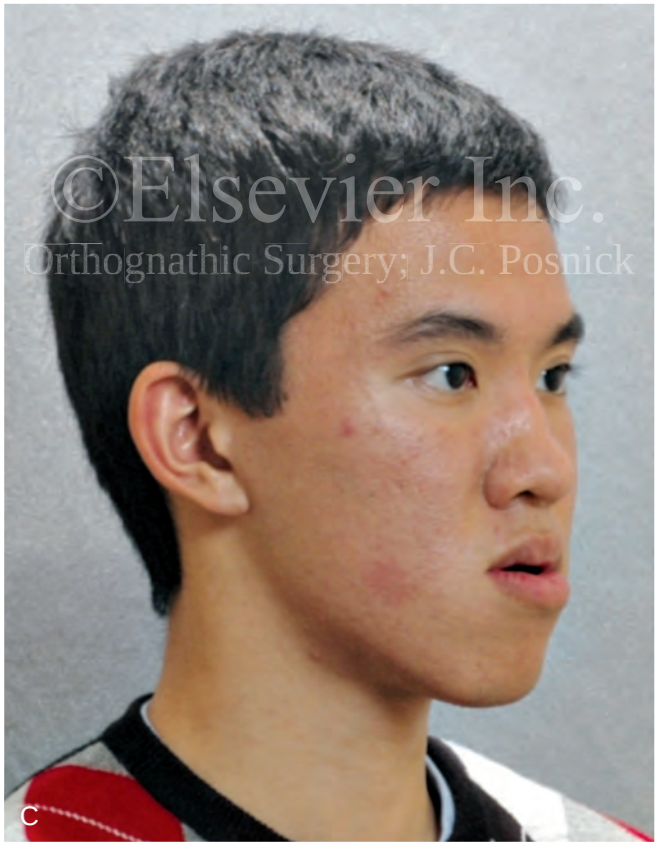
After treatment

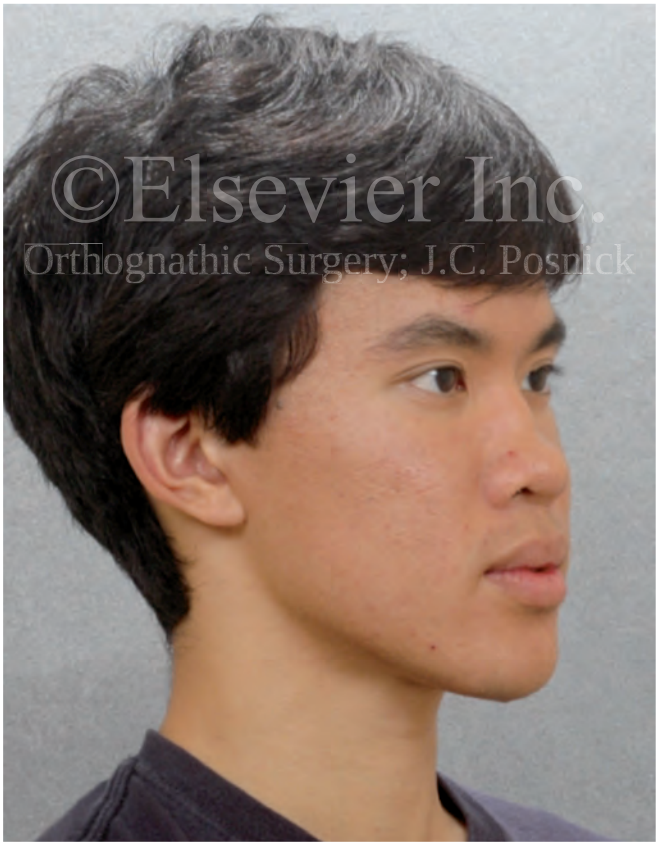
• **Figure 20-5, cont'd E,** Occlusal views before retreatment, with orthodontics in progress, and then after the completion of treatment.

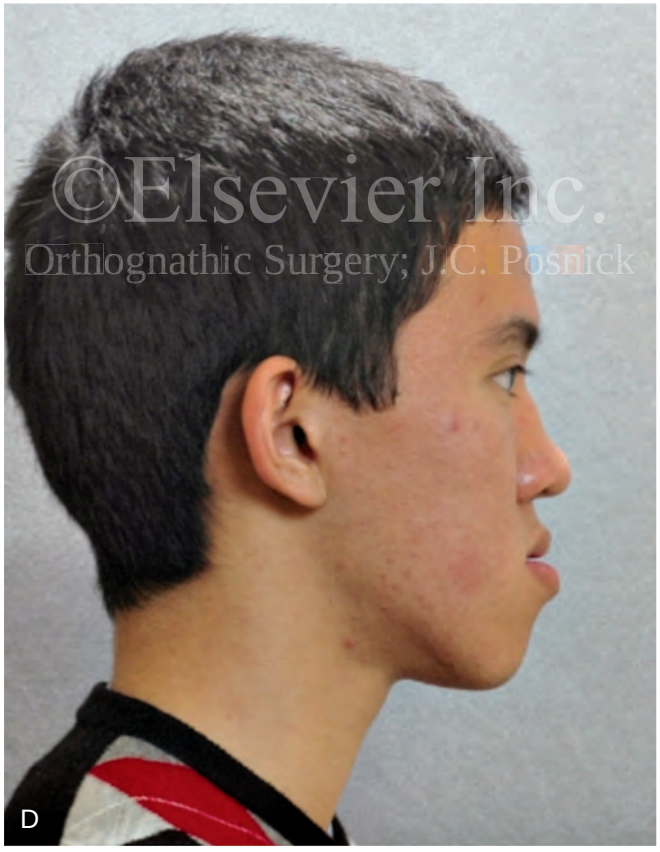


• **Figure 20-5, cont'd** **F,** Articulated dental casts that indicate analytic model planning. **G,** Lateral cephalometric radiographs before and after treatment.

• **Figure 20-6** A 17-year-old high school student was referred by his orthodontist for surgical evaluation. The patient's Class III negative overjet anterior open-bite malocclusion required redo orthodontics, including maxillary bicuspid extractions and jaw surgery. Because the patient had a lifelong history of nasal obstruction and consistent physical findings, intranasal procedures were also required. When orthodontic decompensation was complete, the patient's surgery included maxillary Le Fort I osteotomy in segments (arch expansion, clockwise rotation, horizontal advancement, and vertical lengthening); sagittal split ramus osteotomies (limited corrections); osseous genioplasty (vertical reduction and minimal horizontal advancement); and septoplasty and inferior turbinate reduction. **A,** Frontal views in repose before and after treatment. **B,** Frontal views with smile before and after treatment.







• **Figure 20-6, cont'd C,** Oblique facial views before and after treatment. **D,** Profile views before and after treatment.

Continued

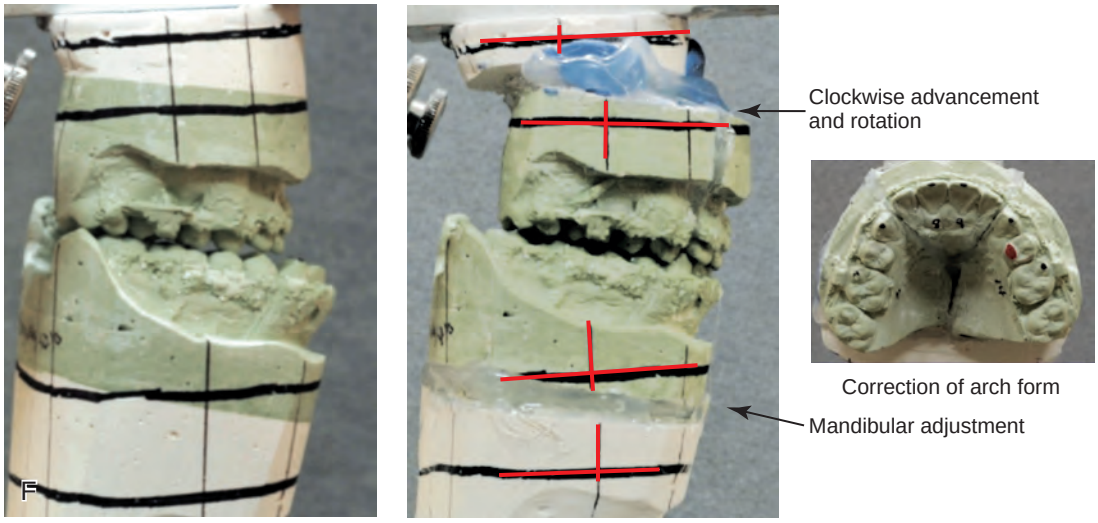
Prior to retreatment after maxillary biscupid extraction

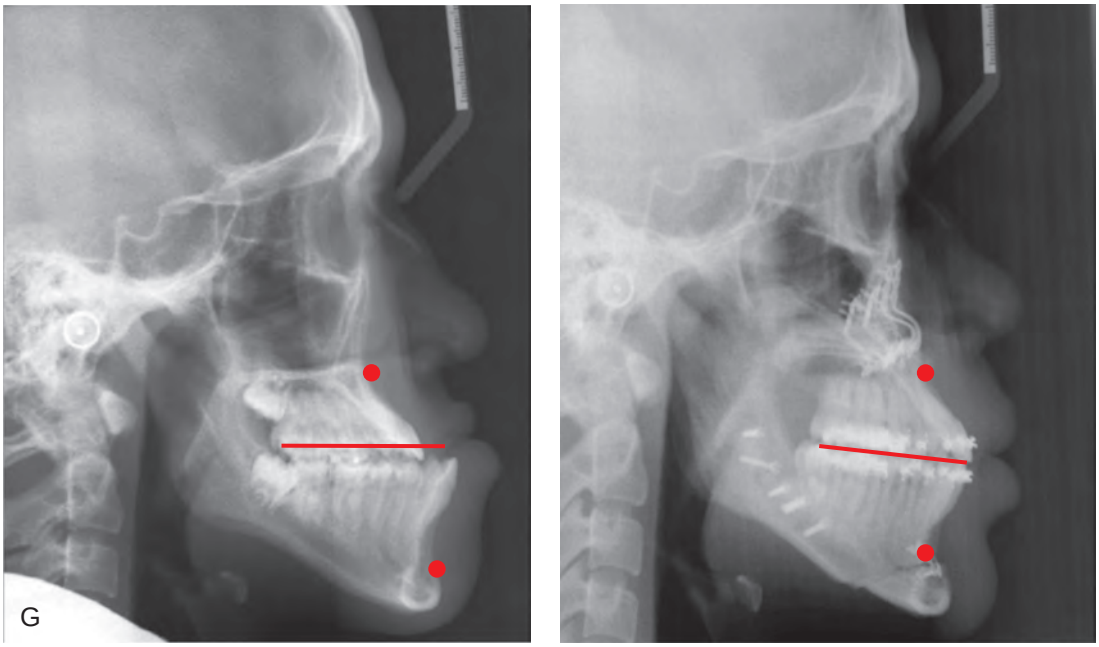
Pre surgery

E

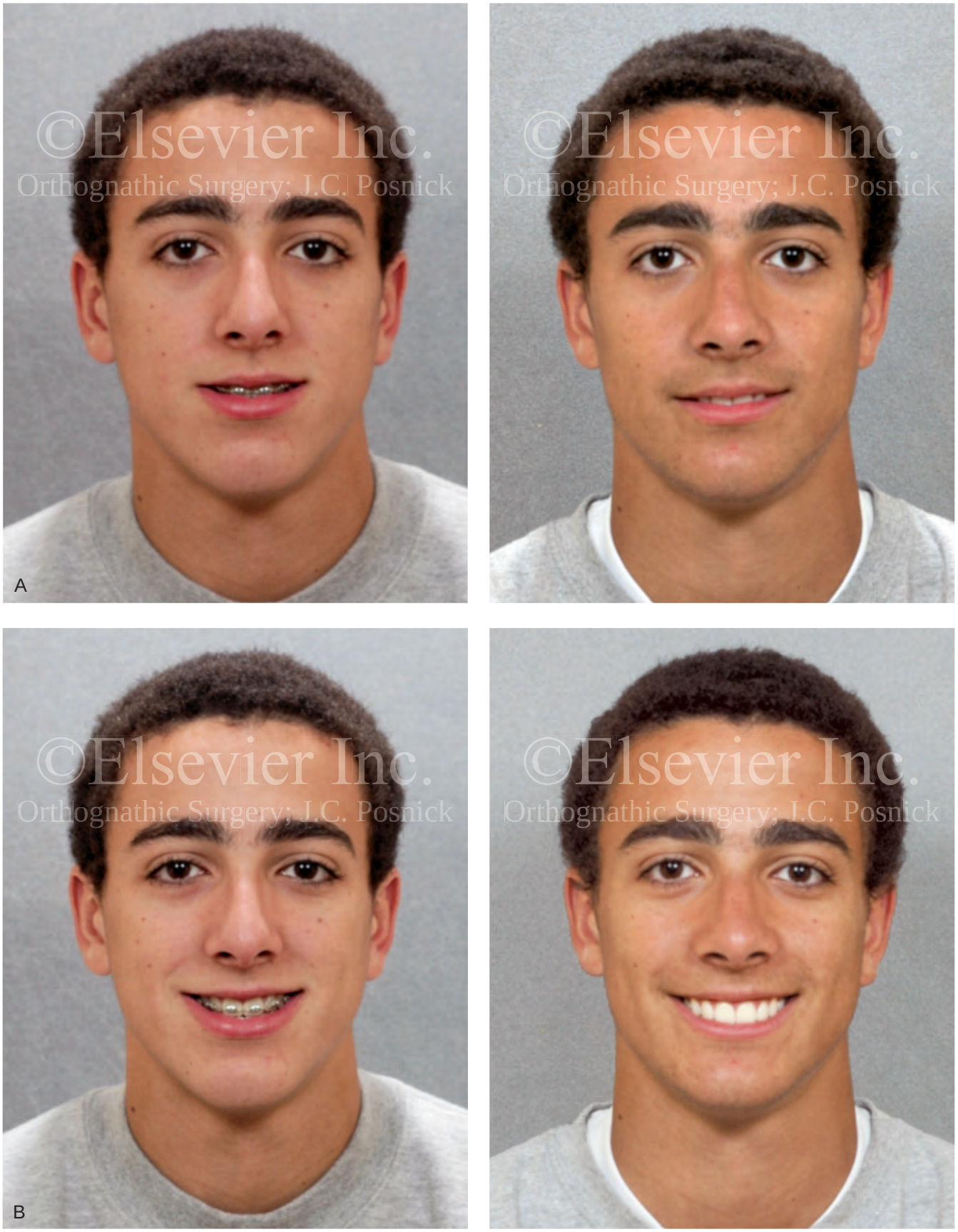
After treatment

• **Figure 20-6, cont'd E,** Occlusal views before retreatment, with orthodontics in progress, and then after the completion of treatment. The patient required gingival grafting on the labial aspect of the right maxillary canine.





• **Figure 20-6, cont'd** **F,** Articulated dental casts that indicate analytic model planning. **G,** Lateral cephalometric radiographs before and after treatment.



©Elsevier Inc.
Orthognathic Surgery; J.C. Posnick

• **Figure 20-7** A 16-year-old high school student was referred by his orthodontist for surgical evaluation. He had previously undergone orthodontics, including four bicuspid extractions. The residual Class III negative overjet malocclusion required further orthodontics in combination with jaw reconstruction. Because the patient had a lifelong history of nasal obstruction and consistent physical findings, intranasal procedures were also required. When the orthodontic decompensation was complete, the patient's surgery included maxillary Le Fort I osteotomy (horizontal advancement); sagittal split ramus osteotomies (horizontal advancement); osseous genioplasty (vertical reduction and horizontal advancement); and septoplasty and inferior turbinate reduction. **A,** Frontal views in repose before and after treatment. **B,** Frontal views with smile before and after treatment.

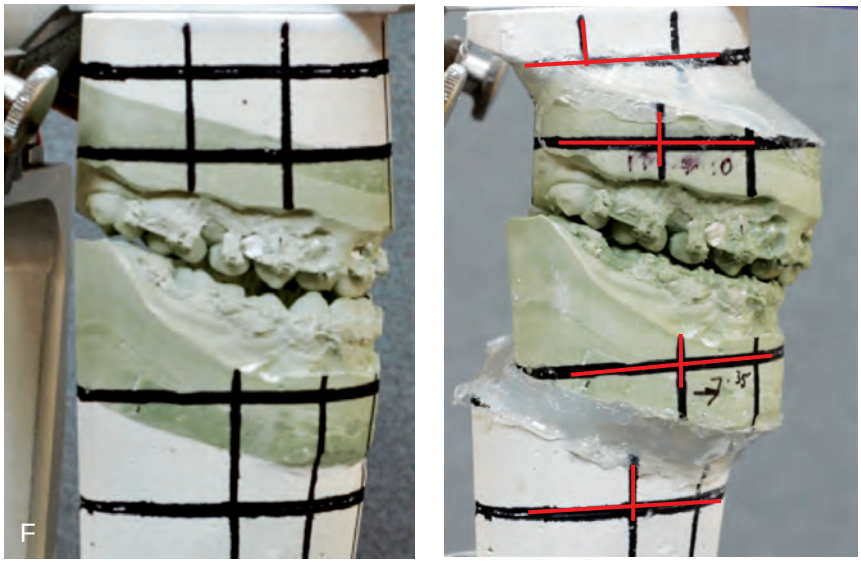
©Elsevier Inc.
Orthognathic Surgery; J.C. Posnick

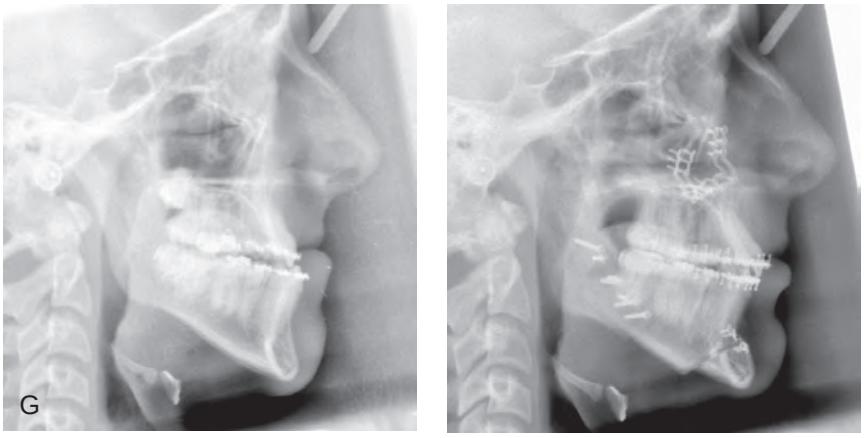
• **Figure 20-7, cont'd** **C,** Oblique facial views before and after treatment. **D,** Profile views before and after treatment.

Continued

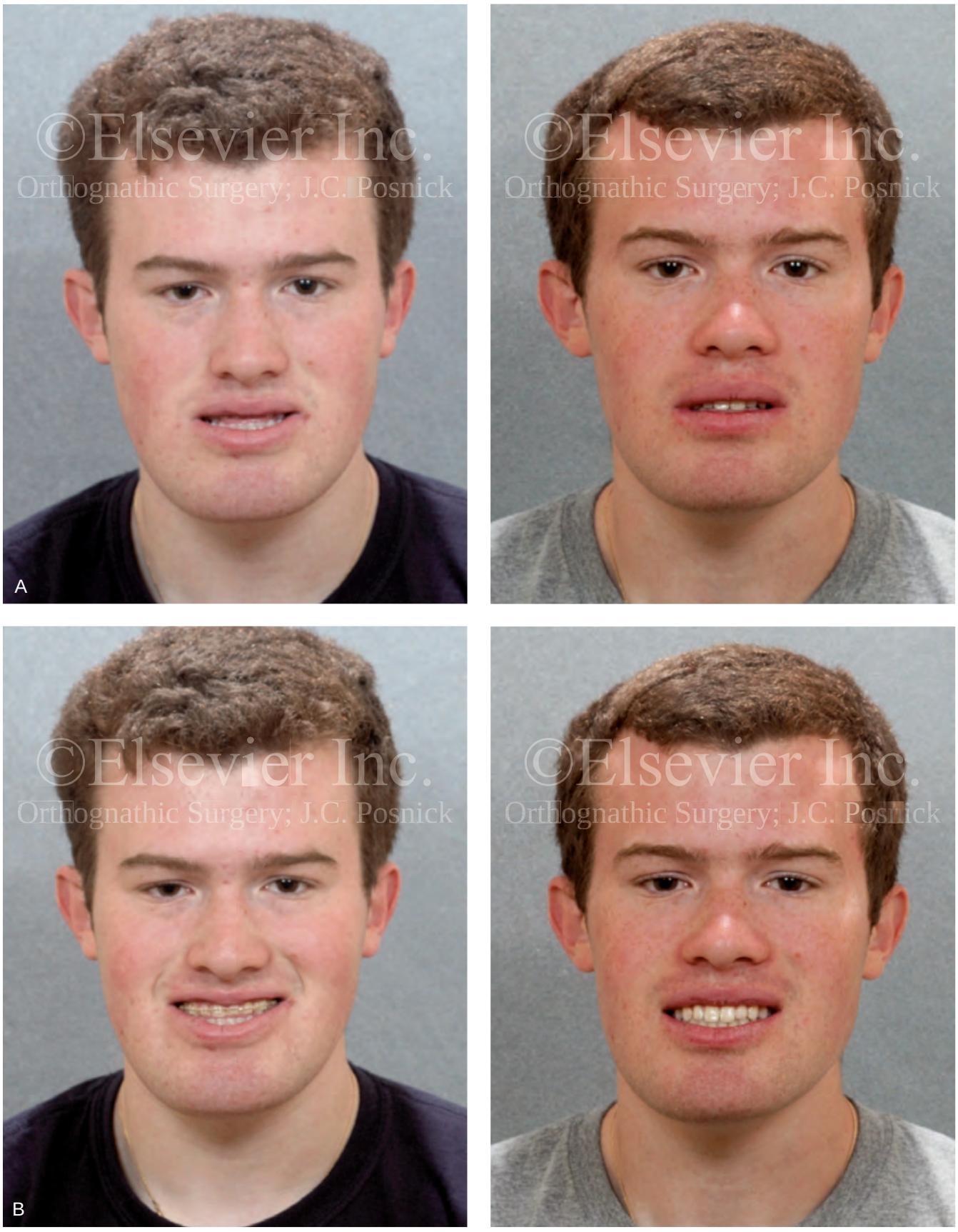
Pre surgery after four bicuspid extractions

After treatment





• **Figure 20-7, cont'd** **E,** Occlusal views with redo orthodontics in progress and after the completion of treatment. **F,** Articulated dental casts that indicate analytic model planning. **G,** Lateral cephalometric radiographs before and after treatment. Note the gingival recession on the labial aspect of the mandibular canines as a result of the orthodontic mechanics.



©Elsevier Inc.
Orthognathic Surgery; J.C. Posnick

• **Figure 20-8** A 20-year-old college student was referred by his orthodontist for surgical evaluation. His Class III negative overjet anterior open-bite malocclusion required jaw surgery. Orthodontic decompensation was carried out without maxillary extractions, which left the incisors moderately procumbent. The patient's surgery included maxillary Le Fort I osteotomy in segments (arch expansion, horizontal advancement, vertical lengthening, and clockwise rotation) and sagittal split ramus osteotomies (clockwise rotation). Note the aesthetic advantage of the maxillomandibular clockwise rotation as part of the correction. **A,** Frontal views in repose before and after treatment. **B,** Frontal views with smile before and after treatment.

Continued

• **Figure 20-8, cont'd C,** Oblique facial views before and after treatment. **D,** Profile views before and after treatment.

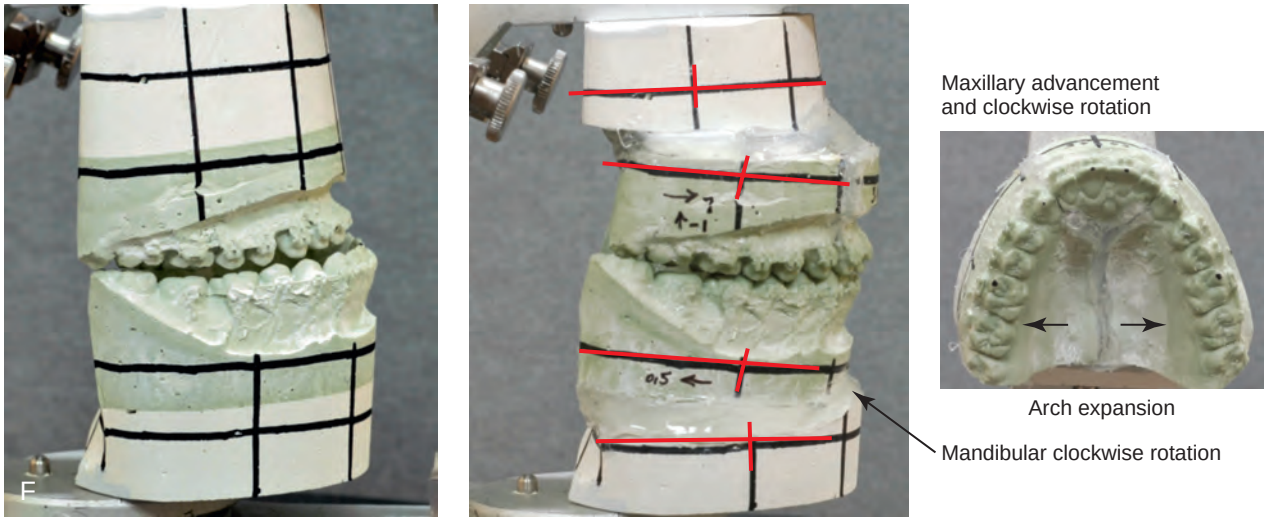
Prior to treatment

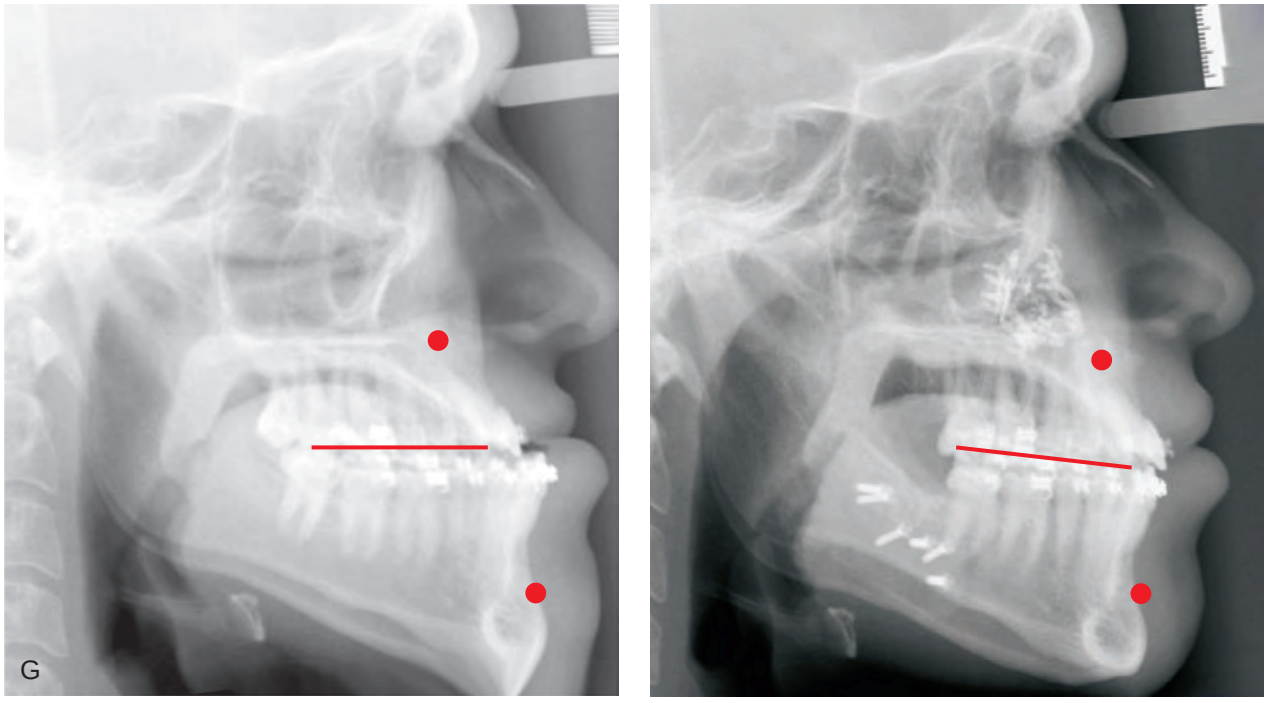
Pre surgery

After treatment

• **Figure 20-8, cont'd E,** Occlusal views before treatment, with orthodontics in progress, and then after the completion of treatment.

Continued





• **Figure 20-8, cont'd** **F,** Articulated dental casts that indicate analytic model planning. **G,** Lateral cephalometric radiographs before and after treatment.

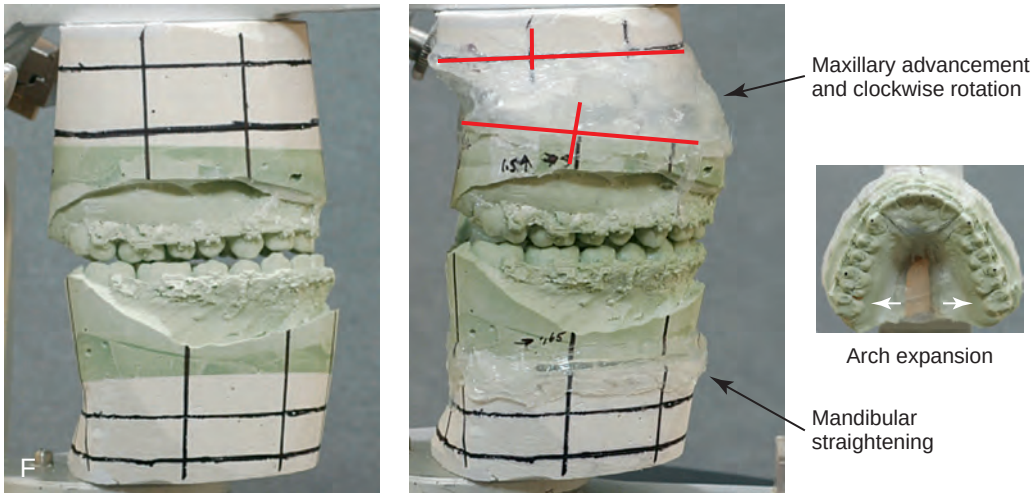
• **Figure 20-9** A 22-year-old man was referred by his orthodontist for surgical evaluation. A non-extraction orthodontic approach had previously been carried out, without success. The patient's Class III negative overjet anterior open-bite malocclusion required redo orthodontics and jaw surgery. Because he had a lifelong history of nasal obstruction and consistent physical findings, intranasal procedures were also needed. When preoperative orthodontics were complete, the patient's surgery included maxillary Le Fort I osteotomy in segments (arch expansion, horizontal advancement, vertical lengthening, and clockwise rotation); sagittal split ramus osteotomies (limited corrections); osseous genioplasty (minimal horizontal advancement); and septoplasty and inferior turbinate reduction. Note the aesthetic advantage of the maxillary clockwise rotation in the presence of baseline incisor procumbancy. **A,** Frontal views in repose before and after treatment. **B,** Frontal views with smile before and after treatment.

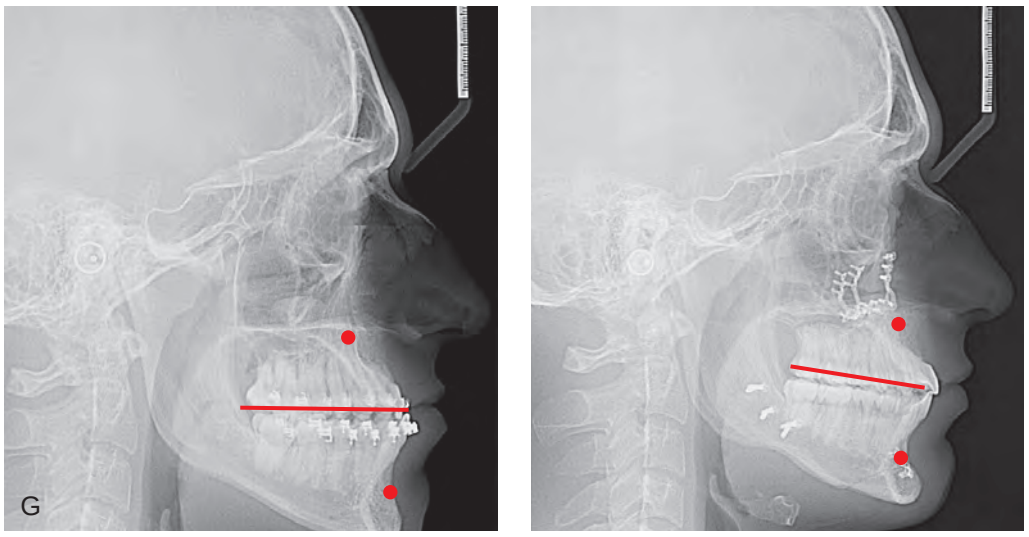
Continued

• **Figure 20-9, cont'd C,** Oblique facial views before and after treatment. **D,** Profile views before and after treatment.

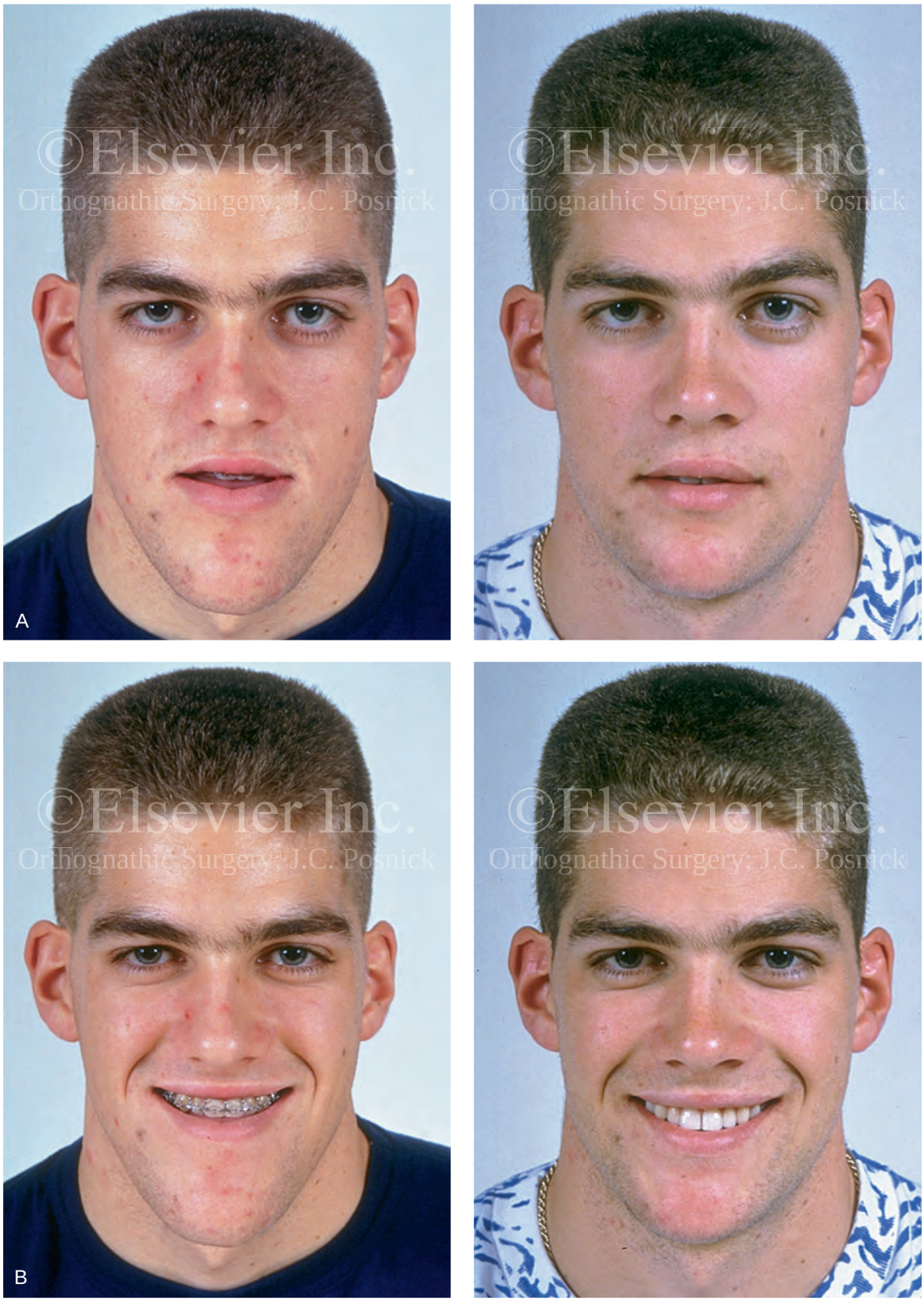
Pre surgery

After treatment



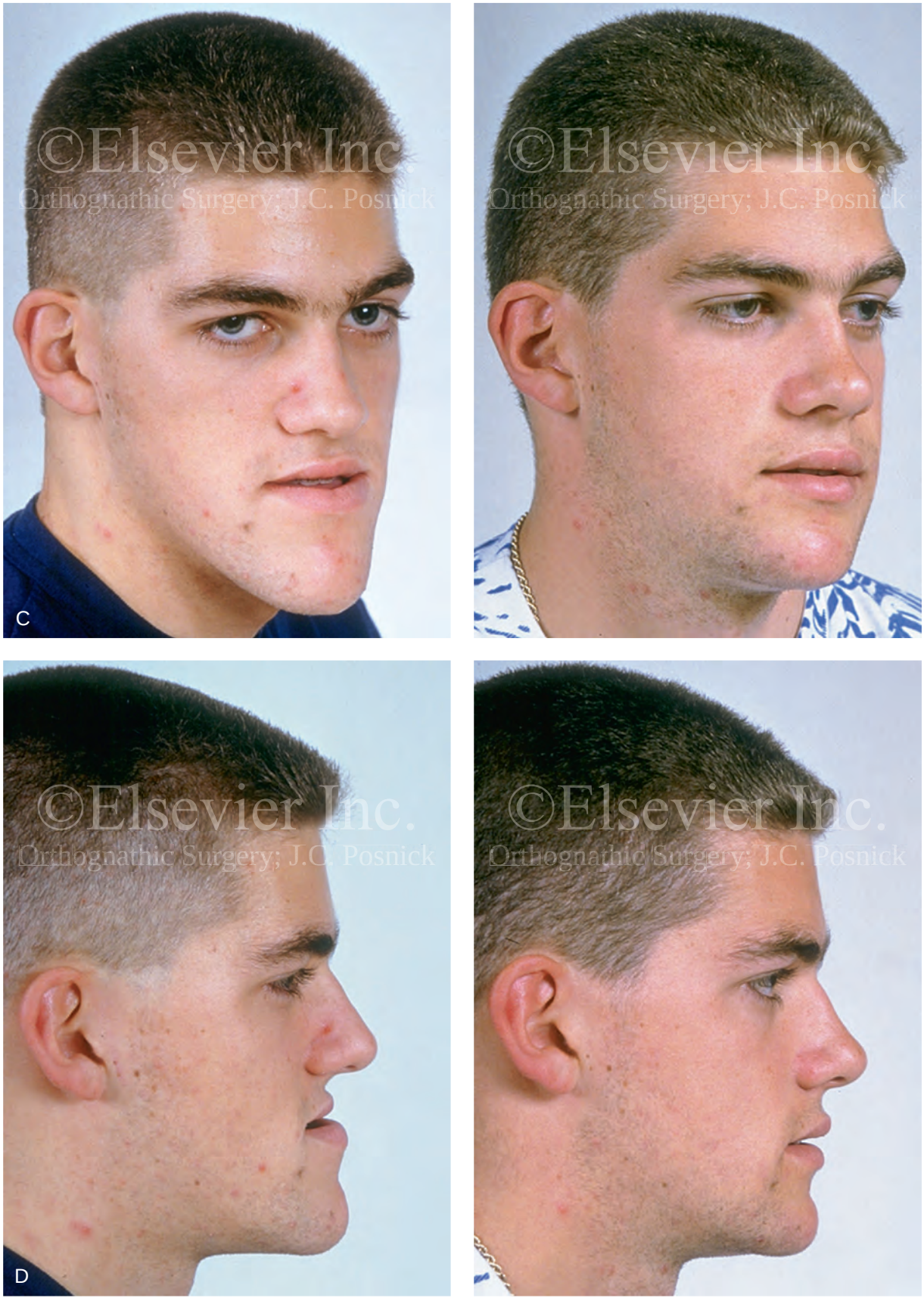


• **Figure 20-9, cont'd E,** Occlusal views with orthodontics in progress and after the completion of treatment. **F,** Articulated dental casts that indicate analytic model planning. **G,** Lateral cephalometric radiographs before and after treatment.



©Elsevier Inc.
Orthognathic Surgery; J.C. Posnick

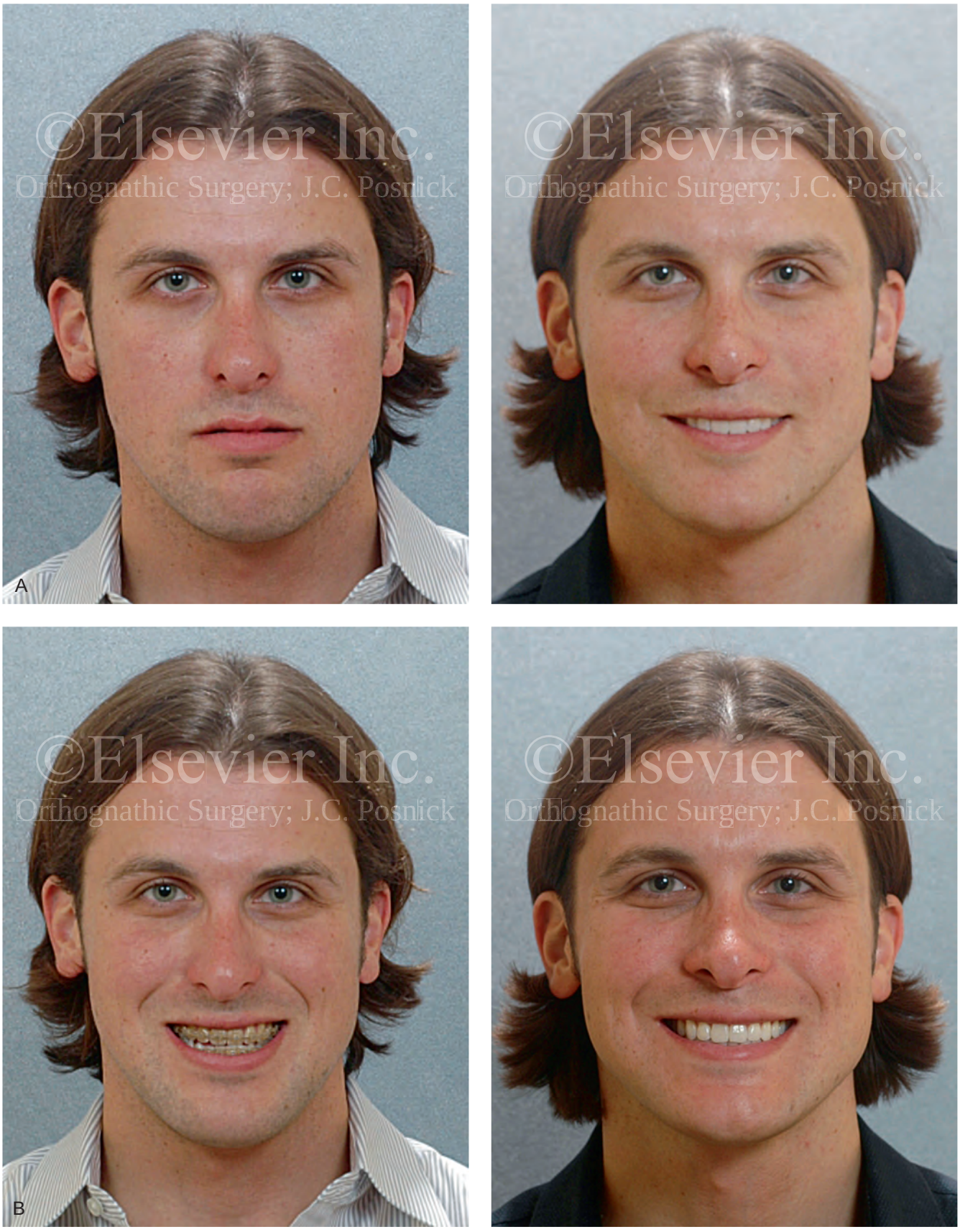
• **Figure 20-10** A 17-year-old high school student was referred by his orthodontist, who requested surgical evaluation for a Le Fort III advancement. The Class III negative overjet malocclusion with maxillary deficiency gave the impression of flat cheekbones and infraorbital rims. Evaluation confirmed that the patient's deformity was limited to the maxilla and the mandible. Because the patient had a lifelong history of nasal obstruction and consistent physical findings, intranasal procedures were also required. When preparatory orthodontics were complete, the patient's surgery included maxillary Le Fort I osteotomy (horizontal advancement, vertical lengthening, and clockwise rotation); sagittal split ramus osteotomies (setback and clockwise rotation); osseous genioplasty (vertical reduction); and septoplasty and inferior turbinate reduction. Note the aesthetic advantage of the maxillomandibular clockwise rotation in the presence of baseline incisor procumbancy. This rotation projects the pyriform rims forward and sets back the pogonion. The orthodontic over-retraction of the lower incisors resulted in labial bone loss and gingival recession. **A,** Frontal views in repose before and after treatment. **B,** Frontal views with smile before and after treatment.



©Elsevier Inc.
Orthognathic Surgery; J.C. Posnick

• **Figure 20-10, cont'd C,** Oblique facial views before and after treatment. **D,** Profile views before and after treatment.

• **Figure 20-10, cont'd** **E,** Worm's-eye views before and after reconstruction. **F,** Occlusal views with orthodontics in progress and after the completion of treatment. Note the mild gingival recession on the labial aspect of the mandibular anterior teeth as a result of orthodontic mechanics. **G,** Articulated dental casts that indicate analytic model planning.



©Elsevier Inc.
Orthognathic Surgery; J.C. Posnick

• **Figure 20-11** A 25-year-old man was referred for surgical evaluation. A non-extraction orthodontic camouflage approach had previously been carried out, without success. He previously underwent a cosmetic rhinoplasty in an attempt to reduce the size of the nose. The Class III negative overjet malocclusion required redo orthodontics and jaw surgery. Because the patient had a lifelong history of nasal obstruction and consistent physical findings, intranasal procedures were also required. When the presurgical orthodontics were complete, the patient's surgery included maxillary Le Fort I osteotomy (horizontal advancement, vertical lengthening, and clockwise rotation); sagittal split ramus osteotomies (clockwise rotation); osseous genioplasty (vertical reduction and minimal horizontal advancement); and septoplasty and inferior turbinate reduction. Note the aesthetic advantage of the maxillomandibular clockwise rotation in the presence of baseline incisor procumbancy. **A,** Frontal views in repose before and after treatment. **B,** Frontal views with smile before and after treatment.

Continued

• **Figure 20-11, cont'd C,** Oblique facial views before and after treatment. **D,** Profile views before and after treatment.

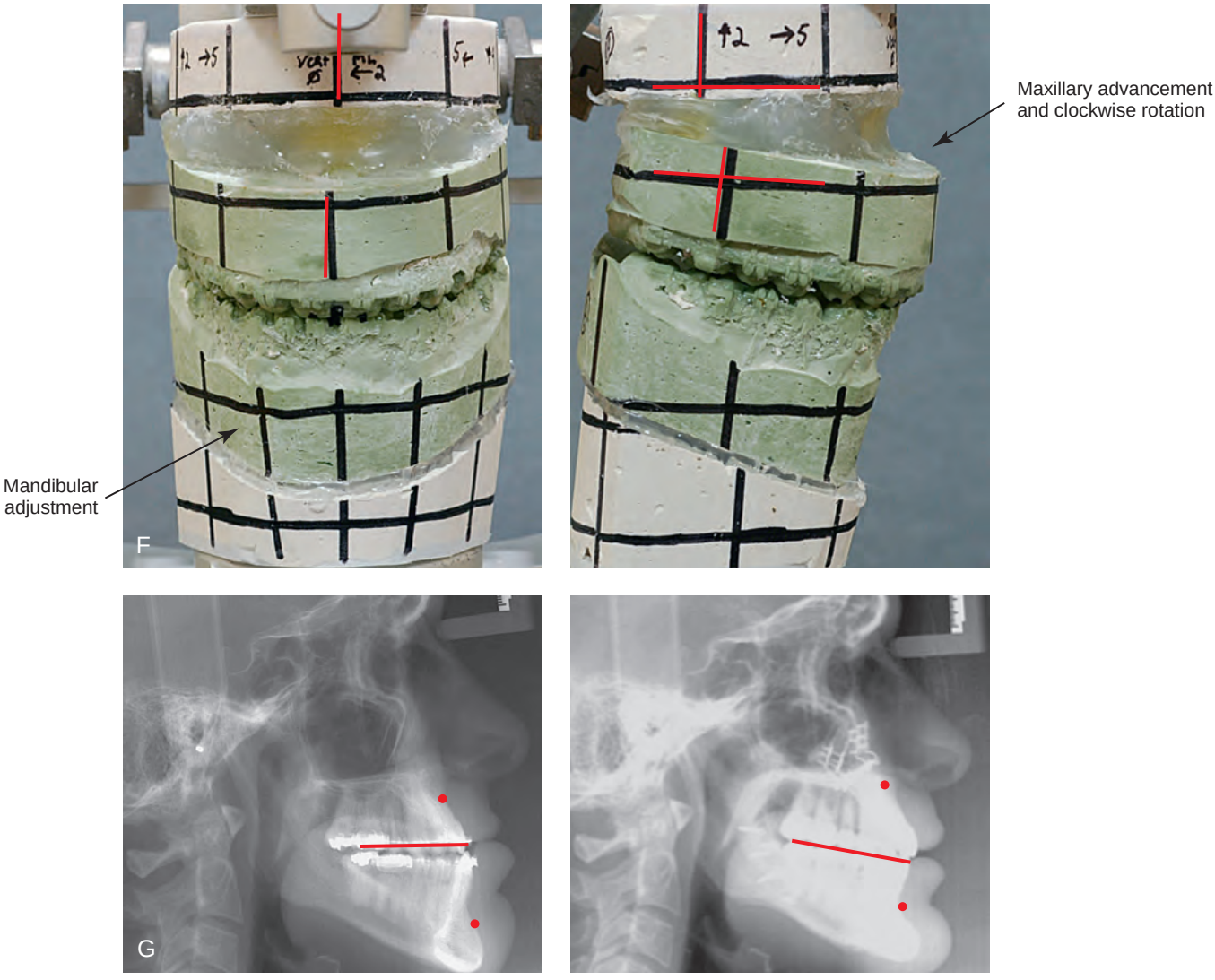
Prior to retreatment

Pre surgery

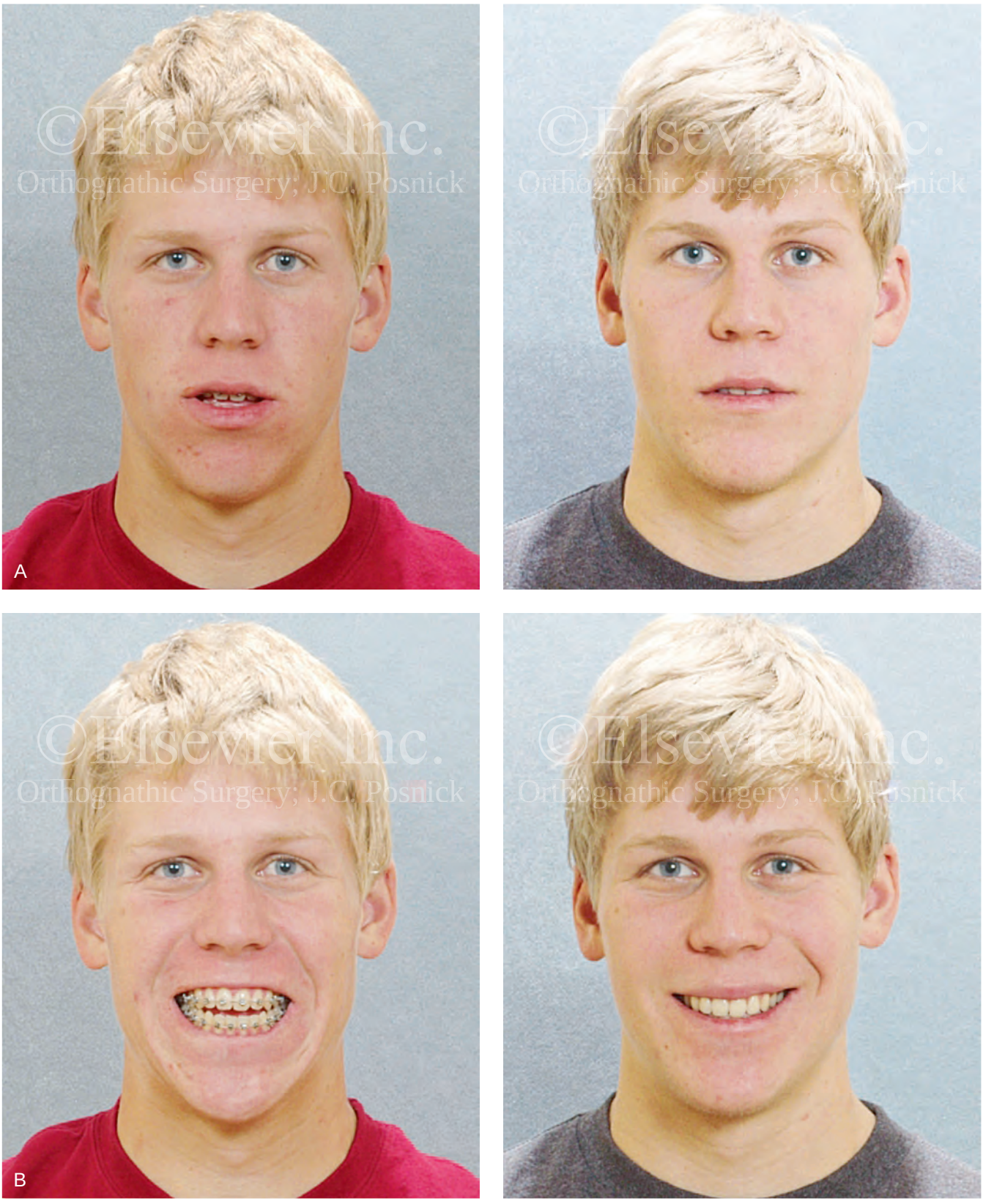
After treatment

• **Figure 20-11, cont'd E,** Occlusal views before retreatment, with orthodontics in progress, and after the completion of treatment. Note the gingival recession on the labial aspect of the mandibular and maxillary anterior dentition as a result of the dental roots being crowded into limited available alveolar bone.

Continued



• **Figure 20-11, cont'd** **F,** Articulated dental casts that indicate analytic model planning. **G,** Lateral cephalometric radiographs before and after treatment.



• **Figure 20-12** An 18-year-old high school student was referred by his orthodontist for surgical evaluation. A non-extraction orthodontic approach had previously been carried out, without success. The Class III negative overjet anterior open-bite malocclusion required redo orthodontics and jaw surgery. Because the patient had a lifelong history of nasal obstruction and consistent physical findings, intranasal procedures were also beneficial. When orthodontic decompensation was complete, the patient's surgery included maxillary Le Fort I osteotomy in segments (arch expansion, correction of curve of Spee, horizontal advancement, vertical lengthening, and clockwise rotation); sagittal split ramus osteotomies (counterclockwise rotation); osseous genioplasty (vertical reduction and horizontal advancement); and septoplasty and inferior turbinate reduction. Note the aesthetic advantage of the maxillary clockwise rotation and the mandibular counterclockwise rotation. **A,** Frontal views in repose before and after treatment. **B,** Frontal views with smile before and after treatment.

Continued

©Elsevier Inc. Orthognathic Surgery; J.C. Posnick

• **Figure 20-12, cont'd** **C,** Oblique facial views before and after treatment. **D,** Profile views before and after treatment.

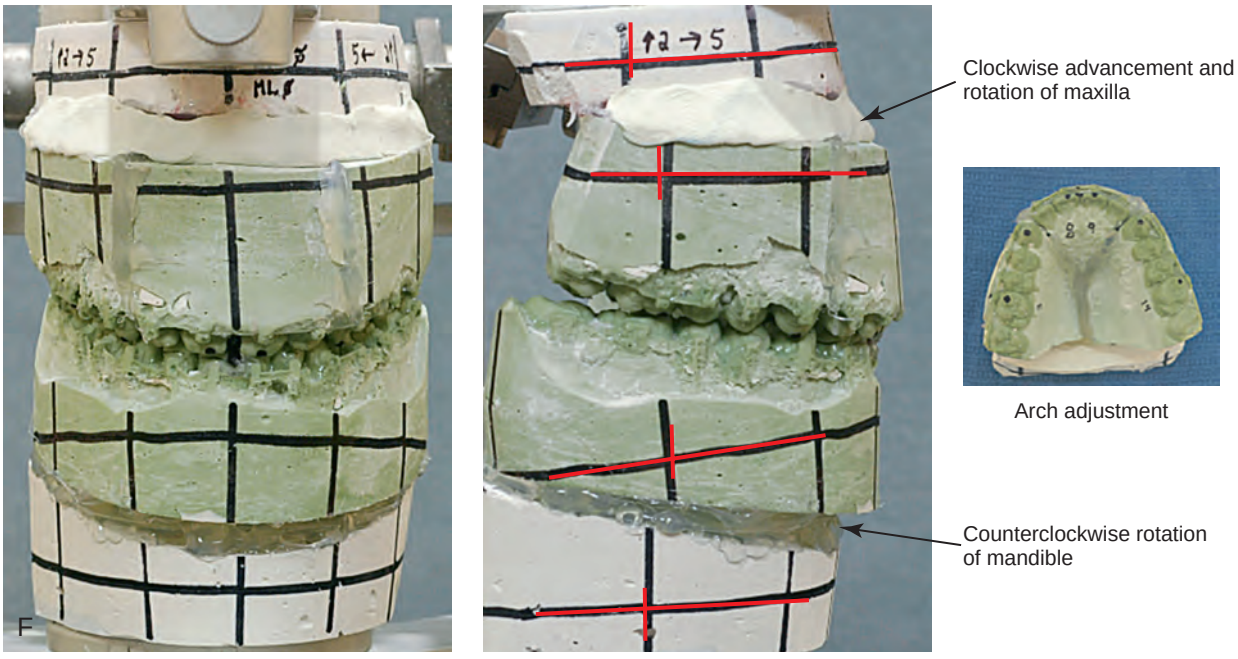
Prior to retreatment

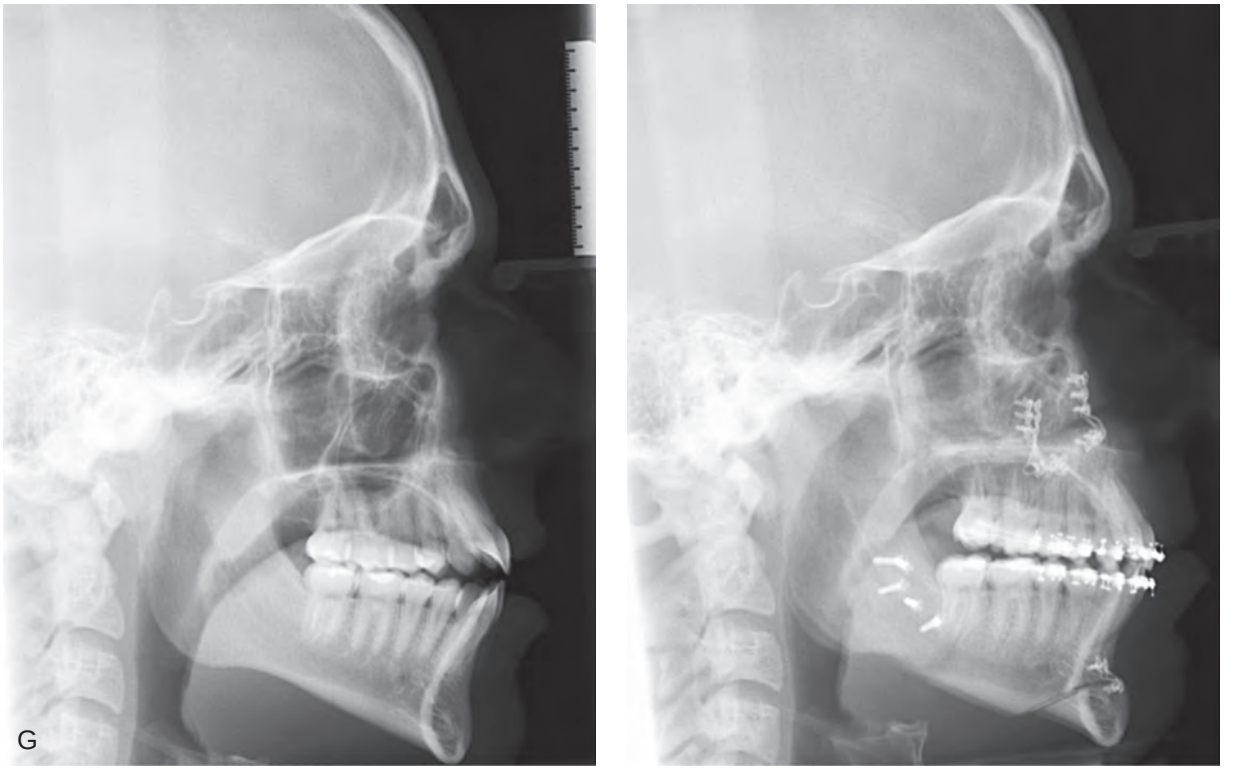
Pre surgery

After treatment

• **Figure 20-12, cont'd E,** Occlusal views before retreatment, with orthodontics in progress, and after the completion of treatment.

Continued





• **Figure 20-12, cont'd F,** Articulated dental casts that indicate analytic model planning. **G,** Lateral cephalometric radiographs before and after treatment.

©Elsevier Inc.
Orthognathic Surgery; J.C. Posnick

• **Figure 20-13** A 20-year-old college student was referred for surgical evaluation. The Class III negative overjet deep-bite malocclusion required orthodontics and jaw surgery. Because the patient had a lifelong history of nasal obstruction and consistent physical findings, intranasal procedures were also beneficial. Orthodontics was carried out without extraction. A degree of maxillary incisor procumbency remained. The patient's surgery included maxillary Le Fort I osteotomy (horizontal advancement, vertical lengthening, and clockwise rotation); sagittal split ramus osteotomies (clockwise rotation); osseous genioplasty (vertical reduction and minimal horizontal advancement); and septoplasty and inferior turbinate reduction. Note the aesthetic advantage of the maxillomandibular clockwise rotation in the presence of baseline incisor procumbancy. **A,** Frontal views in repose before and after treatment. **B,** Frontal views with smile before and after treatment.

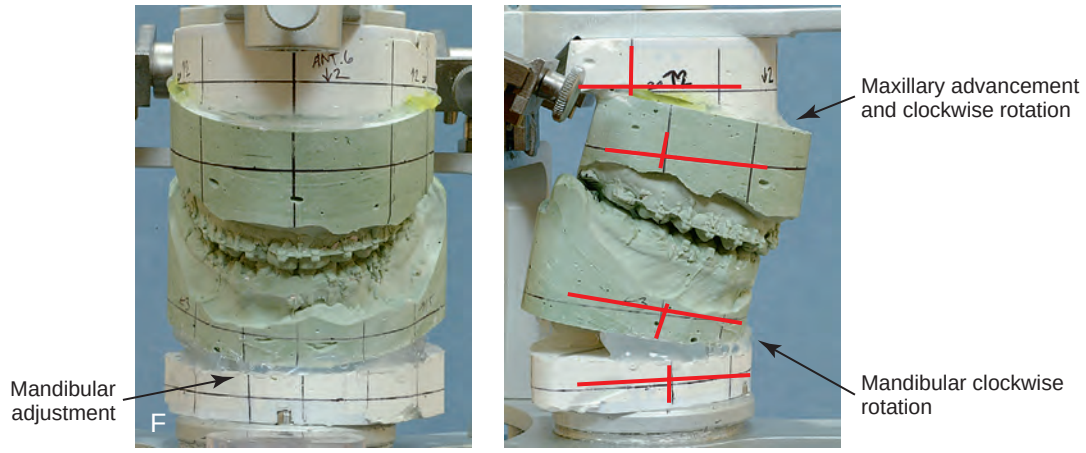
Continued

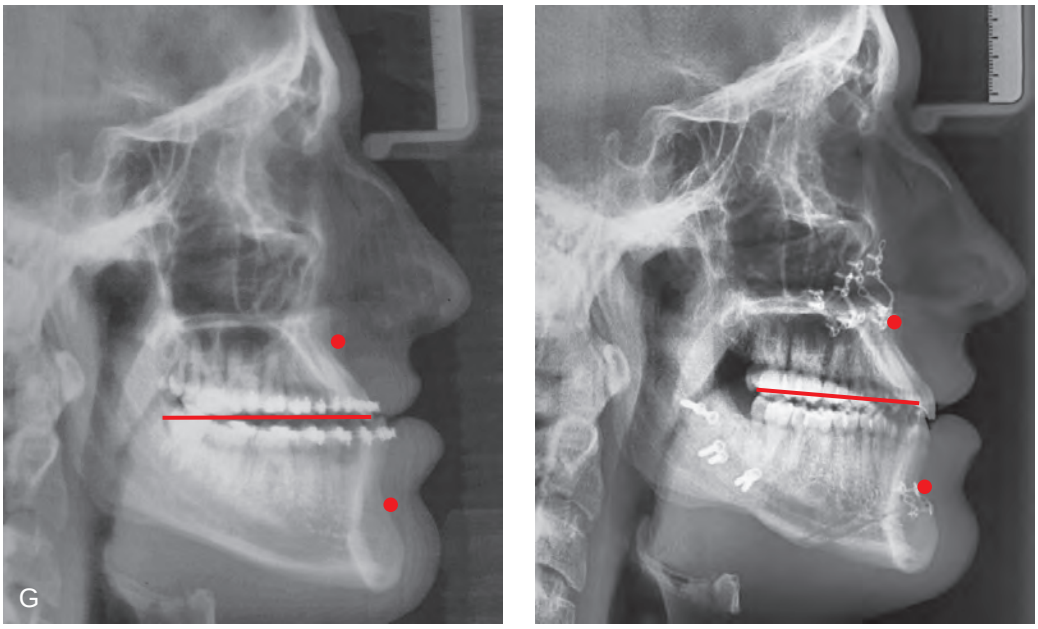
©Elsevier Inc.
Orthognathic Surgery; J.C. Posnick

• **Figure 20-13, cont'd C,** Oblique facial views before and after treatment. **D,** Profile views before and after treatment.

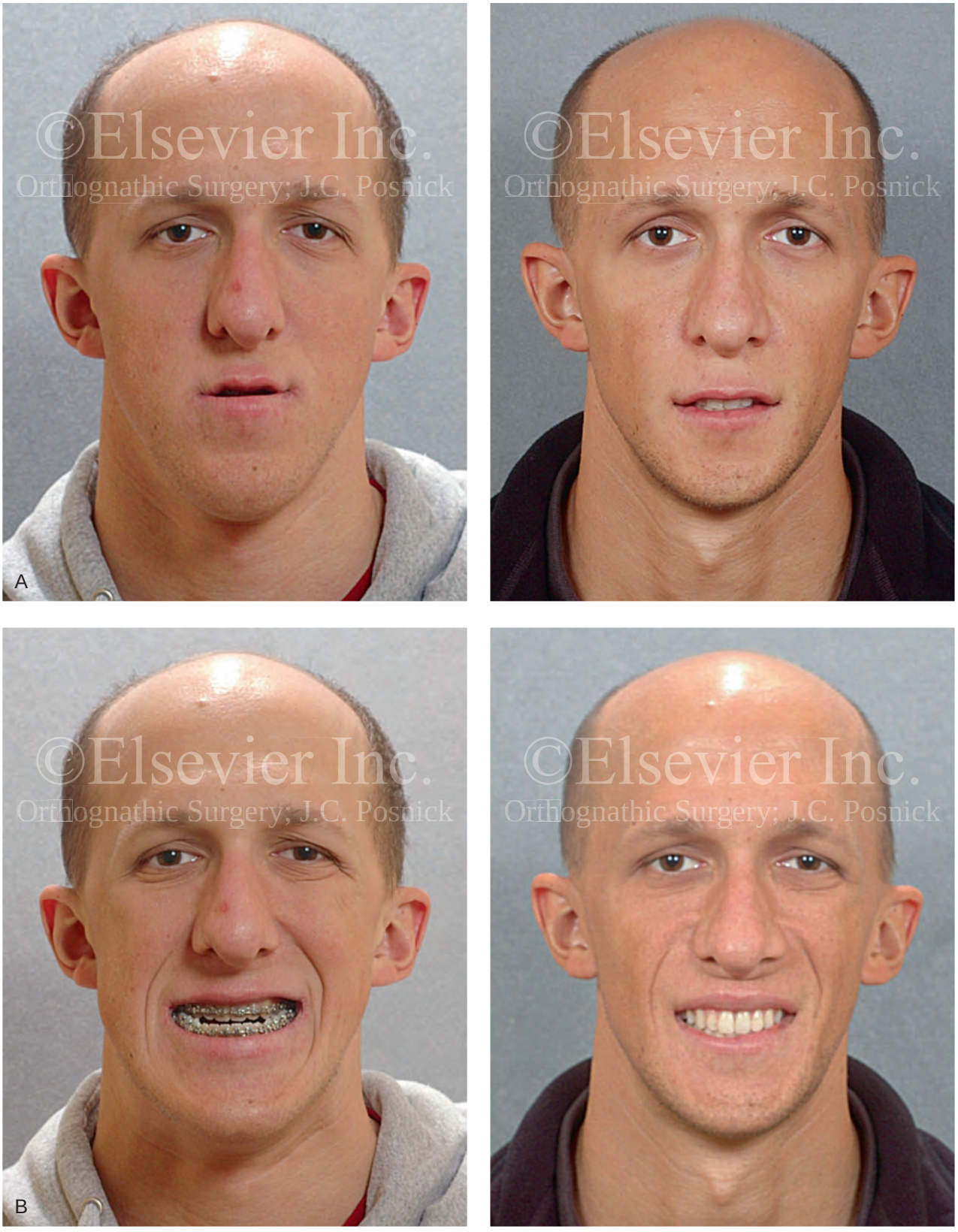
Prior to treatment

After treatment





• **Figure 20-13, cont'd E,** Occlusal views before retreatment and after the completion of treatment. **F,** Articulated dental casts that indicate analytic model planning. **G,** Lateral cephalometric radiographs before and after treatment.



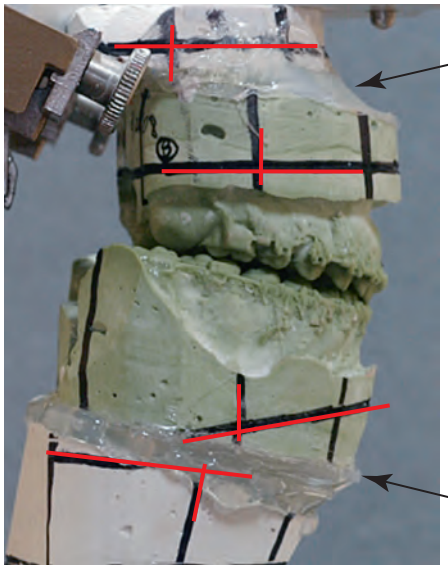
• **Figure 20-14** A 19-year-old college freshman was referred for evaluation. He had a maxillary deficiency with a relative mandibular excess growth pattern and a Class III negative overjet anterior open-bite malocclusion. During the mixed dentition, the patient was treated with growth-modification techniques and rapid palatal expansion. When he was 18 years old, orthodontic mechanics were reinitiated; in preparation for jaw reconstruction. The maxillary and mandibular second molars were poorly developed and required removal. The patient had a lifelong history of nasal obstruction; physical examination confirmed a deviated septum, enlarged inferior turbinates, and a constricted nasal aperture. When orthodontic decompensation was complete, the patient's surgery included maxillary Le Fort I osteotomy (+12 mm horizontal advancement, +2 mm vertical lengthening, 2.5 mm midline correction) and interpositional iliac bone graft; sagittal split ramus osteotomies (horizontal advancement and counterclockwise rotation); osseous genioplasty (vertical shortening); and septoplasty, inferior turbinate reduction, and pyriform rim recontouring. **A,** Frontal views in repose before and after treatment. **B,** Frontal views with smile before and after treatment.

• **Figure 20-14, cont'd C,** Oblique facial views before and after treatment. **D,** Profile views before and after treatment.

Continued

Pre surgery

After treatment



• **Figure 20-14, cont'd E,** Occlusal views with orthodontics in progress and then 4 years after the completion of treatment. **F,** Articulated dental casts that indicate analytic model planning. **G,** Lateral cephalometric radiographs before and after treatment.

• **Figure 20-15** A 27-year-old woman was referred by her orthodontist for surgical evaluation. A non-extraction orthodontic approach had been carried out during her high school years, without success. Her Class III negative overjet anterior open-bite malocclusion required redo orthodontics and jaw surgery. Because the patient had a lifelong history of nasal obstruction and consistent physical findings, intranasal procedures were also beneficial. After the orthodontic decompensation was complete, the patient's surgery included maxillary Le Fort I osteotomy in segments (arch expansion, horizontal advancement, vertical lengthening, and clockwise rotation); sagittal split ramus osteotomies (counterclockwise rotation); osseous genioplasty (vertical reduction); and septoplasty and inferior turbinate reduction. Note the aesthetic advantage of the mandibular counter-clockwise rotation. **A,** Frontal views in repose before and after treatment. **B,** Frontal views with smile before and after treatment. *Continued*

©Elsevier Inc.
Orthognathic Surgery; J.C. Posnick

• **Figure 20-15, cont'd C,** Oblique facial views before and after treatment. **D,** Profile views before and after treatment.

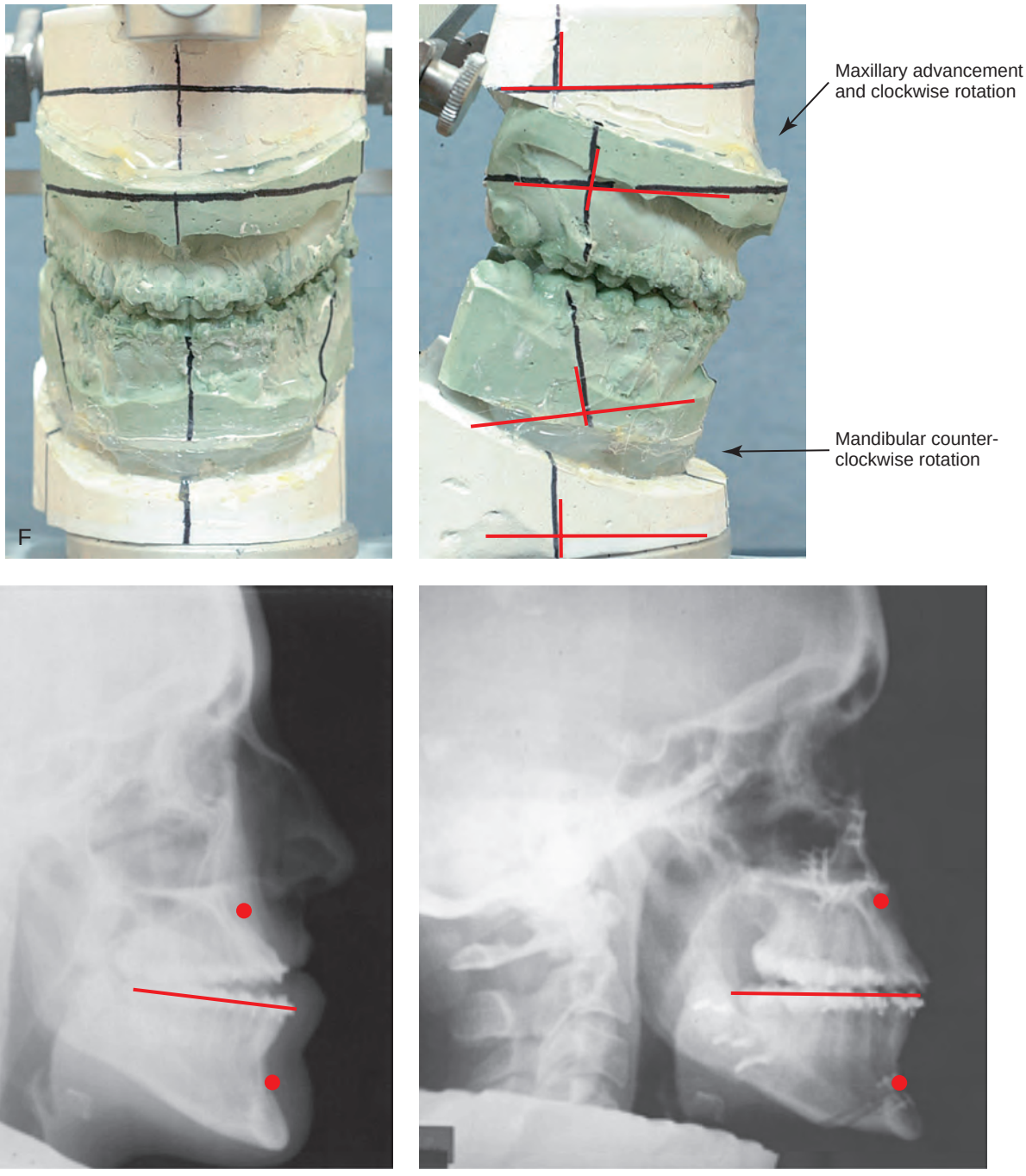
Prior to retreatment

Pre surgery

After treatment

• **Figure 20-15, cont'd E,** Occlusal views before retreatment, with orthodontics in progress, and after the completion of treatment.

Continued



• **Figure 20-15, cont'd** **F,** Articulated dental casts that indicate analytic model planning. **G,** Lateral cephalometric radiographs before and after treatment.

• **Figure 20-16** A 16-year-old high school student was referred by her orthodontist for surgical evaluation. A non-extraction orthodontic approach had previously been carried out, without success. The Class III negative overjet anterior open-bite malocclusion required redo orthodontics and jaw surgery. Because the patient had a lifelong history of nasal obstruction and consistent physical findings, intranasal procedures were also beneficial. Presurgical orthodontics were carried out without extractions, and the maxillary anterior procumbancy remained. The patient's surgery included maxillary Le Fort I osteotomy in segments (arch expansion, horizontal advancement, vertical lengthening, and clockwise rotation); sagittal split ramus osteotomies (minimal clockwise rotation); osseous genioplasty (vertical reduction and minimal horizontal advancement); and septoplasty and inferior turbinate reduction. Note the aesthetic advantage of the maxillary plane clockwise rotation in the presence of baseline incisor procumbency. **A,** Frontal views in repose before and after treatment. **B,** Frontal views with smile before and after treatment.

Continued

• **Figure 20-16, cont'd C,** Oblique facial views before and after treatment. **D,** Profile views before and after treatment.

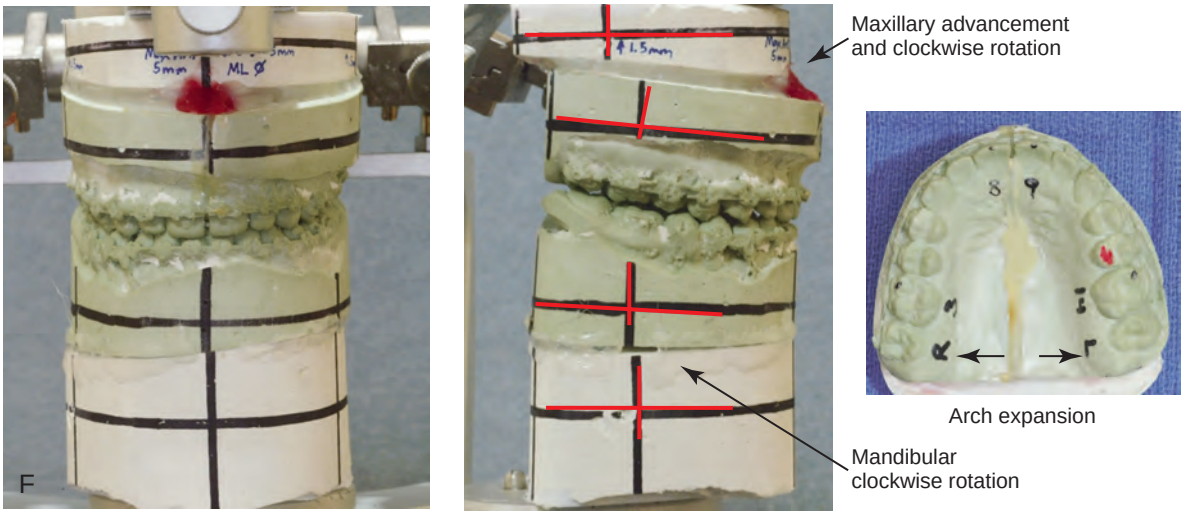
Prior to retreatment

Pre surgery

After treatment

• **Figure 20-16, cont'd E,** Occlusal views before retreatment, with orthodontics in progress, and after the completion of treatment.

Continued



• **Figure 20-16, cont'd F,** Articulated dental casts that indicate analytic model planning. **G,** Lateral cephalometric radiographs before and after treatment.

• **Figure 20-17** A 15-year-old high school student was referred by her orthodontist for surgical evaluation. A non-extraction orthodontic approach had previously been carried out, without success. The Class III negative overjet malocclusion required redo orthodontics and jaw surgery. Because the patient had a lifelong history of nasal obstruction and consistent physical findings, intranasal procedures were also beneficial. Presurgical orthodontics were carried out without extractions, and the maxillary incisor procumbency remained. The patient's surgery included maxillary Le Fort I osteotomy (horizontal advancement, vertical lengthening, and clockwise rotation); sagittal split ramus osteotomies (clockwise rotation); osseous genioplasty (vertical reduction and minimal horizontal advancement); and septoplasty and inferior turbinate reduction. Note the aesthetic advantage of the maxillomandibular clockwise rotation in the presence of baseline incisor procumbency. **A,** Frontal views in repose before and after treatment. **B,** Frontal views with smile before and after treatment.

Continued

©Elsevier Inc.
Orthognathic Surgery; J.C. Posnick

• **Figure 20-17, cont'd C,** Oblique facial views before and after treatment. **D,** Profile views before and after treatment.

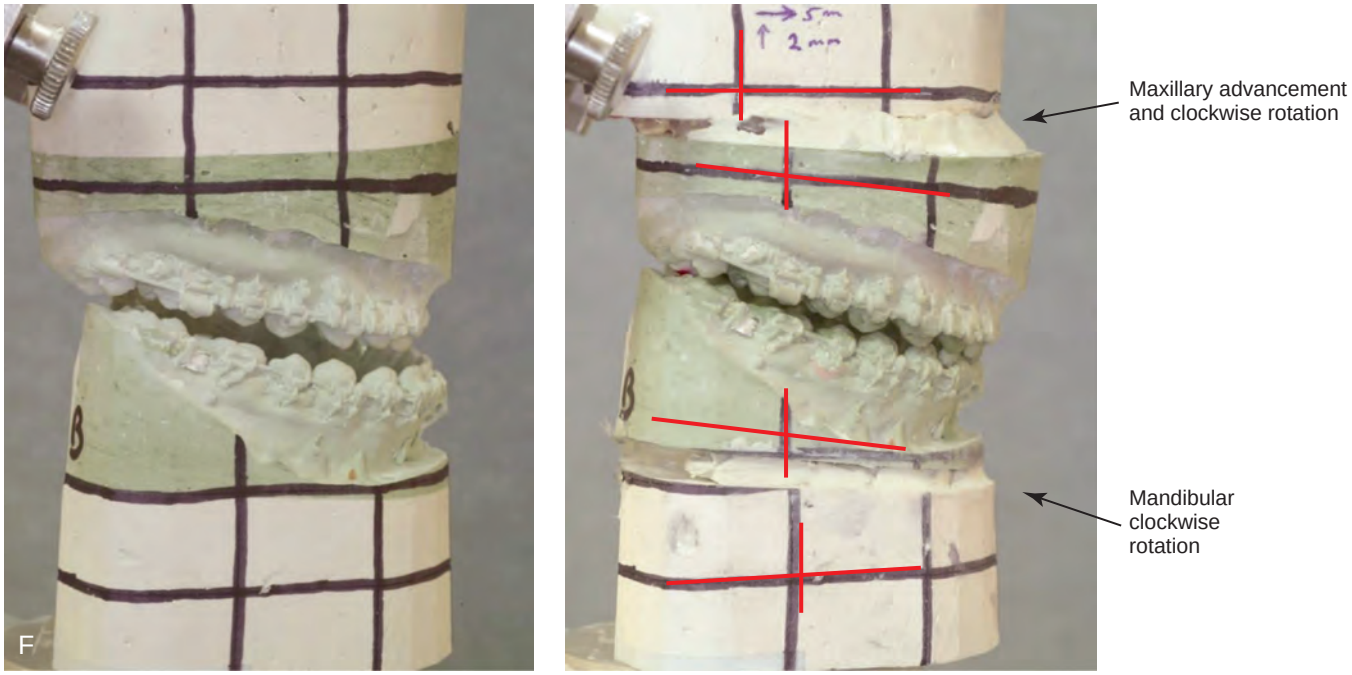
Prior to retreatment

Pre surgery

After treatment

• **Figure 20-17, cont'd E,** Occlusal views before retreatment, with orthodontics in progress, and after the completion of treatment.

Continued



• **Figure 20-17, cont'd F,** Articulated dental casts that indicate analytic model planning. **G,** Lateral cephalometric radiographs before and after treatment.

©Elsevier Inc.
Orthognathic Surgery; J.C. Posnick

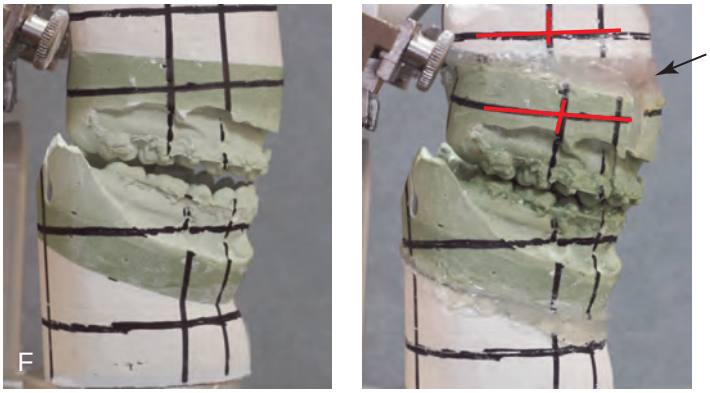
• **Figure 20-18** A 21-year-old woman was referred for surgical evaluation. An orthodontic approach that involved four bicuspid extractions to neutralize the occlusion had previously been carried out, without success. The Class III negative overjet malocclusion required redo orthodontics and jaw surgery. Because the patient had a lifelong history of nasal obstruction and consistent physical findings, intranasal procedures were also beneficial. When the orthodontic decompensation was complete, the patient's surgery included maxillary Le Fort I osteotomy in segments (arch expansion, horizontal advancement, vertical lengthening, and clockwise rotation); sagittal split ramus osteotomies (minimal corrections); osseous genioplasty (minimal horizontal advancement); and septoplasty and inferior turbinate reduction. **A,** Frontal views in repose before and after treatment. **B,** Frontal views with smile before and after treatment.

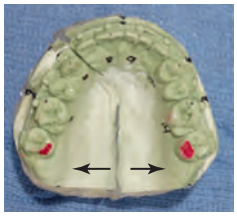
Continued

• **Figure 20-18, cont'd C,** Oblique facial views before and after treatment. **D,** Profile views before and after treatment.

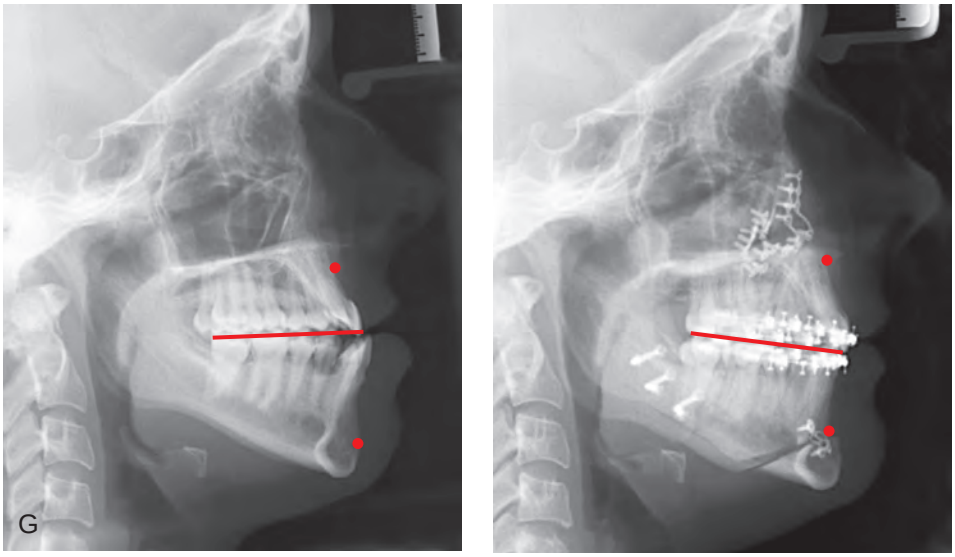
Prior to surgery after four bicuspid extractions

After treatment





Arch expansion



• **Figure 20-18, cont'd** **E,** Occlusal views with orthodontics in progress and after the completion of treatment. **F,** Articulated dental casts that indicate analytic model planning. **G,** Lateral cephalometric radiographs before and after treatment.

• **Figure 20-19** A 16-year-old high school student was referred by her orthodontist for surgical evaluation. Her Class III negative overjet malocclusion required orthodontics and jaw surgery. Because the patient had a lifelong history of nasal obstruction and consistent physical findings, intranasal procedures were also beneficial. Presurgical orthodontics were completed without extractions, and the maxillary dental root crowding in the alveolar bone and the incisor procumbency remained. The patient's surgery included maxillary Le Fort I osteotomy (horizontal advancement, vertical lengthening, and clockwise rotation); sagittal split ramus osteotomies (clockwise rotation); osseous genioplasty (minimal horizontal advancement); and septoplasty and inferior turbinate reduction. Note the aesthetic advantage of the maxillomandibular clockwise rotation. **A,** Frontal views in repose before and after treatment. **B,** Frontal views with smile before and after treatment.

• **Figure 20-19, cont'd C,** Oblique facial views before and after treatment. **D,** Profile views before and after treatment.

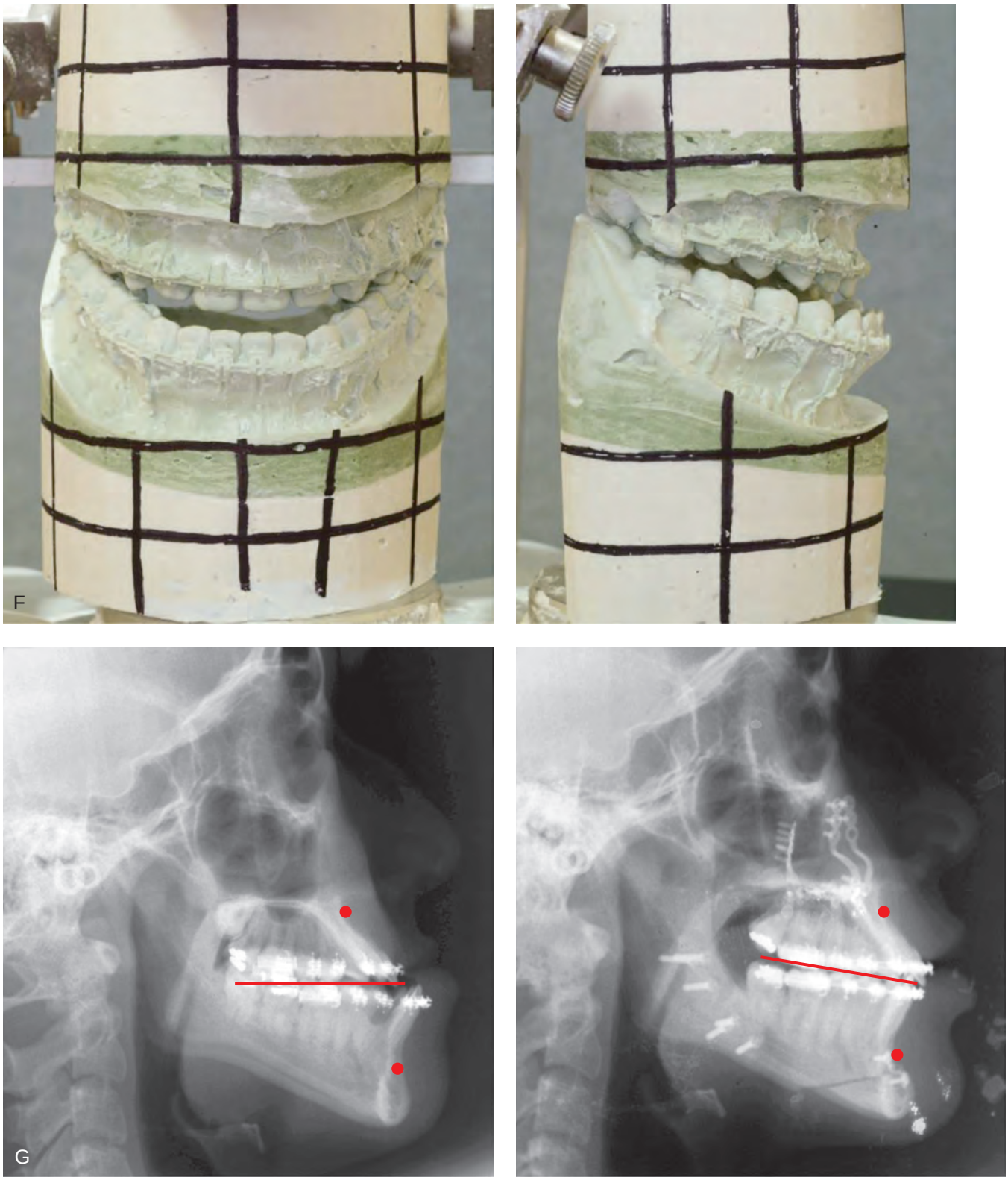
Continued

Prior to treatment

Pre surgery

After treatment

• **Figure 20-19, cont'd E,** Occlusal views before treatment, with orthodontics in progress, and after the completion of treatment. Note that the maxillary dental roots are crowded in the alveolus, which resulted in labial gingival recession.



• **Figure 20-19, cont'd F,** Articulated dental casts before analytic model planning. **G,** Lateral cephalometric radiographs before and after treatment.

• **Figure 20-20** A 16-year-old high school student was referred by her orthodontist for surgical evaluation. The Class III negative overjet malocclusion required orthodontics and jaw surgery. Because the patient had a lifelong history of nasal obstruction and consistent physical findings, intranasal procedures were also beneficial. Presurgical orthodontics was completed without extractions, and the maxillary incisor procumbency remained but without dental root crowding in the alveolar process. The patient's surgery included maxillary Le Fort I osteotomy in segments (horizontal advancement, vertical lengthening, arch expansion, and clockwise rotation); sagittal split ramus osteotomies (clockwise rotation); osseous genioplasty (minimal horizontal advancement); and septoplasty and inferior turbinate reduction. Note the aesthetic advantage of the maxillomandibular clockwise rotation in the presence of baseline excess incisor inclination. **A,** Frontal views in repose before and after treatment. **B,** Frontal views with smile before and after treatment.

• **Figure 20-20, cont'd C,** Oblique facial views before and after treatment. **D,** Profile views before and after treatment.

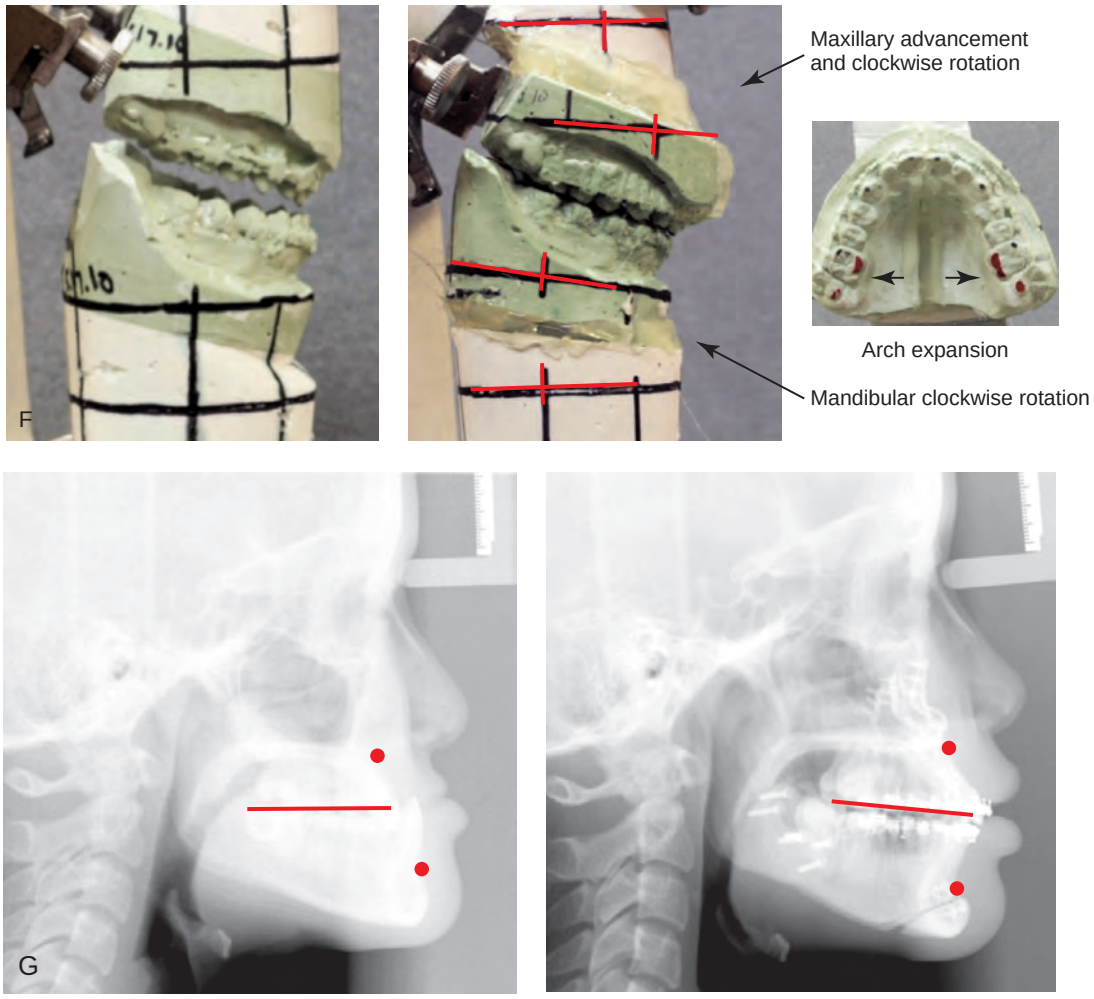
Continued

Prior to treatment

Pre surgery

After treatment

• **Figure 20-20, cont'd E,** Occlusal views before retreatment, with orthodontics in progress, and after the completion of treatment.



• **Figure 20-20, cont'd F,** Articulated dental casts before analytic model planning. **G,** Lateral cephalometric radiographs before and after treatment.

characterized by maxillary deficiency in combination with relative mandibular excess. The patients were divided into two groups. All patients underwent mandibular ramus osteotomies with a degree of set-back. The Group 1 study patients (n = 13) underwent mandibular set-back of 5 mm or more without simultaneous Le Fort I osteotomy. The Group 2 study patients (n = 13) underwent limited mandibular set-back, but their procedures were performed in combination with Le Fort I advancement. All study patients underwent preoperative and postoperative lateral cephalometric radiographs at standard intervals. Any changes that occurred in the position of the hyoid bone, the distance of the palate to the posterior nasal spine, the retrolingual space, and the hypopharyngeal space were measured from the postoperative radiographs. Preoperative and postoperative portable polysomnography were also carried out. The study confirmed that mandibular retropositioning of 5 mm or more can significantly decrease the posterior airway space and cause mild to moderate obstructive sleep apnea. The study also suggested that, when a limited mandibular set-back is carried out in combination with maxillary Le Fort I advancement, significant negative effects on the posterior airway space are likely prevented.

Postsurgical Orthodontic Maintenance and Detailing

The surgeon's effective management of the patient's in-hospital and at-home convalescence during the initial healing period (i.e., 5 weeks) is essential for a successful outcome (see Chapter 11). Cephalometric and Panorex radiographs and facial and occlusal photographs are obtained at standard postoperative intervals to document progress. We prefer to take these records at the 5-week postoperative interval as a matter of routine.

If segmental maxillary osteotomies were carried out, a prefabricated surgical splint will have been secured to the

maxillary occlusal surfaces during the operation. The orthodontist sees the patient within 24 hours of splint removal at approximately 5 weeks after surgery, and he or she replaces the maxillary sectional wires with a rigid continuous arch wire. The teeth are then ligated together to maintain the arch form. The use of a transpalatal wire or an acrylic palatal plate may also be used to stabilize the arch form if significant transverse expansion was carried out. Close orthodontic monitoring for skeletal and dental shifts and detailing during the first 6 months after surgery is essential.

Complications, Informed Consent, and Patient Education

The orthodontist and the surgeon are frequently asked to evaluate jaw disharmonies and malpositioned teeth and then to attempt to alter nature's events. With an understanding of the natural progression of the uncorrected skeletal Class III dentofacial deformity and knowledge of how specific interventions will affect head and neck function and facial aesthetics, predictable results are generally achieved.

Complications and patient dissatisfaction will always occur in a small percentage of individuals.[4,23,44,46,47,53,54,120] The clinician's ability to communicate the potential risks and complications, the alternative approaches available, and the benefits of the recommended treatment plan to the patient and the family is essential to achieve a favorable result. When the patient, the family, and all treating clinicians are fully informed, shared responsibility for the treatment outcome is more likely (see Chapter 16).

Malocclusion after Orthodontics and Orthognathic Surgery

There are inherent biologic challenges to the orthodontic movement of the teeth in the alveolar process and to the surgical repositioning of the jaws or jaw segments in the individual with maxillary deficiency and relative mandibular excess. These include the technical aspects of the repositioning, the initial healing requirements, and the difficulties associated with the long-term maintenance of the mechanically relocated teeth, jaws, and jaw segments. When following sound biomechanical and aesthetic principles, clinicians carry out orthodontic treatment and surgical procedures and then effectively maintain the results, in most cases. Potential intraoperative technical difficulties associated with sagittal split ramus osteotomies of the mandible and maxillary Le Fort I osteotomies can result in an immediate (i.e., early) malocclusion; however, these difficulties are generally preventable (see Chapter 17).*

Timing the surgery to limit ongoing mandibular growth afterward is usually possible, but it is more problematic for the skeletal Class III patient than the Class II patient (see Chapter 17). Significant condylar resorption after orthognathic surgery is rarely seen in the skeletal Class III patient (see Chapter 36).

Proffit and colleagues make a compelling argument that for the Class III patient, when the orthognathic procedure is a mandibular set-back only versus two-jaw surgery then relapse at the ramus osteotomy site is a common occurrence.[79,80] Research confirms better long-term skeletal and occlusal stability when two-jaw surgery (i.e., maxillary advancement with limited mandibular setback) is performed for the management of most Class III skeletal patterns.[81]

All of these factors must be taken into account to best achieve and then maintain the desired results.

Temporomandibular Disorders: The Effects of Orthodontics and Orthognathic Surgery

It is estimated that an average of 32% of the population reports at least one symptom of TMD, whereas an average of 55% demonstrate at least one clinical sign. TMD includes various signs and symptoms of the temporomandibular joint, the masticatory muscles, and related structures. The signs and symptoms may include a spectrum of referred head and neck pain; joint noise (e.g., popping, clicking, crepitus); reduced or altered mandibular movements as a result of muscle spasm or disc displacement, with or without reduction; condylar head erosion, and pain on direct palpation of either of the temporomandibular joints or the masticatory muscles.[3,5]

Occlusal factors (i.e., degrees of malocclusion) are often claimed to be associated with TMD. Interestingly, the correction of a skeletal Class III jaw deformity through orthodontics and orthognathic surgery often relieves the signs and symptoms of TMD, but this is not predictable for each individual patient (see Chapter 9).

Suboptimal Facial Aesthetics after Orthognathic Surgery

An accurate pretreatment facial analysis should limit the incidence of suboptimal aesthetics after surgery. Many of the aesthetic treatment planning pitfalls for the correction of skeletal Class III patterns were reviewed earlier in this chapter. Clearly stated and realistic objectives that are discussed and agreed upon by all parties are essential. Comprehensive and meticulous treatment planning, accurate surgical execution, and good patient cooperation further ensure a favorable result. Despite the following of these steps, patient and family dissatisfaction are to be expected in a small percentage of cases.[40,41,56,68,103,115] Maintaining open lines of communication, reasonable patient and family flexibility, and clinician responsiveness to ongoing needs and concerns will generally result in the satisfactory resolution of these issues (see Chapter 7).

*References 19, 34, 35, 43, 45, 55, 59-61, 67, 69, 79, 80, 82, 96, 100-102, 107, 108, 111, 116, 118

Conclusions

When evaluating the developmental dentofacial deformity of *maxillary deficiency in combination with relative mandibular excess,* attention to both the skeletal anomalies and the soft-tissue envelope distortions is essential. The orthodontist's immediate goal in preparation for jaw reconstruction is to remove all dental compensations and to place the teeth solidly in the alveolar process. If extractions are required to uncrowd the alveolar arch and to ensure the patient's periodontal health, so be it. The surgeon will then reposition the jaws to establish a functional occlusion, an improved upper airway, and enhanced facial aesthetics.

A Le Fort I advancement (beyond cephalometric norms) with vertical adjustment and clockwise rotation in combination with mandibular ramus osteotomies and chin reshaping is the key to the achievement of favorable maxillofacial convexity, A-point to B-point relationships, and enhanced aesthetics in most patients. As a result, the soft-tissue envelope is stretched; this results in the loss of unattractive skin folds and distortions as well as the lessening of the deep creases that would otherwise occur with age.

References

1. Alexander AEZ, McNamara JA, Jr, Franchi L, Baccetti T: Semilongitudinal cephalometric study of craniofacial growth in untreated Class III malocclusion. *Am J Orthod Dentofacial Orthop* 135:700.e1–700.e14, 2009.
2. Angle EH: *Treatment of malocclusion of the teeth: Angle's system*, ed 7, Philadelphia, 1907, SS White.
3. Athanasiou AE: Number and intensity of occlusal contacts following surgical correction of mandibular prognathism. *J Oral Rehabil* 19:145, 1992.
4. Athanasiou AE: Morphologic and functional implications of the surgical-orthodontic management of mandibular prognathism: A comprehensive review. *Am J Orthod Dentofacial Orthop* 103:439, 1993.
5. Athanasiou AE, Mavreas D: Tomographic assessment of alterations of the temporomandibular joint after surgical correction of mandibular prognathism. *Int J Adult Orthodon Orthognath Surg* 6:105, 1991.
6. Baccetti T, McGill JS, Franchi L, et al: Skeletal effects of early treatment of Class III malocclusion with maxillary expansion and face-mask therapy. *Am J Orthod Dentofacial Orthop* 113:333–343, 1998.
7. Baccetti T, Reyes BC, McNamara JA, Jr: Craniofacial changes in Class III malocclusion as related to skeletal and dental maturation. *Am J Orthod Dentofacial Orthop* 132:171.e1–171.e12, 2007.
8. Bailey LJ, White RP, Proffit WR, et al: Segmental Le Fort I osteotomy for management of transverse maxillary deficiency. *J Oral Maxillofac Surg* 55:728, 1997.
9. Becker R: Results in the treatment of prognathism and open bite with simultaneous tongue reduction. *Dtsch Zahnarztl Z* 17:892, 1960.
10. Bell WH: Le Fort I osteotomy for correction of maxillary deformities. *Oral Surg* 33:412–426, 1975.
11. Bell WH, Creekmore DC: Surgical orthodontic correction of mandibular prognathism. *Am J Orthod* 63:256, 1973.
12. Bell WH, Jacobs JD: Surgical orthodontic correction of horizontal maxillary deficiency. *J Oral Surg* 37:897, 1979.
13. Bell WH, White RP, Hall HP, et al: Mandibular excess. In Bell WH, Proffit WP, White R, editors: *Surgical correction of dentofacial deformities*, Philadelphia, 1980, WB Saunders, pp 234–441.
14. Betts NJ, Vanarsdale RL, Barber HD, et al: Diagnosis and treatment of transverse maxillary deficiency. *Int J Adult Orthodon Orthognath Surg* 10:75, 1995.
15. Blair VP: *Surgery of the mouth and jaws*, St Louis, 1914, Mosby.
16. Bloomquist DS: The stability of segmentalized Le Fort I osteotomies with miniplate fixation in patients with maxillary hypoplasia [discussion]. *J Oral Maxillofac Surg* 53:1412, 1995.
17. Broadbent HB, Broadbent HB, Jr, Golden WH: *Bolton standards of dentofacial developmental growth*, St Louis, 1975, CV Mosby.
18. Brunell JA, Bhat M, Lipton JA: Prevalence and distribution of selected occlusal characteristics in the US population. 1988–1991. *J Dent Res* 75:706, 1996.
19. Busby BR, Bailey LJ, Proffit WR, et al: Long-term stability class III treatment: A study of 5-year postsurgical results. *Int J Adult Orthodon Orthognath Surg* 17:159, 2002.
20. Capelozza L, Martins A, Mazzotini R, da Silva HG: Effects of dental decompensation on the surgical treatment of mandibular prognathism. *Int J Adult Orthodon Orthognath Surg* 11:165–180, 1996.
21. Cevidanes L, Baccetti T, Franchi L, et al: Comparison of two protocols for maxillary protraction: Bone anchors versus face mask with rapid maxillary expansion. *Angle Orthod* 80:799–806, 2010.
22. Chamberland S, Proffit WL: Short-term and long-term stability of surgically assisted rapid palatal expansion revisited. *Am J Orthod Dentofacial Orthop* 139:815–822, 2011.
23. Cho HJ: Effect of rigid fixation on orthodontic finishing after mandibular bilateral sagittal split setback: The case for miniplate monocortical fixation. *J Oral Maxillofac Surg* 70:e310–e321, 2012.
24. Chong Y, Ive JC, Årtun J: Changes following the use of protraction headgear for early correction of Class III malocclusion. *Angle Orthod* 66:351–362, 1996.
25. De Clerck H, Cevidanes L, Baccetti T: Dentofacial effects of bone-anchored maxillary protraction: A controlled study of consecutively treated Class III patients. *J Oral Maxillofac Surg* 138(5):577–581, 2010.
26. Demetriades N, Chang DJ, Laskarides C, Papageorge M: Effects of mandibular retropositioning, with or without maxillary advancement, on the oro-naso-pharyngeal airway and development of sleep-related breathing disorders. *J Oral Maxillofac Surg* 68:2431–2436, 2010.
27. Downs WB: Variations in facial relationships: Their significance in treatment and prognosis. *Am J Orthod Dentofacial Orthop* 34:812, 1948.
28. El-Gheriani AA, Maher BS, El-Gheriani AS, et al: Segregation analysis of mandibular prognathism in Libya. *J Dent Res* 82:523–527, 2003.
29. Ellis E, III, McNamara JA, Jr: Components of adult Class III open bite malocclusion. *Am J Orthod Dentofacial Orthod* 86:277, 1984.
30. Epker BN, Fish LC: The surgical-orthodontic correction of class III skeletal open bite. *Am J Orthod Dentofacial Orthop* 73:601, 1978.
31. Epker BN, Fish LC, Paulus PJ: The surgical-orthodontic correction of maxillary deficiency. *Oral Surg Oral Med Oral Pathol* 46:171, 1978.
32. Epker BN, Turvey TA, Fish LC: Indications for simultaneous mobilization of the maxilla and mandible for the correction of dentofacial deformities. *Oral Surg Oral Med Oral Pathol* 54:369, 1982.
33. Farkas LG, Sohm P, Kolar JC, et al: Inclinations of the facial profile: Art versus reality. *Plast Reconstr Surg* 75:509, 1985.
34. Gu G, Gu G, Nagata J, et al: Hyoid position, pharyngeal airway and head posture in relation to relapse after the mandibular

setback in skeletal Class III. *Clin Orthod Res* 3:67–77, 2000.
35. Hirdnaka D, Kelly J: Stability of simultaneous orthognathic surgery on the maxilla and mandible: A computer assisted cephalometric study. *Int J Adult Orthodon Orthognath Surg* 2:193, 1987.
36. Hohl TH, Epker BN: Macrogenia: A study of treatment results with surgical recommendations. *J Oral Surg* 41:545, 1976.
37. Ishii H, Morita S, Takeuchi U, Nakamura S: Treatment effect of combined maxillary protraction and chincap appliance in severe skeletal Class III cases. *Am J Orthod Dentofacial Orthop* 92:304–312, 1987.
38. Jabaley ME, Edgerton ME: Surgical correction of congenital midface retrusion in the presence of mandibular prognathism. *Plast Reconstr Surg* 44:1, 1969.
39. Jacobson A, Evans WG, Preston CB, Sadowsky PL: Mandibular prognathism. *Am J Orthod* 66:140–171, 1974.
40. Joss CU, Joss-Vassalli IM, Bergé SJ, Kuijpers-Jagtman AM: Soft tissue profile changes after bilateral sagittal split osteotomy for mandibular setback: A systematic review. *J Oral Maxillofac Surg* 68:2792–2801, 2010.
41. Kawamoto HK, Jr: Reduction mentoplasty [discussion]. *Plast Reconstr Surg* 70:151, 1982.
42. Kelly JE, Harvey C: *An assessment of the teeth of youths 12-17 years. Department of Health, Education, and Welfare Publication No (HRA) 77-1644*, Washington, DC, 1977, National Center for Health Statistics.
43. Kim YG, Oh SH: Effect of mandibular setback surgery on occlusal force. *J Oral Maxillofac Surg* 55:121, 1997.
44. Kobayashi T, Honma K, Nakajima T, et al: Masticatory function in patients with mandibular prognathism before and after orthognathic surgery. *J Oral Maxillofac Surg* 51:997, 1993.
45. Kobayashi T, Watanabe I, Ueda K, et al: Stability of the mandible after sagittal ramus osteotomy for correction of prognathism. *J Oral Maxillofac Surg* 44:693, 1986.
46. Kondo E: The course of treatment of a class III skeletal open bite case and its analysis. *Aust Orthod J* 12:217, 1993.
47. LaBlanc JP, Turvey TA, Epker BN: Results following simultaneous mobilization of the maxilla and mandible for the correction of dentofacial deformities: Analysis of 100 consecutive patients. *J Oral Surg* 54:607, 1982.
48. Lew KK, Foong WC, Loh E: Malocclusion prevalence in an ethnic Chinese population. *Aust Dent J* 38:8, 1993.
49. Liao YF, Chiu YF, Huang CS, et al: Presurgical orthodontics versus no presurgical orthodontics: Treatment outcomes of surgical-orthodontic correction for skeletal class III open bite. *Plast Reconstr Surg* 126:2074–2083, 2010.
50. de Lira Ade L, de Moura WL, de Barros Vieira JM, et al: Surgical predication of skeletal and soft tissue changes in Class III treatment. *J Oral Maxillofac Surg* 70:e290–e297, 2012
51. Luhr HG: Zur stabilen osteosynthese bei unterkiefer–frakturen. *Dtsch Zahnarztl Z* 23:754, 1968.
52. Luhr HG: Skelettverlagernde operationen zur harmonisierung des gesichtsprofils—probleme der stabilen fixation von osteotomiesegmenten. In Pfeifer G, editor: *Die asthetik von form und funktion in der plastischen und wiederherstellungschirurgie*, Berlin, 1985, Springer-Verlag, p 87.
53. Magnusson T, Ahlborg G, Svartz K: Function of the masticatory system in 20 patients with mandibular hypo- or hyperplasia after correction by a sagittal split osteotomy. *Int J Oral Maxillofac Surg* 19:289, 1990.
54. McHugh M, Van Sickels JE: Effect of stabilization of a bilateral sagittal split on orthodontic finishing after mandibular setback: A case for bicortical fixation. *J Oral Maxillofac Surg* 70:e301–e309, 2012.
55. McNance AM, Moss JP, James DR: Stability of surgical correction of patients with skeletal III and skeletal II anterior open bite, with increased maxillary mandibular plane angle. *Eur J Orthod* 14:198, 1992.
56. McNance AM, Moss JP, Wright WR, et al: A three-dimensional soft tissue analysis of 16 skeletal class III patients following bimaxillary surgery. *Br J Oral Maxillofac Surg* 30:221, 1992.
57. Milicesco V: La valeur de la photographie, de la crâniometrie et de la téléradiographie dans le prognathisme mandibulaire anatomique. *Rev Stomatol* 69:474–480, 1968.
58. Miotti A: Rassegna bibliografica sull'eziologia ed epidemiologia del prognatismo mandibolare. *Dent Cadmos* 50:29–32, 1982.
59. Moser K, Freihofer HP: Long-term experience with simultaneous movement of the upper and lower jaw. *J Oral Maxillofac Surg* 8:271, 1980.
60. Nagai I, Tanaka N, Noguchi M, et al: Changes in occlusal state of patients with mandibular prognathism after orthognathic surgery: A pilot study. *Br J Oral Maxillofac Surg* 39:429, 2001.
61. Nagamine T, Kobayasgi T, Nakajima T, et al: The effects of surgical-orthodontic correction of skeletal class III malocclusion on mandibular movement. *J Oral Maxillofac Surg* 51:385, 1993.
62. Norholt SE, Sindet-Pedersen S, Jensen J: An extended Le Fort I osteotomy for correction of midface hypoplasia: A modified technique and results in 35 patients. *J Oral Maxillofac Surg* 54:1297, 1996.
63. Obwegeser HL: The surgical correction of mandibular prognathism and retrognathia with consideration of genioplasty. Part 1. *Oral Surg* 10:677, 1957.
64. Obwegeser HL: Eingriffe am overkiefer zur korrektur des progenen zustandsbildes. *Schweiz Monatschr Zahnheilkd* 75:365–373, 1965.
65. Obwegeser HL: Surgical correction of small or retrodisplaced maxillae. *Plast Reconstr Surg* 43:352–365, 1969.
66. Obwegeser HL: Die einzeitige vorbewegung des oberkiefers and ruckbewegung des unterkiefers zur korrektur der extremen "progenie." *Schweiz Monatschr Zahnheilkd* 80:547–555, 1970.
67. Ohkura K, Harada K, Morishima S, et al: Changes in bite force and occlusal contact area after orthognathic surgery for correction of mandibular prognathism. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 91:141, 2001.
68. Papadopoulos MA, Lazaridou-Terzoudi T, Oland J, et al: Comparison of soft and hard tissue profiles of orthognathic surgery patients treated recently and 20 years earlier. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 108:e8, 2009.
69. Pepersack WJ, Chausse JM: Long-term follow-up of the sagittal split technique for correction of mandibular prognathism. *J Maxillofac Surg* 6:117–140, 1978.
70. Perthes G: Operative korrektur der progenie. *Zentralbl Chir* 49:1540–1541, 1922.
71. Phillips C, Medland WH, Fields HW, Jr: Stability of surgical maxillary expansion. *Int J Adult Orthodon Orthognath Surg* 7:139, 1992.
72. Posnick JC: Mandibular excess and maxillary deficiency. In Posnick JC, editor: *Craniofacial and maxillofacial surgery in children and young adults,* Vol 38, Philadelphia, 2000, WB Saunders Co, pp 1004–1024.
73. Posnick JC, Agnihotri N: Managing chronic nasal airway obstruction at the time of orthognathic surgery: A twofer. *J Oral Maxillofac Surg* 69(3):695–701, 2011.
74. Posnick JC, Fantuzzo J, Orchin J: Deliberate operative rotation of the maxillo-mandibular complex to alter the A-point to B-point relationship for enhanced facial esthetics. *J Oral Maxillofac Surg* 64:1687–1695, 2006.
75. Posnick JC, Fantuzzo JJ, Troost T: Simultaneous intranasal procedures to improve chronic obstructive nasal breathing in patients undergoing maxillary (Le Fort I) osteotomy. *J Oral Maxillofac Surg* 65:2273–2281, 2007.
76. Proffit W, Fields H, Jr: *Contemporary orthodontics*, ed 3, St Louis, 2007, Mosby.
77. Proffit WR, Fields HW, Moray LJ: Prevalence of malocclusion and orthodontic treatment need in the United States: Estimates from the N-HANES III survey. *Int J Adult Orthodon Orthognath Surg* 13:97–106, 1998.
78. Proffit WR, Phillips C, Dann C, IV: Who seeks surgical orthodontic treatment? *Int J Adult Orthodon Orthognath Surg* 3:153, 1990.
79. Proffit WR, Phillips C, Dann C, IV, et al: Stability after surgical-orthodontic correction of skeletal class III malocclusion: 1.

Mandibular setback. *Int J Adult Orthodon Orthognath Surg* 6:7, 1991.
80. Proffit WR, Phillips C, Dann C, Turvey TA: Stability after surgical-orthodontic correction of skeletal class III malocclusion: 3. Combined maxillary and mandibular procedures. *Int J Adult Orthodon Orthognath Surg* 6:221, 1991.
81. Proffitt WR, Phillips C, Turvey TA: Stability after mandibular setback: Mandible-only versus 2-jaw surgery. *J Oral Maxillofac Surg* 70:e408–e414, 2012.
82. Reitzik M: Skeletal and dental changes after surgical correction of mandibular prognathism. *J Oral Surg* 38:109, 1980.
83. Reyes BC, Baccetti T, McNamara JA, Jr: An estimate of craniofacial growth in Class III malocclusion. *Angle Orthod* 76:577–784, 2006.
84. Reyneke JP, Evans WG: Surgical manipulation of the occlusal plane. *Int J Adult Orthodon Orthognath Surg* 5:99–110, 1990.
85. Rosen H: Facial skeletal expansion: Treatment strategies and rationale. *Plast Reconstr Surg* 89:798, 1992.
86. Rosen H: Aesthetics in facial skeletal surgery. *Perspect Plast Surg* 6:1, 1993.
87. Rosen HM: Porous block hydroxyapatite as an interpositional bone-graft substitute in orthognathic surgery. *Plast Reconstr Surg* 83:985, 1989.
88. Rosen HM: Segmental osteotomies of the maxilla. *Clin Plast Surg* 16:785, 1989.
89. Rosen HM: Maxillary advancement for mandibular prognathism: Indication and rationale. *Plast Reconstr Surg* 87:823, 1991.
90. Sakamoto E, Sugawara J, Umemori M, Mitani H: Craniofacial growth of mandibular prognathism during pubertal growth period in Japanese boys: Longitudinal study from 10–15 years of age. *Jpn J Orthod Soc* 55:372–386, 1996.
91. Samman N, Tong AC, Cheung LK, et al: Analysis of 300 dentofacial deformities in Hong Kong. *Int J Adult Orthodon Orthognath Surg* 7:5, 1992.
92. Sar C, Arman-Özçirpici A, Uçkan S, Yazici AC: Comparative evaluation of maxillary protraction with or without skeletal anchorage. *Am J Orthod Dentofacial Orthop* 139:636–649, 2011
93. Sassouni V, Nanda S: Analysis of dentofacial vertical proportions. *Am J Orthod* 50:801, 1964.
94. Scholssmann A: Cited by G Perthes: Operative korrektur der progenie. *Zentralbl Chir* 49:1540–1541, 1922.
95. Silverstein K, Quinn PD: Surgically-assisted rapid palatal expansion for management of transverse maxillary deficiency. *J Oral Maxillofac Surg* 55:725, 1997.
96. Simpson W: The results of surgery for mandibular prognathism. *Br J Oral Surg* 12:166, 1974.
97. Singh GD: Morphologic determinants in the etiology of class III malocclusion: A review. *Clin Anat* 12:382–405, 1999.
98. Singh GD, McNamara JA, Jr, Lozanoff S: Procrustes, Euclidean and cephalometric analysis of the morphology of the mandible in human class III malocclusions. *Arch Oral Biol* 43:535–543, 1998.
99. Skaloud F: New surgical method for correction of prognathism of the mandible. *Oral Surg Oral Med Oral Pathol* 4:689–694, 1951.
100. Sorokolit CA, Nanda RS: Assessment of the stability of mandibular setback procedures with rigid fixation. *J Oral Maxillofac Surg* 48:817, 1990.
101. Stea G: Notre experience dans le transientdu prognatism. *Rev Stomatol* 72:559, 1971.
102. Stella JP, Astrand P, Epker BN: Patterns and etiology of relapse after correction of class III open bite via subcondylar ramus osteotomy. *Int J Adult Orthodon Orthognath Surg* 1:91, 1986.
103. Stella JP, Streater MR, Epker BN, Sinn DP: Predictability of upper lip soft tissue changes with maxillary advancement. *J Oral Maxillofac Surg* 47:697, 1989.
104. Stiles KA, Luke JE: The inheritance of malocclusion due to mandibular prognathism. *J Hered* 44:241–245, 1953.
105. Sugawara J, Tsuchikawa T, Soya T, Mitani H: Late adolescent growth of skeletal Class III craniofaces in Japanese girls: Average growth from 14–17 years of age based on longitudinal data [Japanese]. *Nihon Kyosei Shika Gakkai Zasshi* 42:399–408, 1983.
106. Thompson EM, Winter RM: Another family with the "Habsburg jaw." *J Med Genet* 25:838–842, 1988.
107. Throckmorton GS: Masticatory function in patients with mandibular prognathism before and after orthognathic surgery [discussion]. *J Oral Maxillofac Surg* 51:1002, 1993.
108. Throckmorton GS: Effect of mandibular setback surgery on occlusal force [discussion]. *J Oral Maxillofac Surg* 55:126, 1997.
109. Tollaro I, Baccetti T, Franchi L: Class III malocclusion in the deciduous dentition: A morphological and correlation study. *Eur J Orthod* 16:401–408, 1994.
110. Tornes K: Extraoral and intraoral vertical subcondylar ramus osteotomy for correction of mandibular prognathism. *Int J Oral Maxillofac Surg* 16:671, 1987.
111. Tornes K, Wisth PJ: Stability after vertical subcondylar ramus osteotomy for correction of mandibular prognathism. *Int J Oral Maxillofac Surg* 17:242, 1988.
112. Trauner R, Obwegeser H: The surgical correction of mandibular prognathism and retrognathia with consideration of genioplasty. II. Operating methods for microgenia and distoocclusion. *Oral Surg Oral Med Oral Pathol* 10:787–792, 1957.
113. Turvey TA, Hall DJ, Fish LC, Epker BN: Surgical orthodontic treatment planning for simultaneous mobilization of the maxilla and mandible in the correction of dentofacial deformities. *Oral Surg Oral Med Oral Pathol* 54:491, 1982.
114. Vanarsdale R, White R: Three-dimensional analysis for skeletal problems. *Int J Adult Orthodon Orthognath Surg* 9:159, 1994.
115. Van der Dussen FN, Egyedi P: Premature aging of the face after orthognathic surgery. *J Craniofac Surg* 18:335, 1990.
116. Wardrop RW, Wolford LM: Maxillary stability following downgraft and/or advancement procedures with stabilization using rigid fixation and porous block hydroxyapatite implants. *J Oral Maxillofac Surg* 47:336, 1989.
117. Watanabe M, Suda N, Ohyama K: Mandibular prognathism in Japanese families ascertained through orthognathically treated patients. *Am J Orthod Dentofacial Orthop* 128:466–470, 2005.
118. Westwood P, McNamara JA, Jr, Baccetti T, et al: Long-term effects of Class III treatment with rapid maxillary expansion and facemask therapy followed by fixed appliances. *Am J Orthod Dentofacial Orthop* 123:306–320, 2003.
119. Williams MD, Sarver DM, Sadowsky PL, et al: Combined rapid maxillary expansion and protraction facemask in the treatment of class III malocclusion in growing children: A prospective long-term study. *Semin Orthod* 3:10, 1997
120. Wisth PJ: Mandibular function and dysfunction in patients with mandibular prognathism. *Am J Orthod* 85:193–198, 1984.
121. Wolfe SA, Berkowitz S: Combined maxillary and mandibular procedures: Sequential or simultaneous. In *Plastic surgery of the facial skeleton*, Boston, 1989, Little, Brown, p 417.
122. Wollf G, Wienker TF, Sander H: On the genetics of mandibular prognathism: Analysis of large European noble families. *J Med Genet* 30:112–116, 1993.
123. Wilhelm-Nold I, Droschl H: Die frühbehandlung der progenie im milchgebiß im vergleich zur behandlung im wechselgebiß. *J Orofacial Orthopedics/Fortschr Kieferorthop* 51(3):165–179, 1990.
124. Yamaguchi T, Park SB, Narita Maki K, Inoue I: Genomewide linkage analysis of mandibular prognathism in Korean and Japanese patients. *J Dent Res* 84:255–259, 2005.
125. Zhou Y, Hagg U, Rabie AB: Severity of dentofacial deformity, the motivations and the outcome of surgery in skeletal Class III patients. *Chin Med J (Engl)* 115:1031, 2002.

21

Long Face Growth Patterns: Maxillary Vertical Excess with Mandibular Deformity

JEFFREY C. POSNICK, DMD, MD

- Functional Aspects
- Clinical Characteristics
- Dental Findings
- Radiographic Findings
- Etiologic Considerations
- The Role of Growth Modification in the Preadolescent Patient
- The Orthodontic Camouflage Approach
- An Orthodontic Molar Intrusion Approach
- Definitive Reconstruction
- Surgical Planning Pitfalls
- Postsurgical Orthodontic Maintenance and Detailing
- Complications, Informed Consent, and Patient Education
- Malocclusion after Orthodontics and Orthognathic Surgery
- Temporomandibular Disorders: Effects of Orthodontics and Orthognathic Surgery
- Conclusions

The prevalence of a *long face growth pattern* at a prominent U.S. maxillofacial center was documented as representing 22% of jaw deformity evaluations as judged from the parameter of excessive anterior facial height.[114] The lower anterior facial height was specifically long in 88% of the affected individuals.[54,74,75] The term *long face* is generally used to describe a maxillofacial growth pattern that typically also includes a gummy smile, an anterior open bite, and a lack of chin prominence. This combination of problems seems to be harder for the individual to live with; it results in a personal and family decision to seek treatment more frequently than most other patterns of dentofacial deformity (e.g., mandibular deficiency alone).[112] Halfway around the world, in South Africa, Reyneke estimated that, in his orthognathic practice, approximately 30% of those seeking treatment have a long face with complaints about a gummy smile and concerns caused by malocclusion.[117]

The basic diagnostic criteria for a long face growth pattern include an excessive anterior facial height (i.e., the elongation of the lower third) that leads to disproportion between facial height and width.[26,28,69,70] Studies confirm that the excessive eruption of the posterior maxillary dentition causes the mandible to rotate downward and backward (clockwise rotation) and results in increased lower facial height.[24,34] This makes any baseline mandibular anteroposterior deficiency seem worse and any true mandibular prognathism seem better.[31,123] As the mandible rotates down and back during facial development, the lower incisors often adapt by becoming more upright, with crowding. The mandibular clockwise rotation tends to separate the upper and lower incisors vertically, thereby creating an open bite with an accentuated curve of Spee.[76] Not all long face growth patterns involve an anterior open bite. The maxillary incisors also supra-erupt.[111,139] The anterior bite may stay closed or even become deep, depending on the extent of maxillary and mandibular incisor supra-eruption.[5,23,71,93-97,133] Whether the incisor relationship remains deep, normal, or open, lip incompetence (i.e., ≥4 mm of lip separation at rest) occurs, with mentalis strain that results from attempts to achieve lip closure.[27,99,100]

Functional Aspects

- Wide lip separation occurs, with excessive effort required during attempts to achieve lip seal.
- Chronic obstructive nasal breathing as a result of increased intranasal airway resistance (e.g., septal

deviation, enlarged inferior turbinates, tight nasal aperture, elevated nasal floor) is frequent (see Chapter 10).
- Classic articulation errors in speech as a result of the anterior open bite and lip incompetence are expected (see Chapter 8).
- Chewing and swallowing difficulties caused by the anterior open-bite malocclusion are expected (see Chapter 8).
- Detrimental effects on the dentition and the periodontium as a result of long-term traumatic malocclusion, the displacement of the teeth outside of the alveolar bone housing, or previous camouflage orthodontic and restorative treatments may be seen.
- The incidence of temporomandibular disorder may be higher than it is among individuals with normal jaw harmony and occlusion (see Chapter 9).

Clinical Characteristics

- Excessive anterior facial height, primarily in the lower third of the face
- Lip incompetence (i.e., resting lip separation of ≥4 mm)
- Increased exposure of the gingiva with a broad smile (i.e., a gummy smile)
- Increased frequency of anterior open bite (although one third of patients present with a normal or deep bite and only 15% have an open bit of >4 mm)
- Mandibular deficiency with Class II malocclusion is frequent (this can range from Class I to Class III)
- A mandible that is rotated downward and backward (i.e., clockwise rotation)
- An excessively full, curled lower lip when in a relaxed position
- Increased vermillion exposure of the lower lip
- A constricted or narrow alar base width, with depressed paranasal regions
- The appearance of a flat or sunken midface, without aesthetically pleasing facial curves or contours
- A negative vector relationship of the ocular globe to the lower eyelid

Dental Findings

- Anterior open bite (may be normal or deep)
- Excessive alveolar height in the anterior maxillary region (this will raise the floor of the nose and partially obstruct nasal airflow)
- Mandibular incisors that are upright, with crowding
- Accentuated curve of Spee as a result of the overeruption of the mandibular incisors
- Tendency toward a narrow, V-shaped maxillary arch with posterior crossbites (this is seen in approximately 50% of patients)
- Occlusal surface enamel wear on the posterior teeth as a result of grinding (secondary occlusal trauma) or previous occlusal equilibration performed in an attempt to close the anterior open bite

Radiographic Findings

- Excessive eruption of the maxillary posterior teeth (i.e., the distance from the palatal plane to the cusps of the molars)
- Maxillary horizontal deficiency
- Clockwise rotation of the maxilla (i.e., an increased palatal plane angle)
- Increased lower anterior facial height
- Clockwise rotation of the mandible (i.e., an increased mandibular plane angle)
- Normal or short mandibular ramus height
- Mandibular posterior teeth not excessively erupted
- Excessive eruption of the maxillary and mandibular incisors: with enough anterior hypereruption, a normal or deep overbite is more likely
- Measurable excessive eruption of the maxillary incisors (i.e., the distance from the floor of the nose to the incisal edge)
- Measurable excessive eruption of the mandibular incisors (i.e., the distance from the menton to the incisal edge)

Fields and colleagues presented simplified cephalometric diagnostic criteria.[44] If a patient has all three of the following criteria, he or she can be considered to have a long face growth pattern:

1. An increased mandibular plane angle
2. An increased total anterior facial height
3. A decreased percentage of upper to lower facial height

Etiologic Considerations

Current thinking is that both environmental influences and inherited tendencies play a role in the development of a long face.[53,66,67,85-87] A significant percentage of patients with long faces have been found to have an increased oral and decreased nasal breathing pattern.[25,33,37,52,59,82,134,146,150] Interestingly, nasal obstruction is not always present with long face growth patterns, but an open-mouth posture during development is typical. Other causes of an open-mouth posture in the preadolescent patient have been shown to result in similar long face dysmorphology.[1,2,4,51,61,78,80,140] This includes muscle weakness syndromes (e.g., Moebius syndrome, facial nerve palsy, cerebral palsy, muscular dystrophy) and the presence of a large tongue (e.g., Beckwith-Wiedemann syndrome, hemangioma, vascular malformations, neurofibromatosis, Proteus syndrome; see Chapter 4).

There is experimental animal model research to support the theory that a long face anterior open-bite malocclusion is a physiologic adaptation to an open-mouth protrusive tongue posture that is present during facial development (see Chapters 4 and 10).[62-65] It has also been clarified that, in humans, a resting open-mouth protrusive tongue posture is an etiologic factor in the development of a long face with an anterior open bite. Interestingly, the intermittent occurrence of tongue thrust during speech and swallowing is not

thought to be the cause of an open bite. In fact, periodic tongue thrusting is an adaptive response to an open bite rather than the cause.[47] When the incisors overlap in an average way (i.e., with a normal overbite), the normal-sized tongue is naturally placed behind the teeth to create the necessary anterior seal for successful swallowing and the articulation of specific consonant sounds (see Chapter 8). In the presence of anterior open bite, the normal tongue must protrude past the teeth to seal against the lips. This is an expected adaptive mechanism to assist with swallowing and speech. Confirmation that the protrusive tongue position during swallowing and speech is a reversible adaptation has been routinely documented after the individual with a long face and an anterior open bite undergoes successful orthognathic surgery and orthodontic treatment.[89,142,155] When the open bite is closed with the use of sound biologic mechanics (i.e., without orthodontic dental compensation) and when lip competence is achieved through surgery (i.e., there is no longer excess lower facial height), there is a fairly reliable elimination of the tongue thrust without the need for active myofunctional physical therapy retraining. However, if a skeletal anterior open bite has been closed with the use of unsound orthodontic maneuvers (i.e., excessive eruption of the anterior teeth or buccal tipping of the molars outside of the alveolar bone housing) and if lip competence is not achieved (i.e., inadequate facial height shortening), dental relapse is to be expected. In this case, tongue thrusting should not be blamed for the recurrent malocclusion.

The Role of Growth Modification in the Preadolescent Patient

When standard growth modification maneuvers for a long face growth pattern are attempted in the preadolescent patient, the primary objective is to restrain the hypereruption of the posterior maxillary teeth. In theory, this would limit the abnormal downward and backward rotation of the mandible. Unfortunately, effective and practical methods for accomplishing this seemingly limited objective have been difficult to orchestrate. It has also been shown that the natural progression of the deformity may persist through adolescence. This means that the effective containment of the maxillary posterior dental hypereruption would need to continue throughout the teenage years. The two traditional methods that are used to impede excessive vertical growth of the posterior maxilla are 1) the use of *high-pull headgear* connected to a complete or partial maxillary fixed appliance and 2) the use of a *functional intra-oral appliance* that incorporates a bite-block device (i.e., an activator) between the teeth.

The intraoral functional appliance is usually designed to exert force on the posterior teeth when the masticatory muscles contract. The occlusal force is then transferred through the biting surfaces of the upper and lower posterior teeth to the periodontal ligaments to limit molar eruption. To be effective, most clinicians recommend that the functional appliance be worn for 14 to 16 hours per day. When headgear is used, it is designed to apply direct external force to the teeth through a fixed maxillary appliance. Unfortunately, true vertical pull is almost impossible to achieve, and this results in a degree of added backward pressure. Because the majority of patients with long faces do not have protrusion of the maxilla, this unavoidable posterior force is counterproductive and results in further horizontal maxillary deficiency. The combination of vertical intrusion vectored headgear and an intra-oral functional appliance to affect the posterior dentition—when initiated during childhood and sustained through the teenage years in a compliant child—is thought to have the potential to produce improvement in the patient with a long face growth pattern. In all but borderline long faces, which are recognized during childhood, growth modification is not likely the preferred approach.

The Orthodontic Camouflage Approach

The patient with a long face often perceives a visual prominence of his or her upper incisors. For this reason, it is apparently tempting for the orthodontist to proceed with a camouflage treatment plan to neutralize the occlusion via the retraction of the upper incisors. This approach seems to be especially difficult for the orthodontist to avoid when viewing the problem primarily as a Class II malocclusion without fully considering the vertical and horizontal skeletal disharmony, the lip incompetence, the upper airway dysfunction, and the overall facial aesthetics. The extraction of the maxillary first premolars followed by the retraction of the anterior teeth will improve and may even neutralize the occlusion, but it does nothing to relieve the vertical facial problem, and it will further accentuate the horizontal facial deficiency. As the incisors are orthodontically retracted, they are also elongated (i.e., extruded), and the nasolabial angle opens, which results in unattractive effects on the lips and the nose.

Before embarking on an orthodontic camouflage approach, the clinician should confirm that the patient and his or her family understand the aesthetic limitations of closing the anterior open bite by extruding the incisors (i.e., residual or worsening gummy smile, residual lip incompetence with mentalis strain, lack of facial aesthetic improvement). The patient's long-term dental stability and periodontal health must also be considered. In addition, the maxilla, the mandible, and the chin will remain horizontally deficient and vertically long, thereby further accentuating lip incompetence and the long, flat, retrusive appearance of the profile.

An Orthodontic Molar Intrusion Approach

Sato and colleagues believe high-angle Class II malocclusions can be corrected with the use of "multiloop edgewise

arch wires."[124] With the use of tip-back mechanics and vertical elastics, the researchers believe that the occlusal plane inclination can be altered by intruding the molars via the autorotation of the mandible and the closure of the open bite. Unfortunately, scientific evidence in support of this method of treatment for a patient with a long face and an anterior open bite has not been forthcoming.

With the use of skeletal temporary anchorage devices, the intrusion of the molars has been recommended by some to close an open bite.[105,157,158] Man-Suk and colleagues completed a study to examine the long-term stability of anterior open-bite correction via the intrusion of the maxillary posterior teeth in adults.[88] In the first group (n = 5 adults), mini-screw implants were placed on the buccal and palatal sides, between the roots of the maxillary second premolars and first molars and between the roots of the maxillary first and second molars. Intrusion forces were directly applied to the molars with elastomeric chains. In the second group (n = 4 adults), mini-screw implants were placed on the buccal side only between the roots of the maxillary second premolars and first molars and between the roots of the maxillary first and second molars. A rigid transpalatal arch was placed before applying the intrusive forces to prevent the buccal tipping of the teeth. Lateral cephalograms were taken at the initial evaluation and again at 1 and 3 years after treatment. The cephalograms were traced and analyzed. The study documented that the maxillary first molars were intruded on an average of 2.39 mm during treatment, with relapse of 0.45 mm at the 3-year follow up (i.e., 22.9% relapse). A majority of the relapse occurred during the initial posttreatment year. The molar relapse had a significant effect on the eventual incisal overbite. There was no significant difference in molar relapse or the maintenance of overbite between the two different methods of skeletal anchorage used. It would appear as though there is currently no known way to fully retain an orthodontically intruded molar.

For patients with borderline long face growth patterns in whom the open bite has been successfully closed with the use of orthodontic mechanics and in whom the remaining gummy smile and lip incompetence is only mild, a vertical reduction with horizontal advancement genioplasty may improve the profile aesthetics and lip competence enough to be a satisfactory compromise for the patient (see Chapter 37 and 40).

Definitive Reconstruction

Timing of Orthognathic Surgery

Orthodontic or surgical interventions undertaken in the individual with a long face growth pattern will not be definitive if significant jaw growth continues after treatment. In our experience, when the increased intranasal airway resistance is simultaneously managed as part of the definitive jaw reconstruction, excessive ongoing vertical growth ceases.[108,110] Even if minor degrees of vertical growth were to continue, it should occur in both the mandible and the maxilla in a proportionate way.

If the individual with a long face is either a normal mandibular grower (i.e. Class I) or a mandibular-deficient grower (i.e. Class II), then after he or she is in the permanent dentition and the teeth are orthodontically aligned, ongoing horizontal jaw growth after reconstruction is unlikely.

Horizontal jaw growth maturation may be analyzed with the use of serial lateral cephalometric radiographs. These sequential radiographs can be used to make direct mandibular measurements. Surgery is delayed until the deceleration of growth can be documented. The best evidence available indicates that, when orthognathic surgery is undertaken in the mandibular-deficient patient as early as completion of the adolescent growth spurt, forward growth of the mandible almost never occurs.[135] Further horizontal growth of the maxilla at this age is also not expected.

For all of these reasons, proceeding with definitive orthognathic and intranasal surgery for the patient with a Class II long face growth pattern when he or she is as young as 14 years old is generally safe from a growth perspective, but only after the eruption and orthodontic alignment of the teeth (Fig. 21-4).

Preorthodontic Periodontal Considerations

Assessing the individuals biologic and anatomic risk factors for periodontal injury should be undertaken before starting orthodontic treatment.

Orthodontic attempts to compensate the occlusion in the patient with a long face growth pattern may result in gingival recession along the labial aspect of the lower incisors and the buccal aspect of the posterior maxillary teeth. The advantage of bicuspid extractions in the mandible or the maxilla should always be considered to achieve full positioning of the dental roots solidly in the alveolar bone (see Chapter 6).

When lip competence and an improved nasal airway have been achieved as a result of jaw and intranasal procedures, the dryness of the gingiva (ex. inflammation) will also diminish (see Chapter 6).

Orthodontic Preparation for Orthognathic Surgery

The orthodontic mechanics should be oriented toward positioning of the teeth solidly in the alveolar housing within each arch and specifically within each maxillary segment as indicated.[128,132,145,149,151] A goal is for the teeth to occlude together appropriately at the time of surgery and after finishing orthodontics. The preferred and anticipated occlusion is best confirmed via the periodic review of dental models (see Chapter 5).

From the beginning, the orthodontist and the surgeon make joint decisions concerning the treatment approach. One aspect that they will consider is whether the maxilla is

to remain in one piece or to be segmented; if segmentation is planned, they will determine at what locations this will occur. The decision for or against segmentation of the maxilla is driven by the baseline skeletal deformity and not for reasons of clinical expedience. A patient with a long face and a moderate or severe anterior open bite will generally have an excessive curve of Spee in the upper arch. Vertical steps in the maxillary arch exist that are coincident with the degree of dentoalveolar dysplasia. The more severe the vertical alveolar and dental step-offs, the more exaggerated the curve of Spee and the more obvious the need for segmental orthodontic mechanics and segmental surgical repositioning as part of the overall correction.

With maxillary surgical segmentation planned, the orthodontist's presurgical goal is to level the teeth independently within each segment. With a decision made to also defer arch expansion to the surgical phase (i.e., segmental osteotomies with widening), the orthodontic alignment of the teeth within each segment becomes the preoperative objective. Furthermore, there is no need or advantage for the orthodontist to create a space at the planned interdental osteotomy sites (i.e., between the lateral incisor and canine on each side or between the central incisors), because the roots are naturally divergent (see Chapter 17). The orthodontic preparation before surgery should incorporate the following basic concepts:

1. *The mandibular arch is leveled with the use of standard orthodontic mechanics before surgery.* A patient with a long face does not generally have a severely exaggerated curve of Spee in the lower arch. Therefore, complete leveling is generally carried out before surgery (with or without the need for extractions).
2. *The presence of an excessive reverse curve of Spee in the maxillary arch is an indication of a skeletal problem rather than a dental one.* Orthodontic leveling of the arch in segments (i.e., four incisors, the canine through the second molar on the left side, and the canine through the second molar on the right side) is generally consistent with the skeletal dysmorphology, and it is carried out before surgery. With the curve of Spee orthodontically leveled in each of the three components, segmental Le Fort I osteotomies will allow for the placement of the three individually leveled components over the orthodontically leveled mandibular arch to achieve the preferred occlusion at operation.
3. *The presence of a narrow maxilla is an indication of skeletal dysplasia rather than dental malposition*. Orthodontic tooth movement should remain consistent with the narrow skeletal width. Each posterior segment (i.e., the canine through the second molar on the left side and the canine through the second molar on the right side) should be independently orthodontically leveled. Segmentation of the downfractured maxilla will then adjust the arch width to the orthodontically leveled mandibular arch. If the posterior teeth have not been orthodontically positioned fully within the alveolar housing (e.g., if there is buccal tipping of molars), dental relapse after surgery with a return of the narrow maxillary arch width and the open-bite malocclusion is more likely to occur (see Chapter 17).

Basic Mandibular Arch Mechanics

- Routine orthodontic mechanics are used to level, align, close up spaces, and achieve appropriate arch form in the lower jaw.
- Extraction of a bicuspid in each quadrant (or of a single incisor, in rare cases) or interproximal reduction may be needed in the lower arch to uncrowd the dental crowns, to set the roots solidly in the alveolar housing, and to correct incisor inclination.

Basic Maxillary Arch Mechanics

- In the presence of vertical skeletal discrepancies, presurgical orthodontic stabilization may be achieved with sectional finishing wires that conform to the arch shape of each segment.
- If no vertical segmental skeletal discrepancies exist, then the maxillary arch is leveled with a continuous arch wire. Care is taken to prevent the forward flaring of the incisors. Posterior intrusion mechanics are also avoided, because they predispose the patient to late dental relapse.
- The advantage of bicuspid extractions to position the remaining roots solidly in alveolar housing should always be considered.
- In our experience, planning the interdental osteotomies between the canines and the first bicuspids is rarely preferred. Segmentation in this location is technically more time-consuming for the orthodontist (i.e., precise intercanine width orthodontic detailing becomes necessary), and it is more prone to dental injury and periodontal sequelae (i.e., pocketing and gingival recession) from the interdental osteotomy at surgery.
- Segmentation between the lateral incisors and the canines is generally preferred. When segmenting in this location, it is not necessary or advantageous for the orthodontist to create space at the planned osteotomy sites, because the roots are naturally divergent. The advantages of segmenting the maxilla in this location (i.e., between the lateral incisor and the canine) on each side and then parasagittally (i.e., down the hard palate) include the simplification of orthodontic mechanics, the prevention of periodontal sequelae, and the ability to positively influence incisor inclination without negatively affecting the vertical facial height.

Basic Surgical Approach

The surgical approach for the treatment of a classic long face is best carried out through a combination of osteotomies, including Le Fort I osteotomy in segments, bilateral sagittal split ramus osteotomies, and oblique osteotomy of the

chin (Figs. 21-1 through 21-10).* Management of the anterior neck soft tissues—including cervical soft-tissue flap elevation, neck defatting, and vertical platysmal muscle plication—can also be carried out, if desired (see Chapter 40). The removal of the impacted wisdom teeth is frequently recommended for long-term dental health. If this is planned, it can be accomplished at the same time (see Chapters 15 and 16).

If septoplasty, inferior turbinate reductions, or both are required to improve nasal airflow, then the procedures are carried out simultaneously (see Chapter 10).[108-110] For the patient with a long face growth pattern, an important aspect of nasal aesthetics and airway management is to also simultaneously lower and recontour the floor of the nose, the pyriform rims, and the anterior nasal spine region. In 1985, Ellis used lateral cephalometric radiographs to study the specific regions of vertical maxillary excess in mature individuals (i.e., >17 years old) with long faces.[36] In the study patients, the linear distance between the incisal edge and the floor of the nose (i.e., the upper incisal edge to the anterior nasal spine) was found to be significantly increased. In other words, the anterior floor of the nose was excessively high, which at least partially accounted for the diminished nasal aperture opening. Ellis recognized that, when a standard Le Fort I maxillary intrusion is carried out to decrease the gummy smile, the elimination of any excessive alveolar bone in the area of the pyriform fossa should also be considered. In these cases, I recommend that the floor of the nose and the anterior nasal spine regions be recontoured to lower the floor. When this is done, the nasal airway is improved, and the "bunching" of the nasal and paranasal soft tissues is avoided. I agree with Ellis and Westermark that the popular use of an alar cinch stitch is generally carried out as a soft-tissue attempt to solve a skeletal problem.[36,154] Although the alar cinch stitch will have effects on the nasal base width, it will also have secondary effects on the nasolabial angle and the nasal tip projection that may not be positive.[118] In addition, if the cinch stitch is completed without fully correcting the skeletal aspects (i.e., lowering the nasal floor and recontouring the anterior nasal spine), suboptimal nasal aesthetics and further airway obstruction are likely.[55,92,144]

One of the indications for the segmenting of the maxilla is to correct transverse dental width deficiencies.[7,9,10,22,56,77,79,129,130] In the past, problems of stability with transverse expansion have led some clinicians to recommend a two-stage surgical approach to maxillary reconstruction for a long face growth pattern. The first stage involves surgically assisted rapid palatal expansion to widen the maxilla during the presurgical orthodontic phase of treatment. Approximately 6 to 18 months later, a Le Fort I down-fracture osteotomy is carried out as part of the definitive orthognathic correction to reposition the maxilla vertically and horizontally, as indicated. As discussed in Chapter 17, I recommend a single-stage orthognathic approach in most cases. This includes a Le Fort I down-fracture osteotomy with segmentation to simultaneously correct the curve of Spee; any transverse discrepancy; vertical dysplasia; horizontal dysplasia; canting (i.e., roll orientation); any midline discrepancies (i.e. yaw orientation); and A-point to B-point discrepancies (i.e., pitch orientation).

NOTE: The literature does not confirm a better outcome with a two-stage surgical approach to maxillary deficiency in individuals with long faces.[30] The potential for increased morbidity as well as the increased time and financial costs of a two-stage surgical approach cannot be justified in most cases (see Chapter 17).

Immediate Presurgical Planning

The immediate presurgical planning takes into account collaborative clinical efforts among the orthodontist, the surgeon, the patient, and the family that will have been ongoing since the initial consultation visits.* Depending on the condition of the dentition and the supporting structures, the restorative dentist and the periodontist may also have been intricately involved.

The patient's specific medical and dental records are reviewed, including radiographs (e.g. computed tomography scans; panoramic, lateral, and posteroanterior cephalometric images and periapical radiographs); facial and occlusal photographs; and dental models. Immediate presurgical records (i.e., within 2 to 6 weeks of surgery) will most importantly include facial measurements taken during a direct visual examination of the patient; alginate impressions of the maxilla and the mandible; centric relation bite recording; and face-bow registration.

First, broad-stroke decisions are made regarding the preferred vector changes for each jaw; only then are the precise millimeter distances and angular changes required in each jaw to achieve the desired end result determined (see Chapter 12). Analytic model planning is carried out on the articulated dental casts with use of the Erickson Model Table. Splints are constructed to assist with the achievement of the precise occlusion and preferred facial aesthetics in the operating room (see Chapters 13 and 14).

The basic vector changes required in the maxilla to correct a long face growth pattern frequently include maxillary horizontal advancement; the shortening of the anterior (vertical) height; transverse widening; and the correction of the curve of Spee to match the mandibular arch. Basic vector changes often incorporate counterclockwise rotation of the maxillary plane (i.e., pitch orientation) as needed to achieve a preferred A-point to B-point

Text continued on p. 801

*References 6, 11-21, 29, 32, 38, 40, 41, 48, 57, 58, 68, 72, 73, 83, 84, 100, 101, 118-121, 125, 138, 148, 152, 17, 102

*References 39, 43, 46, 60, 98, 107, 116, 122, 127, 141, 143, 147, 153, 156

• **Figure 21-1** A 15-year-old girl arrived with her parents for surgical evaluation. Her history and physical examination confirmed nasal obstruction, heavy snoring, lip incompetence, difficulty chewing, and frequent speech articulation errors. The facial aesthetic concerns included an awareness of a weak chin, a gummy smile, and the impression of having a large nose. The patient had been under an orthodontist's care since the mixed dentition. Rapid palatal expansion and full bracketing with attempted closure of the anterior open bite were not successful. Further evaluations were carried out by a speech pathologist and an otolaryngologist, and a fresh look was taken at the patient's orthodontic needs. Orthodontic decompensation in combination with orthognathic and intranasal surgery was chosen. The simultaneous procedures carried out included Le Fort I osteotomy in segments (horizontal advancement, and vertical shortening, counterclockwise rotation, arch expansion, and the correction of the curve of Spee); sagittal split ramus osteotomies (horizontal advancement and counterclockwise rotation); osseous genioplasty (vertical shortening and horizontal advancement); and septoplasty, inferior turbinate reduction, and nasal floor recontouring. **A,** Frontal views in repose before and after reconstruction. **B,** Frontal views with smile before and after reconstruction.

• **Figure 21-1, cont'd C,** Oblique facial views before and after reconstruction. **D,** Profile views before and after reconstruction.

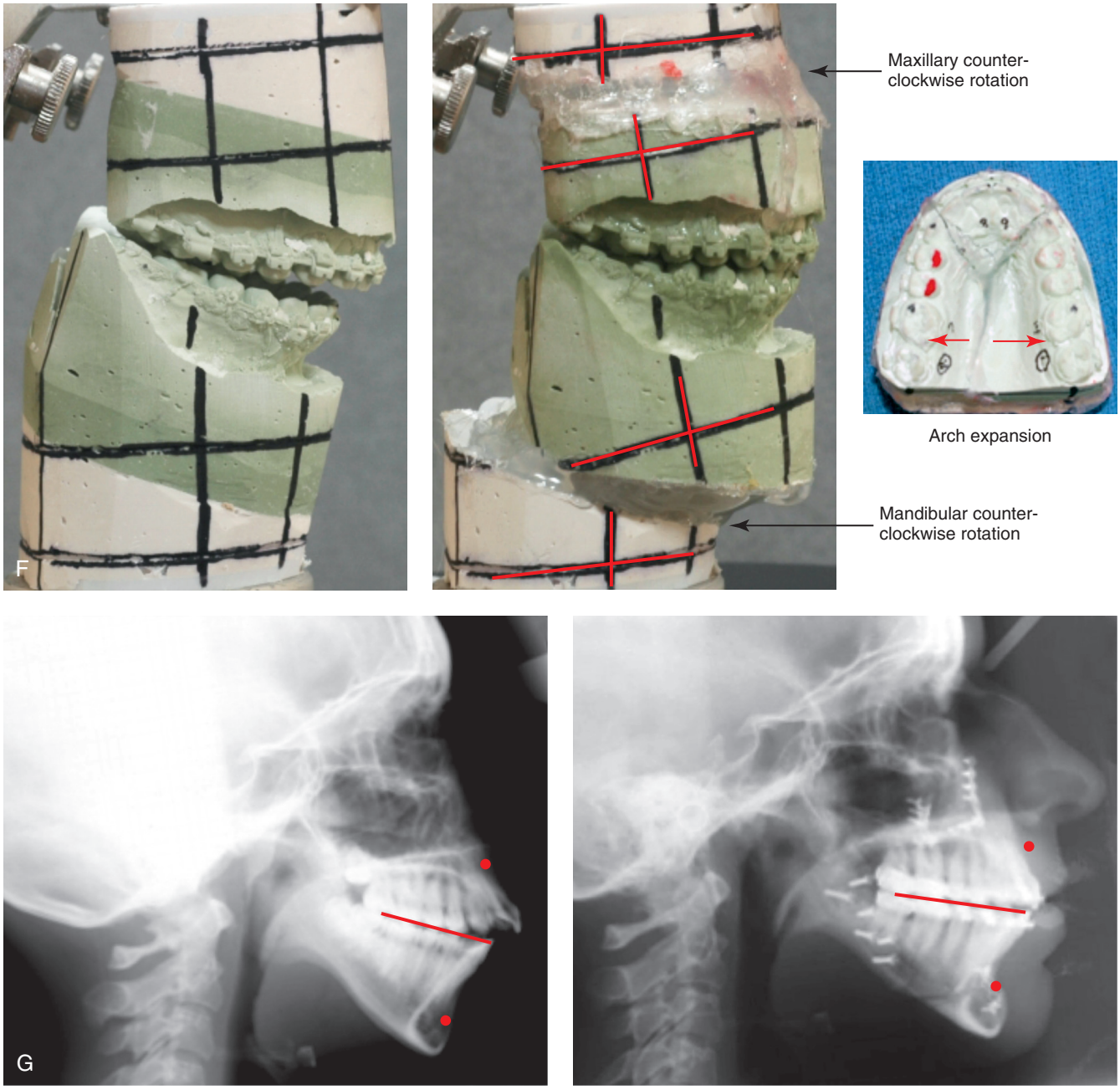
Continued

Prior to retreatment

Pre surgery

After treatment

• **Figure 21-1, cont'd E,** Occlusal views before retreatment, with orthodontic decompensation in progress, and after the completion of treatment.



• **Figure 21-1, cont'd** **F,** Articulated dental casts that indicate analytic model planning. **G,** Lateral cephalometric radiographs before and after surgery.

©Elsevier Inc.
Orthognathic Surgery: J.C. Posnick

• **Figure 21-2** A 15-year-old girl arrived with her parents for surgical evaluation. Her history and physical examination confirmed lifelong nasal obstruction, heavy snoring, and lip incompetence. The facial aesthetic concerns included an awareness of a weak chin, a gummy smile, wide lip separation, and a deep lip curl. The patient had been under orthodontic observation since the mixed dentition. Further evaluations were carried out by a speech pathologist and an otolaryngologist. Orthodontic decompensation in combination with orthognathic and intranasal surgery was chosen. The simultaneous procedures carried out included Le Fort I osteotomy in segments (horizontal advancement, vertical shortening, arch expansion, the correction of the curve of Spee, and counterclockwise rotation); sagittal split ramus osteotomies (horizontal advancement and counterclockwise rotation); osseous genioplasty (vertical shortening and horizontal advancement); and septoplasty, inferior turbinate reduction, and nasal floor recontouring. **A,** Frontal views in repose before and after reconstruction. **B,** Frontal views with smile before and after reconstruction.

©Elsevier Inc.
Orthognathic Surgery; J.C. Posnick

• **Figure 21-2, cont'd** **C,** Profile facial views before and after reconstruction. **D,** Oblique views before and after reconstruction.

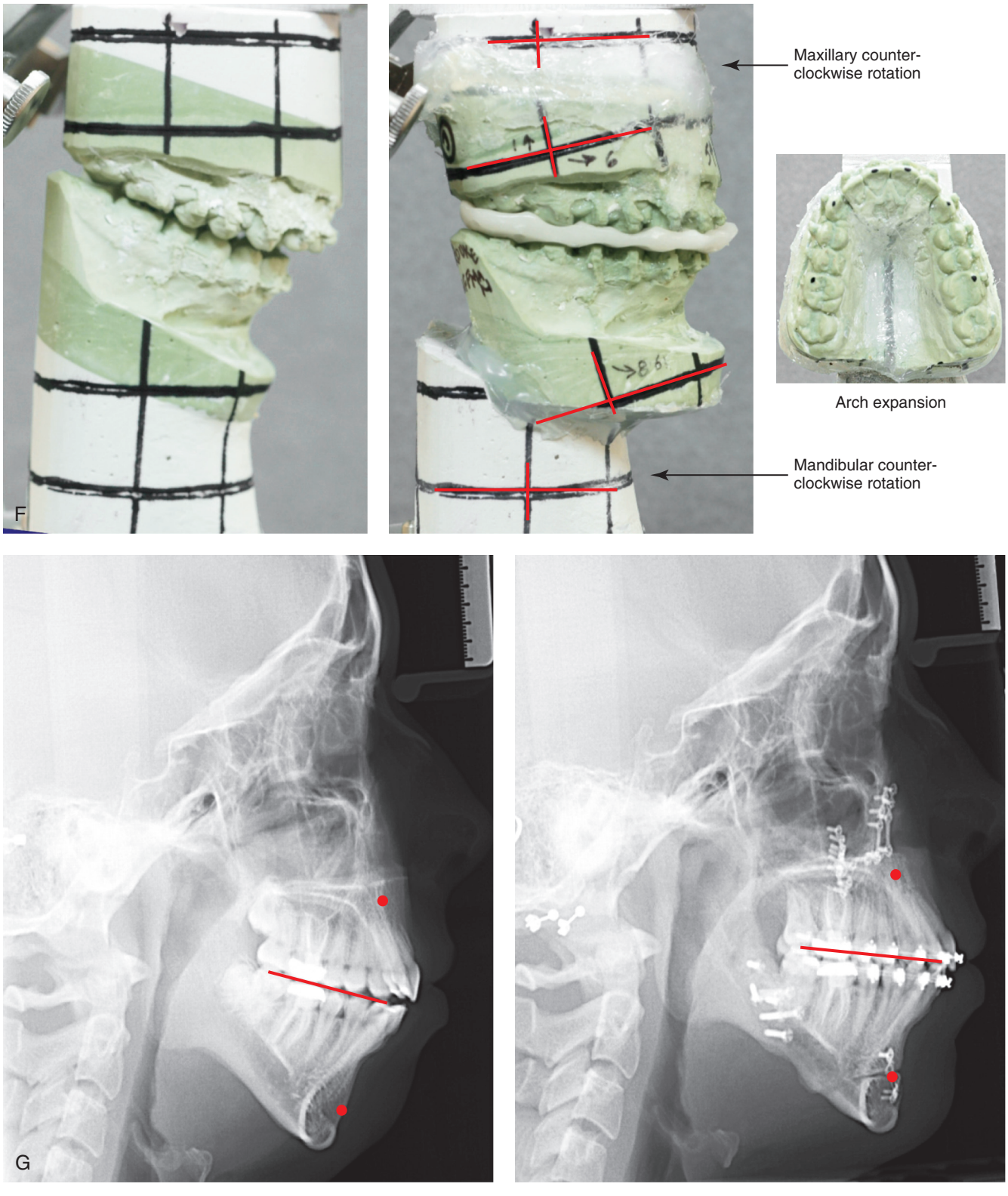
Continued

Prior to treatment

Pre surgery

After treatment

• **Figure 21-2, cont'd E,** Occlusal views before treatment, with orthodontic decompensation in progress, and after the completion of treatment.



• **Figure 21-2, cont'd** **F,** Articulated dental casts that indicate analytic model planning. **G,** Lateral cephalometric radiographs before and after surgery.

• **Figure 21-3** A 15-year-old girl arrived with her parents for surgical evaluation. Her history and physical examination confirmed lifelong nasal obstruction and lip incompetence. The facial aesthetic concerns included an awareness of a weak chin, a gummy smile, and wide lip separation. The patient had been under an orthodontist's care since the mixed dentition and underwent a full course of treatment. Further evaluations were carried out by a speech pathologist and an otolaryngologist, and a fresh look was taken at the patient's orthodontic needs. Orthodontic decompensation in combination with orthognathic and intranasal surgery was chosen. The simultaneous procedures carried out included Le Fort I osteotomy in segments (horizontal advancement, vertical); sagittal split ramus osteotomies (horizontal advancement and counterclockwise rotation); osseous genioplasty (vertical shortening and horizontal advancement); and septoplasty, inferior turbinate reduction, and nasal floor recontouring. **A,** Frontal views in repose before and after reconstruction. **B,** Frontal views with smile before and after reconstruction.

©Elsevier Inc.
Orthognathic Surgery; J.C. Posnick

• **Figure 21-3, cont'd** **C,** Oblique facial views before and after reconstruction. **D,** Profile views before and after reconstruction.

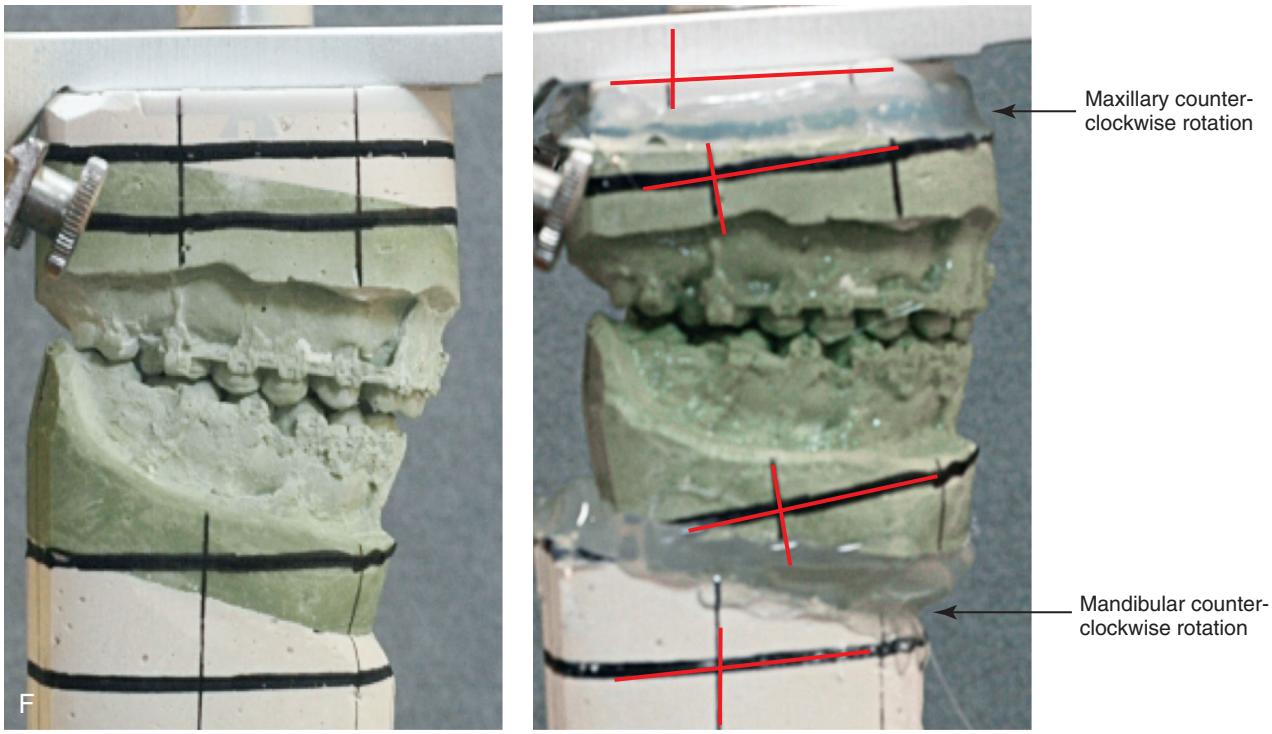
Continued

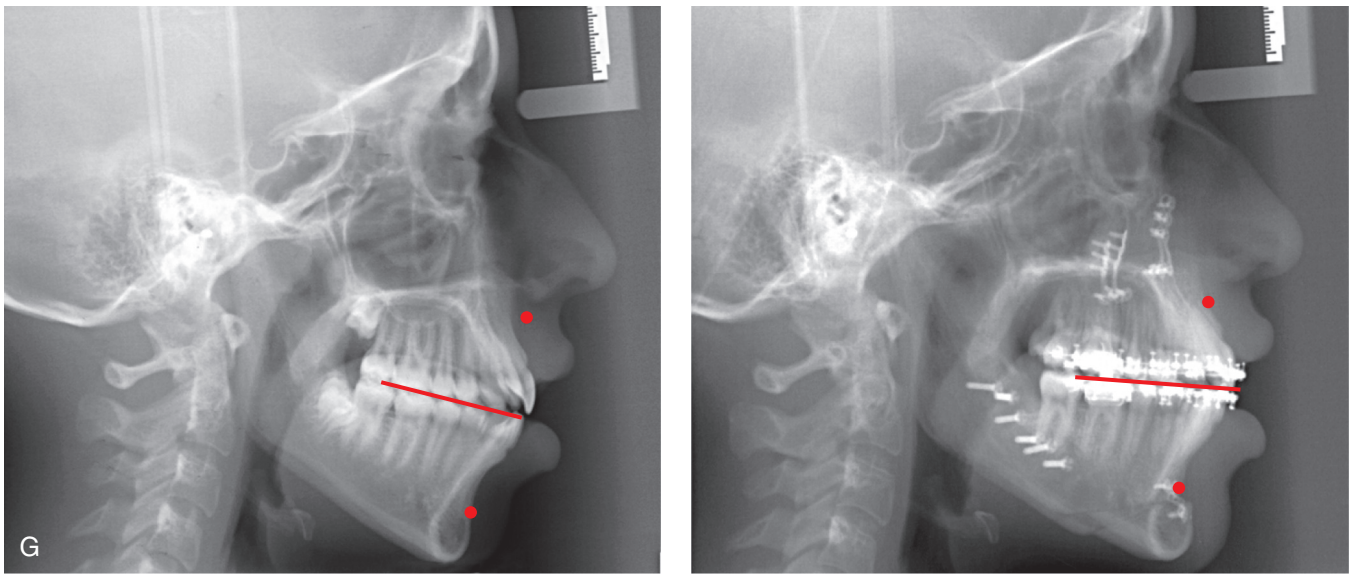
Prior to retreatment

Pre surgery

After treatment

• **Figure 21-3, cont'd E,** Occlusal views before retreatment, with orthodontic decompensation in progress, and after the completion of treatment.





• **Figure 21-3, cont'd** **F,** Articulated dental casts that indicate analytic model planning. **G,** Lateral cephalometric radiographs before and after surgery.

©Elsevier Inc.
Orthognathic Surgery; J.C. Posnick

• **Figure 21-4** A 13-year-old girl arrived with her parents for surgical evaluation. Her history and physical examination confirmed lifelong nasal obstruction, heavy snoring, lip incompetence, and persistent gingivitis despite good home care. The facial aesthetic concerns included an awareness of a weak chin, a gummy smile, and severe lip strain. Further evaluations were carried out by a speech pathologist and an otolaryngologist, and a fresh look was taken at the patient's orthodontic needs. Orthodontic decompensation in combination with orthognathic and intranasal surgery was chosen. The simultaneous procedures carried out included Le Fort I osteotomy (horizontal advancement, and vertical shortening); sagittal split ramus osteotomies (horizontal advancement and counterclockwise rotation); osseous genioplasty (vertical shortening and horizontal advancement); and septoplasty, inferior turbinate reduction, and nasal floor recontouring. **A,** Frontal views in repose before and after reconstruction. **B,** Frontal views with smile before and after reconstruction.

©Elsevier Inc.
Orthognathic Surgery; J.C. Posnick

• **Figure 21-4, cont'd** **C,** Oblique facial views before and after reconstruction. **D,** Profile views before and after reconstruction.

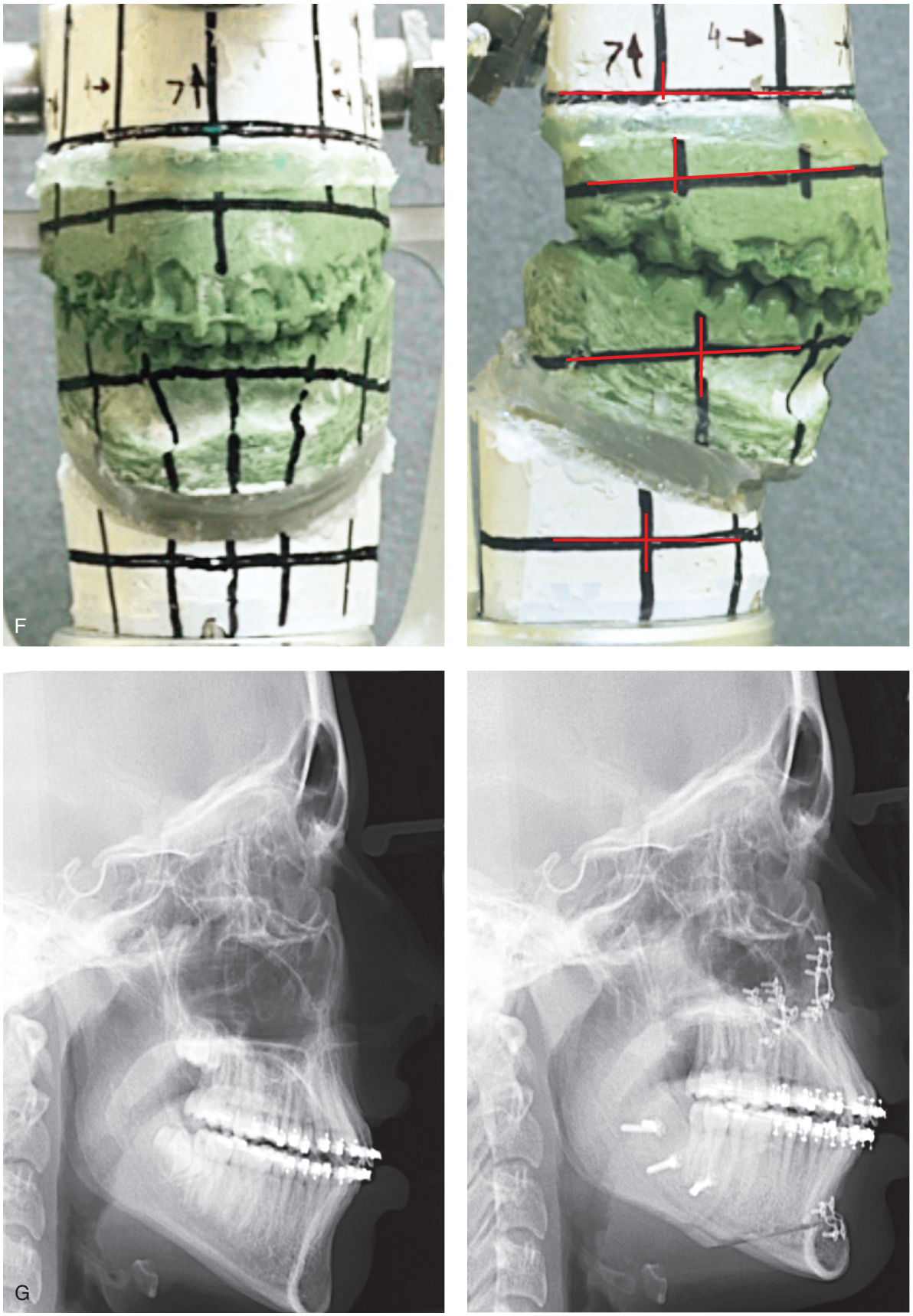
Continued

Prior to treatment

Pre surgery

After treatment

• **Figure 21-4, cont'd E,** Occlusal views before treatment, with orthodontic decompensation in progress, and after the completion of treatment.



• **Figure 21-4, cont'd** **F,** Articulated dental casts that indicate analytic model planning. **G,** Lateral cephalometric radiographs before and after surgery.

• **Figure 21-5** A 21-year-old woman arrived for surgical evaluation. Her history and physical examination confirmed lifelong nasal obstruction, lip incompetence, difficulty chewing, and frequent speech articulation errors. The facial aesthetic concerns included an awareness of a weak chin, a gummy smile, lip strain, and the impression of having a larger nose. The patient had been under an orthodontist's care since the mixed dentition and through the high school years. Growth modification attempts followed by rapid palatal expansion and full bracketing with four bicuspid extractions were unsuccessful to close the anterior open bite. Early periodontal and dental deterioration were evident. Further evaluations were carried out by a speech pathologist, an otolaryngologist, and a periodontist, and a fresh look was taken at the patient's orthodontic needs. Orthodontic decompensation in combination with orthognathic and intranasal surgery was chosen. The simultaneous procedures carried out included Le Fort I osteotomy in segments (horizontal advancement, vertical shortening, counterclockwise rotation, arch expansion, and the correction of the curve of Spee); sagittal split ramus osteotomies (horizontal advancement and counterclockwise rotation); osseous genioplasty (vertical shortening and horizontal advancement); and septoplasty, inferior turbinate reduction, and nasal floor recontouring. **A,** Frontal views in repose before and after reconstruction. **B,** Frontal views with smile before and after reconstruction.

©Elsevier Inc.
Orthognathic Surgery; J.C. Posnick

• **Figure 21-5, cont'd** **C,** Oblique facial views before and after reconstruction. **D,** Profile views before and after reconstruction.

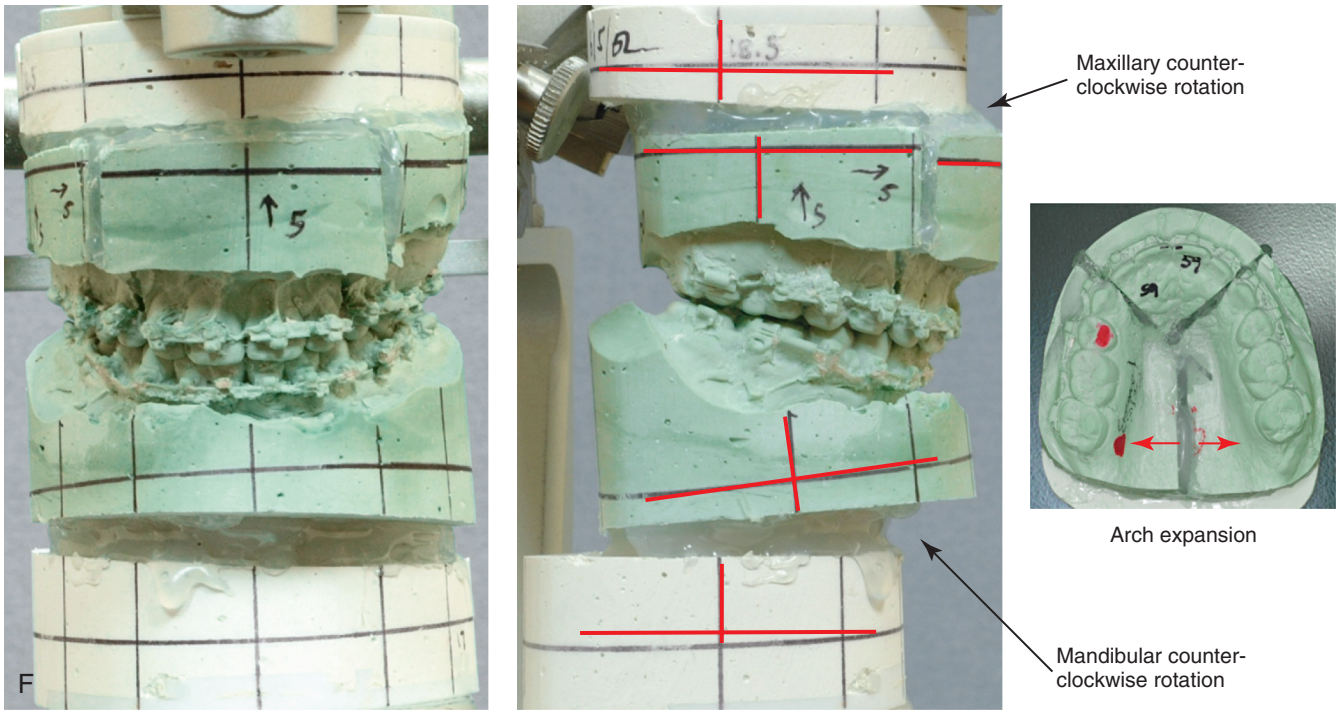
Continued

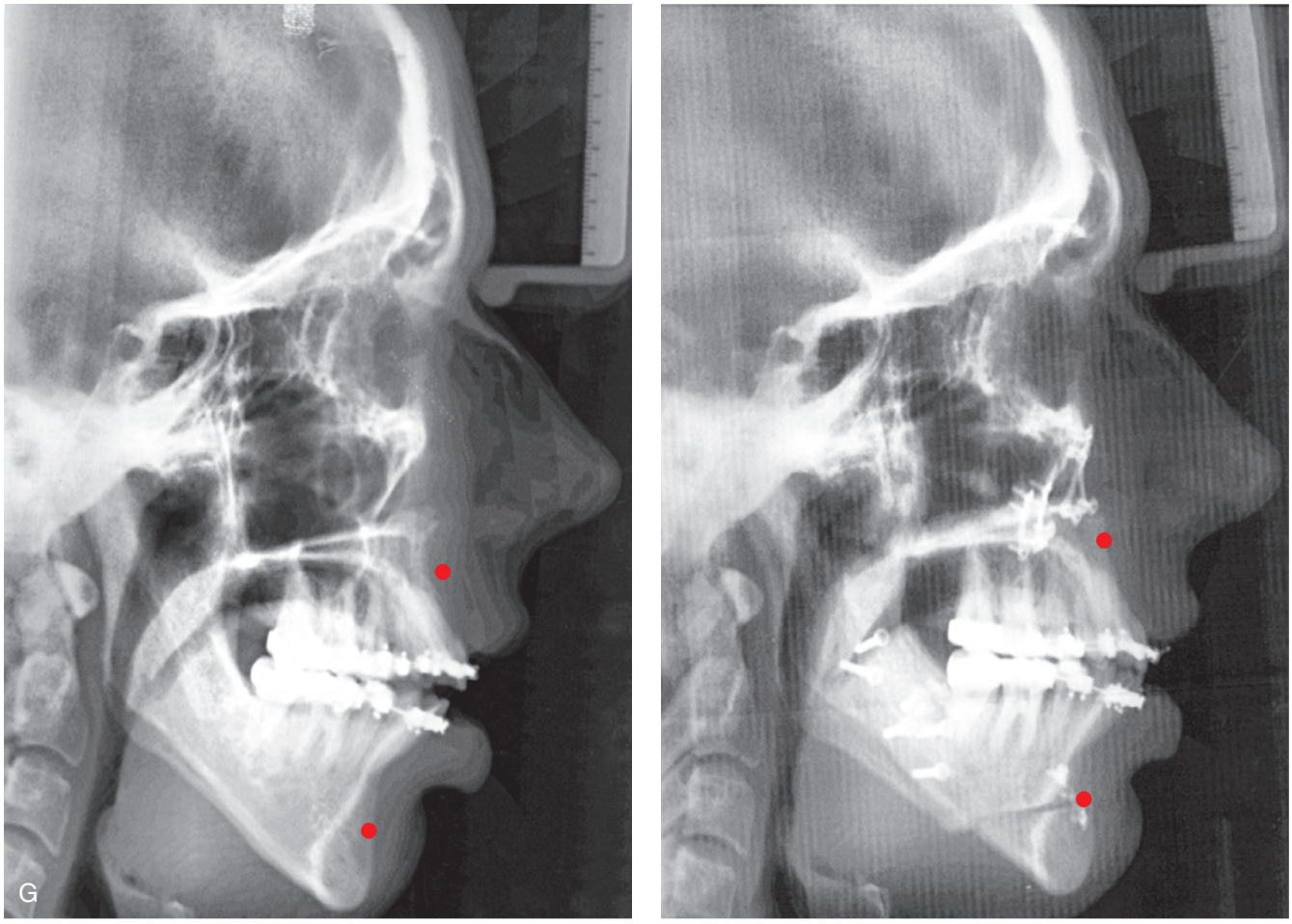
4 bicuspid extractions and dental compensation carried out earlier in life

Pre surgery

After treatment

• **Figure 21-5, cont'd E,** Occlusal views before retreatment, with orthodontic decompensation in progress, and after the completion of treatment.





• **Figure 21-5, cont'd** **F,** Articulated dental casts that indicate analytic model planning. **G,** Lateral cephalometric radiographs before and after surgery.

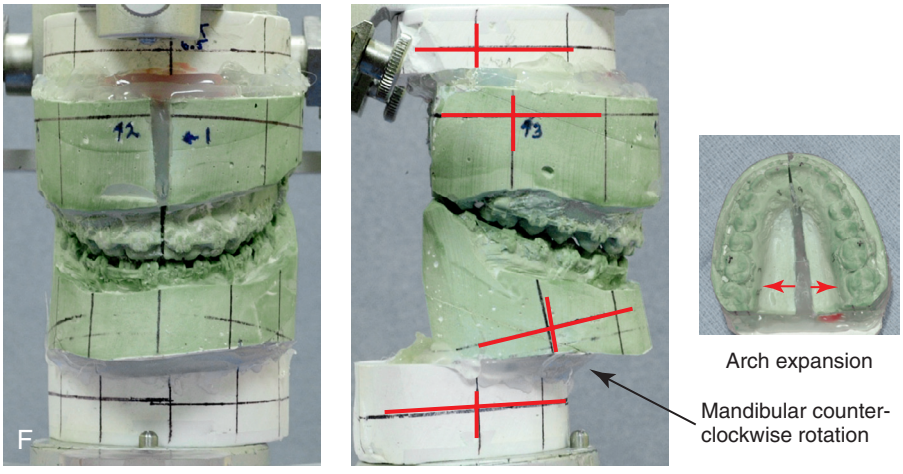
• **Figure 21-6** A 15-year-old girl arrived with her parents for surgical evaluation. Her history and physical examination confirmed lifelong nasal obstruction, heavy snoring, lip incompetence, difficulty chewing, and frequent speech articulation errors. The facial aesthetic concerns included an awareness of a weak chin, a gummy smile, and difficulty holding the lips together. The patient had been under an orthodontist's care since the mixed dentition. Rapid palatal expansion and full bracketing with attempted closure of the anterior open bite were unsuccessful. Further evaluations were carried out by a speech pathologist and an otolaryngologist, and a fresh look was taken at the patient's orthodontic needs. Orthodontic decompensation in combination with orthognathic and intranasal surgery was chosen. The simultaneous procedures carried out included Le Fort I osteotomy in segments (horizontal advancement, vertical shortening, counterclockwise rotation, and arch expansion); sagittal split ramus osteotomies (horizontal advancement and counterclockwise rotation); osseous genioplasty (vertical shortening and horizontal advancement); and septoplasty, inferior turbinate reduction, and nasal floor recontouring. **A,** Frontal views in repose before and after reconstruction. **B,** Frontal views with smile before and after reconstruction.

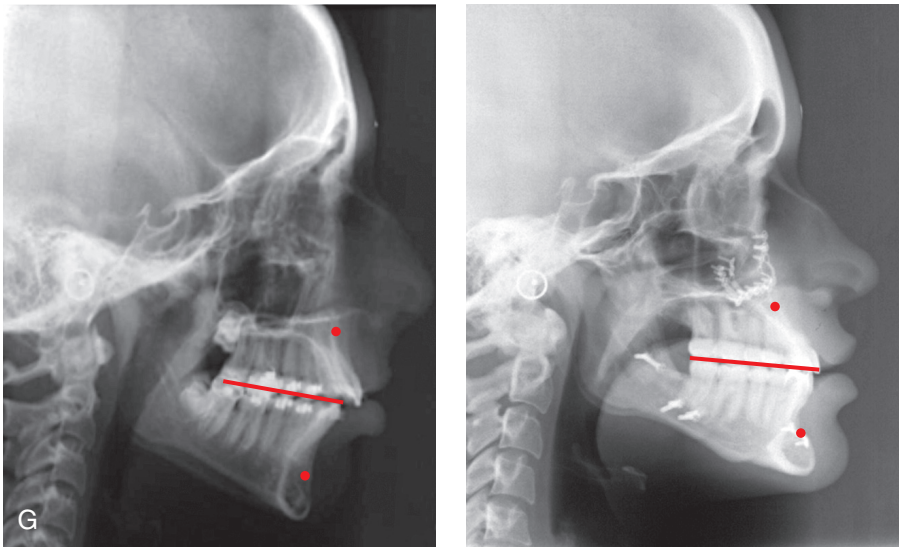
• **Figure 21-6, cont'd** **C,** Oblique facial views before and after reconstruction. **D,** Profile views before and after reconstruction.

Continued

Pre surgery

After treatment





• **Figure 21-6, cont'd** **E,** Occlusal views with orthodontic decompensation in progress and after the completion of treatment. **F,** Articulated dental casts that indicate analytic model planning. **G,** Lateral cephalometric radiographs before and after surgery.

©Elsevier Inc.
Orthognathic Surgery; J.C. Posnick

• **Figure 21-7** A high-school girl arrived with her parents for evaluation. She refused an orthognathic approach at the time but then returned during her college years to request correction. Her history and physical examination confirmed nasal obstruction, lip incompetence, and difficulty chewing. The facial aesthetic concerns included an awareness of a weak chin, a gummy smile, lip strain, and the impression of having a larger nose. The patient had been under an orthodontist's care since the mixed dentition. Rapid palatal expansion and full bracketing with attempted closure of the anterior open bite were not successful. Further evaluations were carried out by a speech pathologist and an otolaryngologist, and a fresh look was taken at the patient's orthodontic needs. Orthodontic decompensation that included four bicuspid extractions in combination with orthognathic and intranasal surgery was chosen. The simultaneous procedures carried out included Le Fort I osteotomy in segments (horizontal advancement, vertical shortening, and arch expansion); sagittal split ramus osteotomies (horizontal advancement); osseous genioplasty (vertical shortening and horizontal advancement); and septoplasty, inferior turbinate reduction, and nasal floor recontouring. **A,** Frontal views in repose before and after reconstruction. **B,** Frontal views with smile before and after reconstruction.

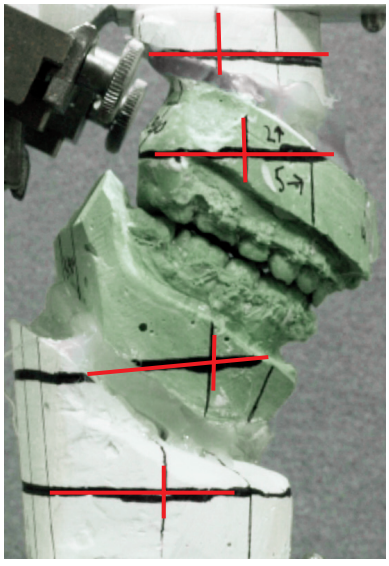
Continued

©Elsevier Inc.
Orthognathic Surgery; J.C. Posnick

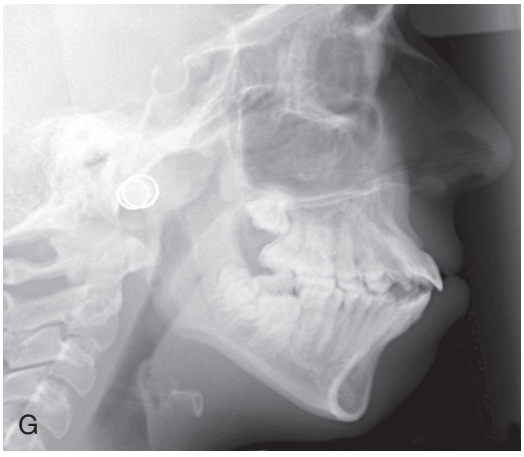
• **Figure 21-7, cont'd** **C,** Oblique facial views before and after reconstruction. **D,** Profile views before and after reconstruction.

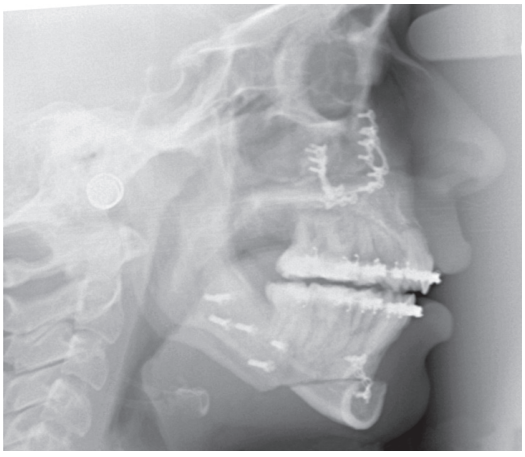
Pre surgery just prior to 4 bicuspid extractions

After treatment with four bicuspid extractions



Arch expansion





• **Figure 21-7, cont'd** **E,** Occlusal views with orthodontic decompensation in progress and then after the completion of treatment. **F,** Articulated dental casts that indicate analytic model planning. **G,** Lateral cephalometric radiographs before and after surgery.

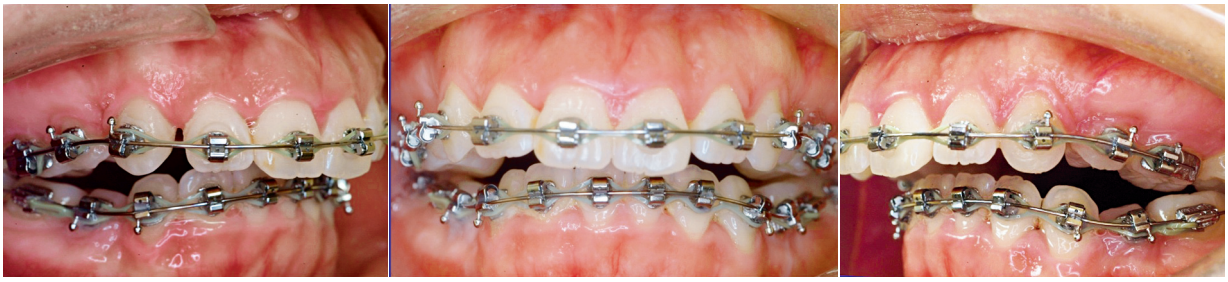
• **Figure 21-8** A high-school girl arrived with her parents for evaluation. Her history and physical examination confirm a long face growth pattern with nasal obstruction, lip incompetence, and a gummy smile. Orthodontic decompensation including four bicuspid extractions in combination with orthognathic and intranasal surgery was chosen. The simultaneous procedures carried out included Le Fort I osteotomy in segments (horizontal advancement, vertical shortening, and arch expansion); sagittal split ramus osteotomies (horizontal advancement); osseous genioplasty (vertical shortening and horizontal advancement); and septoplasty, inferior turbinate reduction, and nasal floor recontouring. **A,** Frontal views in repose before and four years after reconstruction. **B,** Frontal views with smile before and after reconstruction. A degree of gummy smile remains but without lip strain during closure and with satisfactory lip separation in repose.

©Elsevier Inc.
Orthognathic Surgery; J.C. Posnick

• **Figure 21-8, cont'd** **C,** Oblique facial views before and after reconstruction. **D,** Profile views before and after reconstruction.

Continued

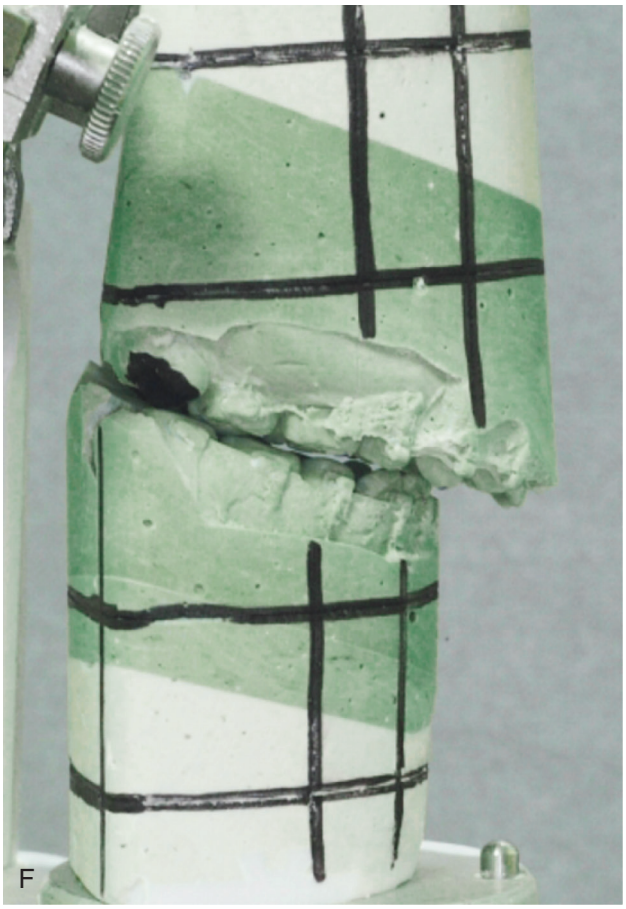
Prior to treatment

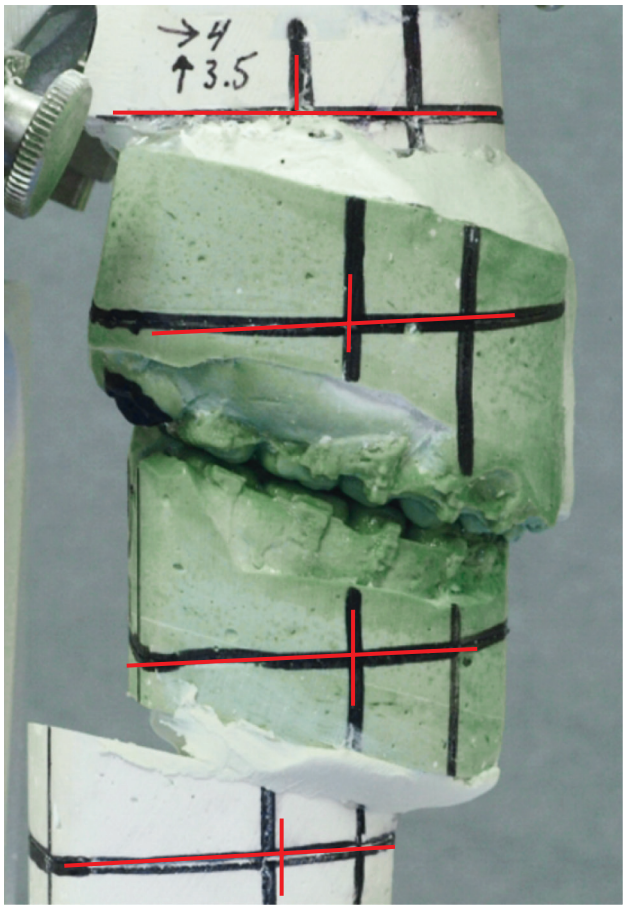


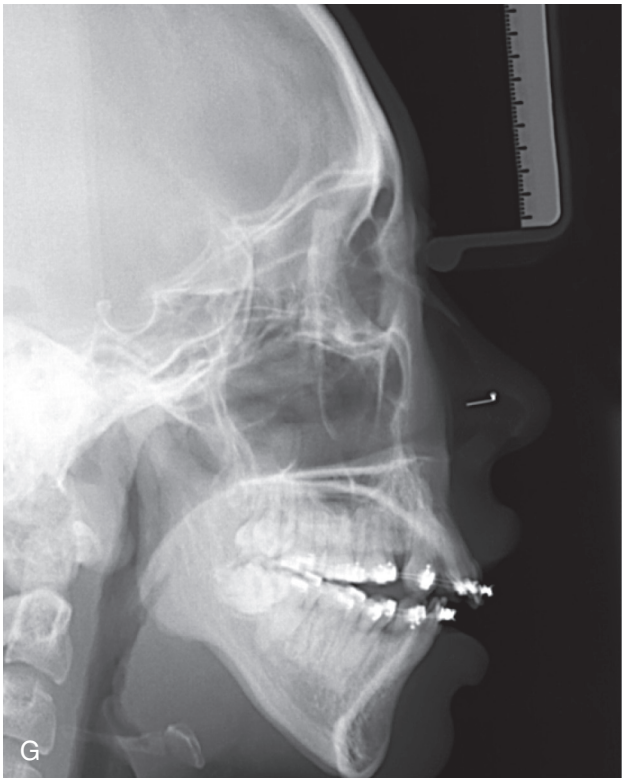
Pre surgery with four bicuspid extractions

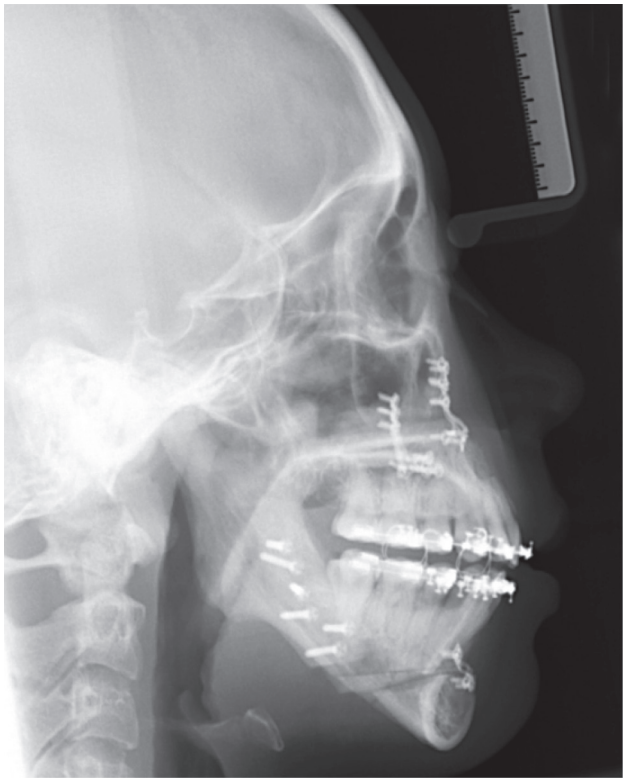
After treatment

• **Figure 21-8, cont'd E,** Occlusal views before treatment, with orthodontic decompensation including four bicuspid extractions in progress, and after the completion of treatment.









• **Figure 21-8, cont'd** **F,** Articulated dental casts that indicate analytic model planning. **G,** Lateral cephalometric radiographs before and after surgery.

©Elsevier Inc.
Orthognathic Surgery; J.C. Posnick

• **Figure 21-9** A young woman arrived for surgical evaluation. Her history and physical examination confirmed lifelong nasal obstruction, lip incompetence, difficulty chewing, and frequent speech articulation errors. The facial aesthetic concerns included an awareness of a weak chin, a gummy smile, lip strain, and the impression of having a larger nose. The patient had experienced secondary occlusal trauma with a need for root canal therapy and crowns on multiple posterior teeth. Further investigations were carried out by a speech pathologist and an otolaryngologist, and a fresh look was taken at the patient's requirements for the achievement of a satisfactory occlusion. Orthodontic decompensation including four bicuspid extractions in combination with orthognathic and intranasal surgery was chosen. The simultaneous procedures carried out included Le Fort I osteotomy (horizontal advancement, vertical shortening, and counterclockwise rotation); sagittal split ramus osteotomies (horizontal advancement and counterclockwise rotation); osseous genioplasty (vertical shortening and horizontal advancement); and septoplasty, inferior turbinate reduction, and nasal floor recontouring. **A,** Frontal views in repose before and after reconstruction. **B,** Frontal views with smile before and after reconstruction.

• **Figure 21-9, cont'd** **C,** Oblique facial views before and after reconstruction. **D,** Profile views before and after reconstruction.

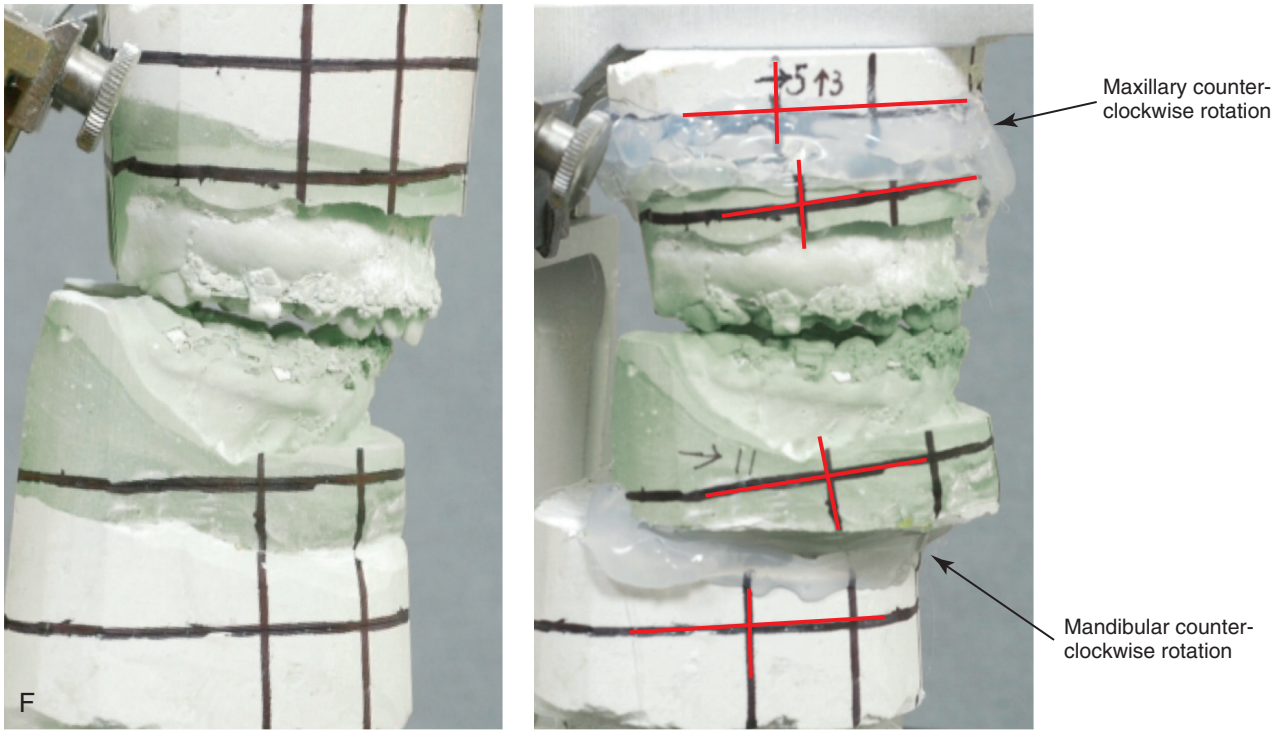
Continued

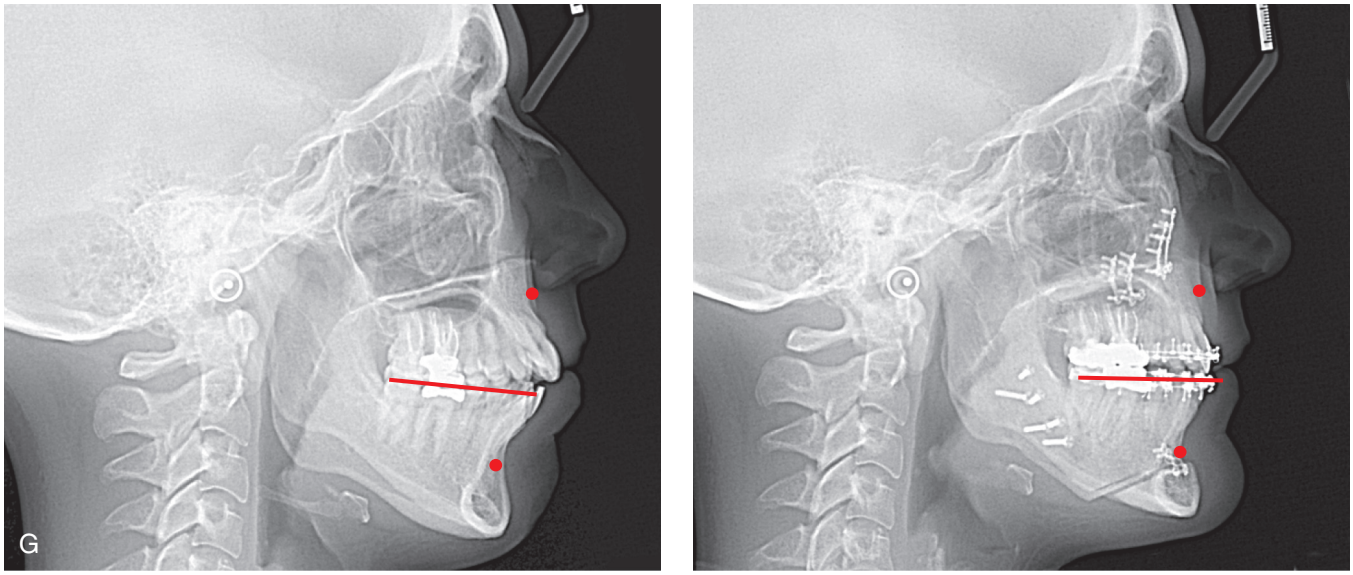
Prior to treatment

Orthodontics in progress with 4 bicuspid extractions

After treatment

• **Figure 21-9, cont'd E,** Occlusal views before retreatment, with orthodontics in progress including four bicuspid extractions in progress, and after the completion of treatment.





• **Figure 21-9, cont'd** **F,** Articulated dental casts that indicate analytic model planning. **G,** Lateral cephalometric radiographs before and after surgery.

©Elsevier Inc.
Orthognathic Surgery; J.C. Posnick

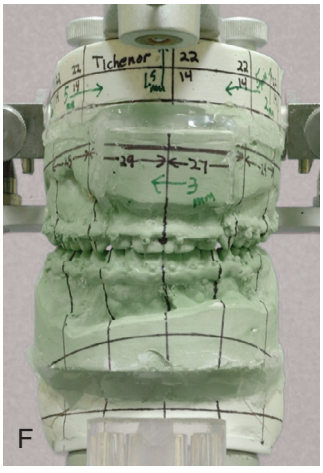
• **Figure 21-10** A 17-year-old boy arrived with his parents for surgical evaluation. His history and physical examination confirmed lifelong nasal obstruction, heavy snoring, difficulty chewing, and frequent speech articulation errors. The facial aesthetic concerns include an awareness of a gummy smile, lip strain, and a weak profile. Earlier during his life, he had undergone orthodontics that included rapid palatal expansion and full bracketing, without success. An anterior open bite and constricted arch-width malocclusion remained. Further evaluations were carried out by a speech pathologist and an otolaryngologist, and a fresh look was taken at the patient's orthodontic needs. Orthodontic decompensation in combination with orthognathic and intranasal surgery was chosen. The simultaneous procedures carried out included Le Fort I osteotomy in segments (horizontal advancement, vertical shortening, counterclockwise rotation, arch expansion, and the correction of the curve of Spee); sagittal split ramus osteotomies (horizontal advancement and counterclockwise rotation); osseous genioplasty (vertical shortening and horizontal advancement); and septoplasty, inferior turbinate reduction, and nasal floor recontouring. **A,** Frontal views in repose before and after reconstruction. **B,** Frontal views with smile before and after reconstruction.

• **Figure 21-10, cont'd C,** Oblique facial views before and after reconstruction. **D,** Profile views before and after reconstruction.

Continued

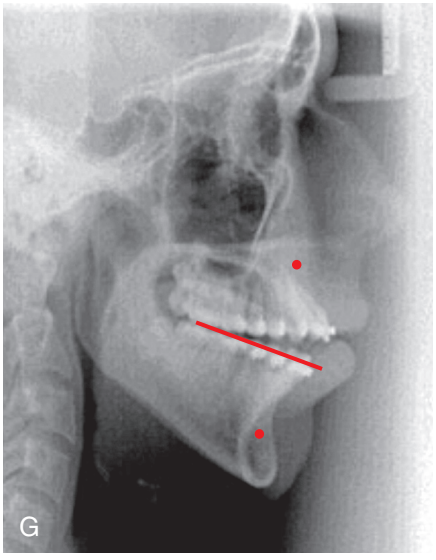
Pre surgery

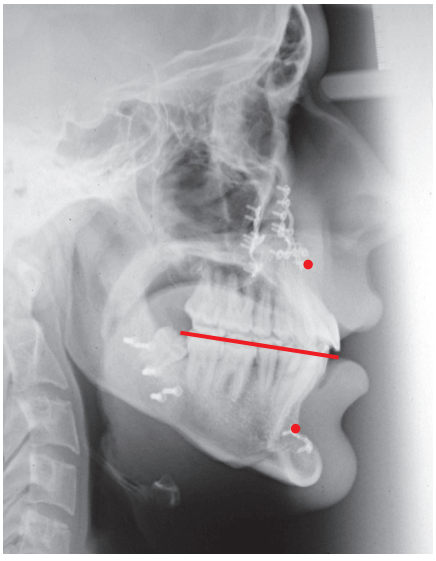
After treatment



Arch expansion

Mandibular counter-clockwise rotation





• **Figure 21-10, cont'd E,** Occlusal views with orthodontic decompensation in progress and after the completion of treatment. **F,** Articulated dental casts that indicate analytic model planning. **G,** Lateral cephalometric radiographs before and after surgery.

relationship and pogonion projection when viewing the patient's profile in the natural head position. The immediate presurgical models will confirm details of the planned segmentation of the maxilla and the need for any occlusal equilibration to achieve the preferred occlusion in the operating room.

Linear (millimeter) and angular (degree) decisions regarding the preferred repositioning of the maxilla specifically incorporate the following:

- The desired vertical positioning of the maxillary incisor crown
- The desired horizontal positioning of the maxillary incisor crown
- The need to reposition the maxillary dental midline to match the facial midline (i.e., yaw orientation)
- The need to alter or correct any maxillary cant (i.e., roll orientation)
- The need to vertically adjust the posterior maxillary height to establish a preferred A-point to B-point relationship of the jaws (i.e., pitch orientation); counterclockwise rotation of the maxillary plane is a frequent maneuver
- The need to or benefit of segmenting the maxilla to alter the arch width or the curve of Spee

Immediate presurgical planning will confirm the anticipated functional and aesthetic advantages of sagittal split ramus osteotomies to reposition the mandible into occlusion with the new maxillary position versus accepting the limitations of mandibular autorotation (i.e., one-jaw surgery). The baseline anterior projection of the maxilla in a patient with a long face is generally deficient and requires horizontal advancement at the level of the maxillary incisors. Horizontal advancement in combination with vertical intrusion and pyriform rim recontouring will limit the undesirable widening of the nose that would otherwise occur with a "straight-up" intrusion of the maxilla and autorotation of the mandible. *Simultaneous sagittal split ramus osteotomies of the mandible (i.e., two-jaw surgery) are required to achieve these objectives in almost all cases.*[118-121] It is also likely that an oblique osteotomy of the chin with vertical shortening and limited horizontal advancement will be advantageous.

The clinician should confirm or refute a lifelong history of chronic obstructive nasal breathing in the patient (see Chapter 10). During the physical examination, intranasal findings that are consistent with increased nasal airway resistance are likely to include septal deviations and inferior turbinate hypertrophy. If the history and physical findings warrant, then septoplasty and inferior turbinate reduction should be carried out to improve nasal breathing. The baseline presence of excess vertical height in the anterior maxilla (i.e., a high nasal floor) with lip incompetence and a constricted nasal width (i.e., a tight nasal aperture) is also typical and will benefit from midface intrusion, arch expansion through segmental osteotomies, and horizontal advancement. Recontouring of the nasal floor, the pyriform rims, and the anterior nasal spine region with a bur on a rotary drill is also an important surgical maneuver to achieve preferred upper lip and nasal aesthetics as well as to ensure maximum improvements in upper airway function (see Chapters 10 and 15).

These maxillary surgical maneuvers will also improve the negative vector ocular globe to lower eyelid relationship that was present preoperatively (see Chapter 25). In the teenager and young adult, this will stabilize the midface and periorbital soft tissues and limit the negative effects that would otherwise likely occur with age (e.g., soft-tissue descent, rounding of the palpebral fissure, lengthening of the lower lid, loss of cheek region soft-tissue prominence).

Surgical Planning Pitfalls

The Need for Two-Jaw Surgery

It is better to accept the need for bimaxillary and chin osteotomies in combination with intranasal procedures to obtain a favorable result rather than to compromise facial aesthetics and airway improvements in an attempt to keep the surgery within one jaw. For the patient with a long face, bimaxillary surgery is almost always beneficial to achieve the desired midface (maxillary) projection; to achieve the desired lower face (mandibular) projection; and to achieve the preferred profile line (i.e., via the control of A-point to B-point positioning through the use of counterclockwise rotation).

Maxillomandibular Counterclockwise Rotation

The achievement of a preferred A-point to B-point relationship (i.e., pitch orientation) when the patient is in the natural head position is essential for enhanced facial aesthetics. For a patient with a long face growth pattern, counterclockwise rotation of the mandibular plane is typically carried out. The maxillary plane may also require counterclockwise rotation with intrusion of the anterior maxilla (thus lessening the gummy smile) in combination with either neutral or, to a lesser extent, intrusion of the posterior maxilla. The achievement of an actual numerical plane angle is not in itself an objective. The maxillomandibular complex is counterclockwise rotated as needed to achieve the preferred profile aesthetics. Although it has been much discussed, the use of sagittal split ramus osteotomies of the mandible prevents the lengthening of the posterior facial height during these maneuvers. The proximal segment of the mandible (i.e., the condyle and ramus section) remains essentially neutral, regardless of clockwise or counterclockwise rotation of the distal aspect of the lower jaw.

Managing Incisor Inclination

When the maxillomandibular complex is counterclockwise rotated to improve the pitch orientation, effects on both the base of the nose and the relative maxillary incisor

inclination above and the pogonion and the relative mandibular incisor inclination below are to be expected. The extent of baseline deformities and the need for pitch orientation correction will influence the extent of recontouring required at the pyriform rims above and chin repositioning below to enhance facial aesthetics.

Treating the Gummy Smile

When considering the enhancement of the relationship of the upper lip to the maxillary upper incisors, it is important to avoid the planning pitfall of treating the gummy smile as an isolated feature. The clinician should make decisions about the degree of excessive anterior maxillary height and the extent of planned intrusion primarily on the basis of the anticipated upper lip position in repose and not on its position during a smile (see Fig. 21-8). In the patient with a long face, surgical alteration of the upper lip to tooth relationship will occur for a variety of reasons, including vertical maxillary intrusion; horizontal maxillary advancement; arch expansion; closure of the open bite; shortening of the chin; recontouring the pyriform rims; and recontouring and lowering of the nasal floor. The dynamic lip function and the static lip position after healing cannot be entirely predicted from the combination of skeletal changes that are generally carried out. No accurate quantitative mathematic formula for predicting the postoperative hard tissue to soft tissue changes with this condition is currently available. Keep in mind that as much as 4 mm of incisor crown exposure at rest is fine; a few millimeters of lip incompetence is acceptable, and it is better to show more teeth than less.

Postsurgical Orthodontic Maintenance and Maintenance

In general, the maintenance of the new arch form is the most challenging aspect of the postsurgical orthodontic phase of treatment. With segmentation of the maxilla, a rigid acrylic occlusal splint is prefabricated and secured to the maxillary dentition in the operating room (see Chapter 13). The splint remains in place during the initial bone-healing phase, (approximately 5 weeks). Intermittent intermaxillary elastics are used during this time to retrain the masticatory muscles, to provide motion in the temporomandibular joints, to increase patient comfort, and to confirm the stability of the occlusion. When the splint is removed, the patient should immediately (ideally within 24 hours) return to the orthodontist for the placement of a continuous maxillary arch wire. The surgically expanded arch width and the corrected curve of Spee must be actively orthodontically maintained throughout occlusal finishing (see Chapter 17). In accordance with the orthodontist's clinical judgment, this is accomplished via some combination of a rigid labial wire, a transpalatal appliance, and a palatal acrylic plate. The placement of a transpalatal appliance at the time of splint removal is facilitated by preoperatively banding the first molars with lingual attachments. The transpalatal appliance can be constructed in advance from the maxillary cast of the model planning. It is often tempting for the orthodontist to rapidly finish treatment and remove the brackets, but this is almost always a mistake. The dentoalveolar segments are generally not fully stable for approximately 6 months. If the maxillary posterior segments relapse after surgery, not only will there be a return of posterior crossbites, but the occlusion also tends to open anteriorly (see Chapter 16).

Complications, Informed Consent, and Patient Education

The orthodontist and the surgeon are asked to evaluate the individual with a long face and then to attempt to alter nature's events. With a sound understanding of the natural progression of the uncorrected dentofacial deformity and with knowledge of how specific interventions will affect function and facial aesthetics, predictable results are generally expected. The potential risks as compared with the anticipated benefits of each procedure and the review of alternative approaches to manage each problem are taken into account, because complications (i.e., unfavorable outcomes) will always occur in a small percentage of patients. The patient's willingness to make an informed decision and then to accept responsibility for that choice is an integral aspect of the patient–doctor interaction (see Chapter 17).[115]

Malocclusion after Orthodontics and Orthognathic Surgery

There are inherent biologic challenges to the orthodontic movement of the teeth within the alveolar process and the surgical repositioning of the jaws and jaw segments in the individual with a long face.* This includes the technical aspects of the repositioning; the initial healing requirements; and the difficulties associated with the long-term maintenance of the mechanically relocated teeth, jaws, and jaw segments. When following sound aesthetic and biomechanical principles, clinicians are able to carry out orthodontic treatment and surgical procedures in the individual with a long face to safely and reliably achieve the desired results in most cases. Potential intraoperative technical difficulties associated with sagittal split ramus osteotomies of the mandible and the maxillary Le Fort I osteotomy that can result in an immediate (early) malocclusion are preventable in most cases (see Chapter 17). Timing the surgical procedures to limit the risk of ongoing jaw growth after surgery is generally possible. The orthodontic removal of dental compensations with the use of bicuspid extractions as needed to place the teeth solidly in the alveolar housing

*References 8, 35, 42, 45, 49, 50, 81, 90, 91, 104, 106, 113, 126, 131, 136, 137

will best ensure stable long-term occlusion and periodontal health (see Chapter 17).

Temporomandibular Disorders: Effects of Orthodontics and Orthognathic Surgery

It is estimated that an average of 32% of the population reports at least one symptom of temporomandibular disorder, whereas an average of 55% demonstrate at least one clinical sign. Temporomandibular disorders include various symptoms and signs of the temporomandibular joints, the masticatory muscles, and the related structures. The symptoms and signs may include a spectrum of referred head and neck discomforts; joint noise (e.g., popping, clicking, crepitus); reduced or altered mandibular movements as a result of muscle spasm or disc displacement, with or without reduction; condylar head erosion; and pain on direct palpation of either temporomandibular joint or of the masticatory muscles. The presence of an anterior open-bite malocclusion is often claimed to be a factor in temporomandibular disorder. The successful correction of the Long Face dentofacial deformity through orthodontics and orthognathic surgery has been documented to improve the pretreatment symptoms of many individuals, but it may accentuate symptoms in others (see Chapter 9).[3,103]

Conclusions

The *long face growth pattern* represents a frequent presentation of dentofacial deformity for which many individuals request treatment. The opportunities for the treating clinicians to effectively improve the presenting head and neck dysfunctions (e.g., mastication, speech articulation, swallowing, breathing, lip closure/posture) and also to achieve enhanced facial aesthetics are significant. A coordinated approach and a well-informed patient and family generally result in orthodontic and surgical treatment being a life-changing event.

References

1. Adamides IP, Spyropoulos MN: The effects of lymphadenoid hypertrophy on the position of the tongue, the mandible and the hyoid bone. *Eur J Orthod* 5:287–294, 1983.
2. Ackerman RI, Klapper L: Tongue position and open bite: The key roles in growth and nasopharyngeal airway. *ASDC J Dent Child* 48:339, 1981.
3. Aghabeigi B, Hiranaka D, Keith DA, et al: Effect of orthognathic surgery on the temporomandibular joint in patients with anterior open bite. *Int J Adult Orthodon Orthognath Surg* 16:153, 2001.
4. Alexander RG: The effects on tooth position and maxillofacial vertical growth during treatment of scoliosis with the Milwaukee brace. *Am J Orthod* 52:161, 1966.
5. Angle EH: *Malocclusion of the teeth*, ed 7, Philadelphia, 1907, SS White.
6. Arvystas MG: Treatment of anterior skeletal open-bite deformity. *Am J Orthod* 72:147–164, 1977.
7. Bailey LJ, White RP, Proffit WR, et al: Segmental Le Fort I osteotomy for management of transverse maxillary deficiency. *J Oral Maxillofac Surg* 55:728, 1997.
8. Baumrind S, Korn EL, West E: Prediction of mandibular rotation: An empirical test of clinician performance. *Am J Orthod* 86:371–385, 1984.
9. Bays RA, Greco JM: Surgically assisted rapid palatal expansion. *J Oral Maxillofac Surg* 50:110, 1992.
10. Bell RA: A review of maxillary expansion in relation to rate of expansion and patient's age. *Am J Orthod* 81:32, 1982.
11. Bell WH: Revascularization and bone healing after anterior maxillary osteotomy: A study using adult rhesus monkeys. *J Oral Surg* 27:249–255, 1969.
12. Bell WH, Levy BM: Revascularization and bone healing after posterior maxillary osteotomy. *J Oral Surg* 29:4, 313–320, 1971.
13. Bell WH: Correction of skeletal type of anterior open bite. *J Oral Surg* 29:706, 1971.
14. Bell WH, Dann JJ: Correction of dentofacial deformities by surgery in the anterior part of the jaws. *Am J Orthod* 64:162, 1973.
15. Bell WH, Turvey TA: Surgical correction of posterior crossbite. *J Oral Surg* 32:811, 1974.
16. Bell WH: Le Fort I osteotomy for correction of maxillary deformities. *J Oral Surg* 33:412, 1975.
17. Bell WH, Epker BN: Surgical-orthodontic expansion of the maxilla. *Am J Orthod* 70:517, 1976.
18. Bell WH, Creekmore TD, Alexander RG: Surgical correction of the long face syndrome. *Am J Orthod* 71:40, 1977.
19. Bell WH, McBride KL: Correction of the long face syndrome by Le Fort I osteotomy. *Oral Surg* 44:493, 1977.
20. Bell WH, Sinn D: Turbinectomy to facilitate superior movement of the maxilla by Le Fort I osteotomy. *J Oral Surg* 37:129, 1979.
21. Bell WH, Proffit WP: Vertical maxillary excess. In Bell WH, Proffit WP, White RP, editors: *Surgical correction of dentofacial deformities*, Philadelphia, 1980, WB Saunders, p 272.
22. Betts NJ, Vanarsdale RL, Barber HD, et al: Diagnosis and treatment of transverse maxillary deficiency. *Int J Adult Orthodon Orthognath Surg* 10:75, 1995.
23. Bishara SE, Jakobsen JR: Longitudinal changes in three normal facial types. *Am J Orthod* 88:466–502, 1985.
24. Bjork A: Prediction of mandibular growth rotation. *Am J Orthod* 55:585–599, 1969.
25. Bresolin D, Shapiro PA, Shapiro GG, et al: Mouth breathing in allergic children: its relationship to dentofacial development. *Am J Orthod* 84:334, 1983.
26. Brunell JA, Bhat M, Lipton JA: Prevalence and distribution of selected occlusal characteristics in the US population, 1988–1991. *J Dent Res* 75:706, 1996.
27. Burstone CJ: Lip posture and its significance in treatment planning. *Am J Orthod* 53:262, 1967.
28. Cangialosi T: Skeletal morphologic features of anterior open bite. *Am J Orthod* 85:28–36, 1984.
29. Cao Y, Zhou Y, Li Z: Surgical-orthodontic treatment of Class III patients with long face problems: A retrospective study. *J Oral Maxillofac Surg* 67:1032–1038, 2009.
30. Chamberland S, Proffit WL: Short-term and long-term stability of surgically assisted rapid palatal expansion revisited. *Am J Orthod Dentofacial Orthop* 139:815–822, 2011.
31. Coben SE: Growth and class II treatment. *Am J Orthod* 52:5–26, 1966.
32. Daniel HT, White RP, Proffit WR: Anterior maxillary osteotomy in dental treatment. *J Am Dent Assoc* 83:338, 1971.
33. D'Ascanio L, Lancione C, Pompa G, et al: Craniofacial growth in children with nasal septum deviation: a cephalometric comparative study. *Int J Pediatr Otorhinolaryngol* 74:1180, 2010.
34. Dibbets JMH: The puzzle of growth rotation. *Am J Orthod* 87:473–480, 1985.
35. Donatsky O, Bjorn-Jorgensen J, Holmqvist-Larsen M, et al: Computerized cephalometric evaluation of orthognathic surgical precision and stability in relation to maxillary superior

repositioning combined with mandibular advancement or setback. *J Oral Maxillofac Surg* 55:1071, 1997.
36. Ellis, E: The nature of vertical maxillary deformities: Implications for surgical intervention. *J Oral Maxillofac Surg* 43:756–762, 1985.
37. Enache AM, Nimigean VR, Mihaltan F, et al: Assessment of sagittal and vertical skeletal patterns in Romanian patients with obstructive sleep apnea. *Rom J Morphol Embryol* 51:505, 2010
38. Epker BN, Wolford LM: Middle third facial osteotomies: Their use in the correction of acquired and developmental dentofacial and craniofacial deformities. *J Oral Surg* 33:491, 1975.
39. Epker BN, Fish LC: Surgical-orthodontic correction of open-bite deformity. *Am J Orthod* 71:278–299, 1977.
40. Epker BN: A modified anterior maxillary osteotomy. *J Maxillofac Surg* 5:35, 1977.
41. Epker BN, Fish LC: Superior repositioning of the maxilla: What to do with the mandible? *Am J Orthod Dentofacial Orthop* 78:164, 1980.
42. Epker BN: Superior surgical repositioning of the maxilla: Long-term results. *J Maxillofac Surg* 9:199, 1981.
43. Epker BN, Turvey TA, Fish LD: Indications of simultaneous mobilization of the maxilla and mandible for the correction of dentofacial deformities. *Oral Surg Oral Med Oral Pathol* 54:369, 1982.
44. Fields HW, Proffit HW, Nixon WL, et al: Facial pattern differences in long-faced children and adults. *Am J Orthod* 85:217–223, 1984.
45. Finn RA: Biomechanical considerations in the surgical correction of mandibular deficiency. *J Oral Surg* 38:257–264, 1980.
46. Fish LC, Wolford LM, Epker BN: Surgical-orthodontic correction of vertical maxillary excess. *Am J Orthod* 73:241–257, 1978.
47. Fletcher SG, Casteel RL, Bradley DP: Tongue-thrust swallow, speech articulation, and age. *J Speech Hear Disord* 26:201, 1961.
48. Forssell K, Turvey TA, Phillips C, et al: Superior repositioning of the maxilla combined with mandibular advancement: Mandibular RIF improves stability. *Am J Orthod Dentofacial Orthop* 102:342, 1992.
49. Freihofer HP, Jr: Results after midface osteotomies. *J Maxillofac Surg* 1:30, 1973.
50. Freihofer HP, Jr: Results of osteotomies of the facial skeleton in adolescence. *J Maxillofac Surg* 5:267, 1977.
51. Freng A, Kvam E: Facial sagittal growth following partial basal resection of the nasal septum: A retrospective study in man. *Eur J Orthod* 1:89–96, 1979.
52. Freng A: Dentofacial development in the long-lasting nasal stenosis. *Scand J Dent Res* 89:260–267, 1979.
53. Ghafari J, Brin I, Kelley MB: Mandibular rotation and lower face height indicators. *Angle Orthod* 59:31–36, 1989.
54. Grainger RM: *The Orthodontic Treatment Priority Index*, Washington, DC, 1967, US Department of HEW, DHEW publication (PHS) No 1000, Series 2, No 25.
55. Guenther TA, Sather AH, Kern EB: The effect of Le Fort I maxillary impaction on nasal airway resistance. *Am J Orthod Dentofacial Orthop* 85:308, 1984.
56. Haas AJ: Long-term post treatment evaluation of rapid palatal expansion. *Angle Orthod* 50:189, 1980.
57. Hall HD, Roddy SC, Jr: Treatment of maxillary alveolar hyperplasia by total maxillary osteotomy. *J Oral Surg* 33:180, 1975.
58. Hall HD, West RA: Combined anterior and posterior maxillary osteotomy. *J Oral Surg* 34:126, 1976.
59. Hannuksela A: The effects of moderate and severe atopy on the facial skeleton. *Eur J Orthod* 3:187–193, 1981.
60. Hapak FM: Cephalometric appraisal of the open-bite case. *Angle Orthod* 34:65–72, 1964.
61. Hart DL, Lundquist DO, Davis HC: The effect of vertical dimension on muscular strength. *J Orthop Sports Phys Ther* 3:57–61, 1981.
62. Harvold EP: The role of function in the etiology and treatment of malocclusion. *Am J Orthod* 54:883–898, 1968.
63. Harvold EP, Chierici G, Varervik K: Experiments on the development of dental malocclusion. *Am J Orthod* 61:38–44, 1972.
64. Harvold EP, Vargervik K, Chierici G: Primate experiments on oral sensation and dental malocclusion. *Am J Orthod Dentofacial Orthop* 63:494, 1973.
65. Harvold EP, Tomer BS, Varervik K, Chierici G: Primate experiments on oral respiration. *Am J Orthod* 79:359–372, 1981.
66. Haryett RD, Hansen FC, Davidson PO: Chronic thumb sucking: The psychological effects and relative effectiveness of various methods of treatment. *Am J Orthod Dentofacial Orthop* 53:569, 1967.
67. Haryett RD, Hansen FC, Davidson PO: Chronic thumb sucking: A second report on treatment and its psychological effects. *Am J Orthod Dentofacial Orthop* 57:164, 1970.
68. Hedemark A, Freihofer HP, Jr: The behaviour of the maxilla in vertical movements after Le Fort I osteotomy. *J Maxillofac Surg* 6:244, 1978.
69. Isaacson JR: Extreme variation in vertical facial growth and associated variation in skeletal and dental relations. *Angle Orthod* 71:219–229, 1970.
70. Isaacson JR, Isaacson RJ, Speidel MD, et al: Extreme variations in vertical growth and associated variations in skeletal and dental relations. *Angle Orthod* 41:219, 1971.
71. Isaacson JR, Zappel RJ, Worms FW, et al: Effects of rotational jaw growth on the occlusion and profile. *Am J Orthod* 72:276–286, 1977.
72. Kahnberg KE, Ridell A: Combined Le Fort I osteotomy and oblique sliding osteotomy of the mandibular rami: A follow-up. *J Craniomaxillofac Surg* 16:151, 1988.
73. Kawamoto HK, Jr: Treatment of the elongated lower face and the gummy smile. *Clin Plast Surg* 9:479, 1982.
74. Kelley JE, Sanchez M, Van Kirk GE: *An assessment of the occlusion of teeth of children 6-11 years*, Washington, DC, 1973, National Center for Health Statistics, US Dept of Health, Education, and Welfare, Public Health Service publication (HRA) No. 74-1612.
75. Kelley JE, Harvey CR: *An assessment of the occlusion of teeth of youths 12-17 years*, Washington, DC, 1977, National Center for Health Statistics, US Dept of Health, Education, and Welfare, Public Health Service publication (HRA) No. 77-1644.
76. Kondo E: The course of treatment of a class III skeletal open bite case and its analysis. *Aust Orthod J* 12:217, 1993.
77. Krebs A: Expansion of the midpalatal suture, studied by means of metallic implants. *Acta Odontol Scand* 17:491, 1958.
78. Kreiborg S, Jensen BL, Møller E, Björk A: Craniofacial growth in a case of congenital muscular dystrophy. *Am J Orthod* 74:207–215, 1978.
79. Kuo PC, Will LW: Surgical-orthodontic treatment of maxillary constriction. *Oral Maxillofac Surg Clin North Am* 2:751, 1990.
80. Lear CSC, Moorrees CFA: Bucco-lingual muscle force and dental arch form. *Am J Orthod Dentofacial Orthop* 56:379, 1969.
81. LeBanc JP, Turvey TA, Epker BN: Results following simultaneous mobilization of the maxilla and mandible for the correction of dentofacial deformities: Analysis of 100 consecutive patients. *Oral Surg Oral Med Oral Pathol* 54:607, 1982.
82. Linder-Aronson S: Adenoids: Their effect on mode of breathing and nasal airflow and their relationship to characteristics of the facial skeleton and the dentition. A biometric, rhino-manometric and cephalometro-radiographic study on children with and without adenoids. *Acta Otolaryngol Suppl* 265:1, 1970.
83. Luhr HG: Zur stabilen osteosynthese bei unterkiefer-frakturen. *Dtsch Zahnarztl Z* 23:754, 1968.
84. Luhr HG: Skelettverlagernde operationen zur harmonisierung des gesichtsprofils-probleme der stabilen fixation von osteotomiesegmenten. In Pfeifer G von, editor: *Die esthetik von form und funktion in der plastischen und wiederherstellungs-chirurgie*, Berlin, 1985, Springer-Verlag, p 87.
85. Lundström A: The importance of genetic and non-genetic factors in the facial skeleton studies in 100 pairs of twins. *Trans Eur Orthod Soc* 1954.

86. Lundström A, Woodside DG: Individual variation in growth directions expressed at the chin and the midface. *Eur J Orthod* 2:65–79, 1980.
87. Lundström A: Nature versus nurture in dento-facial variation. *Eur J Orthod* 6:77–91, 1984.
88. Man-Suk B, Yoon-Jeong C, Yu HS, et al: Long-term stability of anterior open-bite treatment by intrusion of maxillary posterior teeth. *Am J Orthod Dentofacial Orthop* 138:396e1–396e9, 2010.
89. Mason RM, Proffit WR: The tongue thrust controversy: Background and recommendations. *J Speech Hear Disord* 39:115, 1974.
90. McNance AM, Moss JP, James DR: Stability of surgical correction of patients with skeletal III and skeletal II anterior open bite, with increased maxillary mandibular plane angle. *Eur J Orthod* 14:198, 1992.
91. Miguel JA, Turvey TA, Phillips C, Proffit WR: Long-term stability of two-jaw surgery for treatment of mandibular deficiency and vertical maxillary excess. *Int J Adult Orthodon Orthognath Surg* 10:235–245, 1995.
92. Mommaerts MY, Lippens F, Abeloos JV, Neyt LF: Nasal profile changes after maxillary impaction and advancement surgery. *J Oral Maxillofac Surg* 58:470–475, 2000.
93. Moss M, Salentijn L: Differences between the functional matrices in anterior open-bite and in deep-bite. *Am J Orthod* 60:264–280, 1971.
94. Moyers RE, van der Linden FPGM, Riolo ML, et al: *Standards of human occlusal development*, Ann Arbor, 1976, University of Michigan Center for Human Growth and Development.
95. Muller G: Growth and development of the middle face. *J Dent Res* 42:385–399, 1963.
96. Nahoum HI: Vertical proportions and the palatal plane in anterior open-bite. *Am J Orthod* 59:273–282, 1971.
97. Nahoum HI, Horowitz SL, Benedicto EA: Varieties of anterior open-bite. *Am J Orthod* 61:486–492, 1972.
98. Nahoum HI: Anterior open-bite: A cephalometric analysis and suggested treatment procedures. *Am J Orthod* 67:513–521, 1975.
99. Nanda SK: Patterns of vertical growth in the face. *Am J Orthod Dentofacial Orthop* 93:103–116, 1988.
100. Nanda RS: Growth patterns in subjects with long and short faces. *Am J Orthod Dentofacial Orthop* 98:247–258, 1990.
101. Obwegeser HL, Hadjianghelou O: Two ways to correct bird-face deformity. *Oral Surg Oral Med Oral Pathol* 64:507–518, 1987.
102. Obwegeser HL: Variations of a standard approach for correction of the bird-face deformity. *J Craniomaxillofac Surg* 16:247–265, 1988.
103. O'Ryan F, Epker BN: Surgical orthodontics and the temporomandibular joint: I. Superior repositioning of the maxilla. *Am J Orthod Dentofacial Orthop* 83:407, 1983.
104. Otuyemi OD, Jones SP: Long-term evaluation of treated class II division 1 malocclusions utilizing the PAR index. *Br J Orthod* 22:171–178, 1995.
105. Park YC, Lee HA, Choi NC, Kim DH: Open bite correction by intrusion of posterior teeth with miniscrews. *Angle Orthod* 78:699–710, 2008.
106. Phillips C, Medland WH, Fields HW, Jr: Stability of surgical maxillary expansion. *Int J Adult Orthodon Orthognath Surg* 7:139, 1992.
107. Posnick JC: Maxillary excess with or without mandibular deficiency. In Posnick JC, editor: *Craniofacial and maxillofacial surgery in children and young adults*, Vol 39, Philadelphia, 2000, WB Saunders Co, pp 1025–1056.
108. Posnick JC, Fantuzzo J, Orchin J: Deliberate operative rotation of the maxillo-mandibular complex to alter the A-point to B-point relationship for enhanced facial esthetics. *J Oral Maxillofac Surg* 64:1687–1695, 2006.
109. Posnick JC, Fantuzzo JJ, Troost T: Simultaneous intranasal procedures to improve chronic obstructive nasal breathing in patients undergoing maxillary (Le Fort I) osteotomy. *J Oral Maxillofac Surg* 65:2273–2281, 2007.
110. Posnick JC, Agnihotri N: Managing chronic nasal airway obstruction at the time of orthognathic surgery: A twofer. *J Oral Maxillofac Surg* 69:695–701, 2011.
111. Proffit WR, Fields HW: Occlusal forces in normal- and long-face children. *J Dent Res* 62:571–574, 1983.
112. Proffit WR, Phillips C, Dann C, IV: Who seeks surgical orthodontic treatment? *Int J Adult Orthodon Orthognath Surg* 3:153, 1990.
113. Proffit WR, Phillips C, Douvartzidis N: A comparison of outcomes of orthodontic and surgical-orthodontic treatment of Class II malocclusion in adults. *Am J Orthod Dentofacial Orthop* 101:556–565, 1992.
114. Proffit WR, Fields HW, Moray LJ: Prevalence of malocclusion and orthodontic treatment need in the United States: estimates from the NHANES III survey. *Int J Adult Orthodon Orthognath Surg* 13:97–106, 1998.
115. Rees TD, La Trenta GS: The long face syndrome and rhinoplasty. *Perspect Plast Surg* 3:24, 1989.
116. Reyneke JP, Evans WG: Surgical manipulation of the occlusal plane. *Int J Adult Orthodon Orthognath Surg* 5:99–110, 1990.
117. Reyneke JP: *Essentials of orthognathic surgery*, Chicago, 2003, Quintessence Publishing Co Inc.
118. Rosen H: Lip-nasal aesthetics following Le Fort I osteotomies. *Plast Reconstr Surg* 82:171, 1988.
119. Rosen H: Facial skeletal expansion: Treatment strategies and rationale. *Plast Reconstr Surg* 89:798, 1992.
120. Rosen H: Occlusal plane rotation: Aesthetic enhancement in mandibular micrognathia. *Plast Reconstr Surg* 91:1231, 1993.
121. Rosen H: Aesthetics in facial skeletal surgery. *Perspect Plast Surgery* 6:1, 1993.
122. Sassouni V, Nanda S: Analysis of dentofacial vertical proportions. *Am J Orthod* 50:801–823, 1964.
123. Sassouni V: A classification of skeletal facial types. *Am J Orthod Dentofacial Orthop* 55:109, 1969.
124. Sato S, Kaneko M, Sasaguri K, et al: Different mechanics for orthodontic correction of Class II openbite and Class III openbite malocclusions. *Bull Kanagawa Dent Coll* 35:65–77, 2007.
125. Schendel SA, Eisenfeld J, Bell WH, Epker B: The long face syndrome: Vertical maxillary excess. *Am J Orthod* 70:398–408, 1976.
126. Schendel SA, Eisenfeld JH, Bell WH, et al: Superior repositioning of the maxilla: Stability and soft tissue osseous relations. *Am J Orthod Dentofacial Orthop* 70:663, 1976.
127. Schudy F: Vertical growth versus anteroposterior growth as related to function and treatment. *Angle Orthod* 34–35:75–93, 1964.
128. Schudy FF: The rotation of the mandible resulting from growth: Its implications in orthodontic treatment. *Angle Orthod* 35:36–50, 1965.
129. Shetty V, Cardid JM, Caputo AA, et al: Biomechanical rationale for surgical orthodontic expansion of the adult maxilla. *J Oral Maxillofac Surg* 52:742, 1994.
130. Silverstein K, Quinn PD: Surgically-assisted rapid palatal expansion for management of transverse maxillary deficiency. *J Oral Maxillofac Surg* 55:725, 1997.
131. Simmons KE, Turvey TA, Phillips C, Proffit WR: Surgical-orthodontic correction of mandibular deficiency: Five-year follow-up. *Int J Adult Orthodon Orthognath Surg* 7:67–80, 1992.
132. Simmons KE, Turvey TA, Phillips C, Proffit WR: Sagittal deficiency and vertical maxillary excess. Int J Adult Orthod orthodontic correction of mandibular deficiency: five-year. *Orthognath Surg* 10:235–245, 1995.
133. Siriwat PP, Jarabak JR: Malocclusion and facial morphology: Is there a relationship? An epidemiologic study. *Angle Orthod* 55:127–138, 1985.
134. Sousa JB, Anselmo-Lima WT, Valera FC, et al: Cephalometric assessment of the mandibular growth pattern in mouth-breathing children. *Int J Pediatr Otorhinolaryngol* 69:311, 2005.
135. Stahl F, Baccetti T, Franchi L, McNamara JA: Longitudinal growth changes in untreated subjects with class II division malocclusion. *Am J Orthod Dentofacial Orthop* 134:125–137, 2008.

136. Stansbury CD, Evans CA, Miloro M, et al: Stability of open bite correction with sagittal split osteotomy and closing rotation of the mandible. *J Oral Maxillofac Surg* 68:149–159, 2010.
137. Stoker NG, Epker BN: The posterior maxillary osteotomy: A retrospective study of treatment results. *Int J Oral Surg* 3:153, 1974.
138. Subtelny JD, Sakuda M: Open bite diagnosis and treatment. *Am J Orthod* 50:337–358, 1964.
139. Swinehart EW: A clinical study of open-bite. *Am J Orthod* 28:18–34, 1942.
140. Throckmorton G, Finn R, Bell W: Biomechanics of differences in lower face height. *Am J Orthod* 77:410–420, 1980.
141. Trouten JC, Enlow DH, Rabine M, et al: Morphologic factors in open-bite and deep-bite. *Angle Orthod* 53:192–211, 1983.
142. Turvey TA, Journot V, Epker BN: Correction of anterior open-bite deformity: A study of tongue function, speech changes and stability. *J Maxillofac Surg* 4:93, 1976.
143. Turvey TA, Hall DJ, Fish LC, Epker BN: Surgical-orthodontic treatment planning for simultaneous mobilization of the maxilla and mandible in the correction of dentofacial deformities. *Oral Surg Oral Med Oral Pathol* 54:491, 1982.
144. Turvey TA, Hall DJ, Warren DW: Alterations in nasal airway resistance following superior repositioning of the maxilla. *Am J Orthod* 85:109, 1984.
145. Tweed CH: *Clinical orthodontics*, St. Louis, 1966, Mosby.
146. Valera FC, Travitzki LV, Mattar SE, et al: Muscular, functional and orthodontic changes in preschool children with enlarged adenoids and tonsils. *Int J Pediatr Otorhinolaryngol* 67:761, 2003.
147. Vargervik K, Mill AJ, Chierici G, et al: Morphologic response to changes in neuromuscular patterns experimentally induced by altered mode of respiration. *Am J Orthod* 85:115–124, 1984.
148. Verdi GD, Alpert B: Correction of the long face: Case report. *Plast Reconstr Surg* 62:613, 1978.
149. Weinberg H, Kronman JH: Orthodontic influence upon anterior face height. *Angle Orthod* 36:80–88, 1966.
150. Wenzel A, Henriksen J, Melsen B: Nasal respiratory resistance and head posture: Effect of intra-nasal corticosteroid (Budesonide) in children with asthma and perennial rhinitis. *Am J Orthod* 84:422–426, 1983.
151. Wertz R, Dreskin M: Midpalatal suture opening, a normative study. *Am J Orthod Dentofacial Orthop* 71:367, 1977.
152. West RA, Epker BN: Posterior maxillary surgery: Its place in the treatment of dentofacial deformities. *J Oral Surg* 30:562, 1972.
153. West RA, McNiell RN: Maxillary alveolar hyperplasia, diagnosis and treatment planning. *J Maxillofac Surg* 3:239, 1975.
154. Westermark AH, Bystedt H, von Konow L, Sällström KO: Nasolabial morphology after Le Fort I osteotomies. *Int J Oral Maxillofac Surg* 20:25–30, 1991.
155. Wickwire NA, White RP, Proffit WR: The effect of mandibular osteotomy on tongue position. *J Oral Surg* 30:184, 1972.
156. Wylie WL, Johnson EL: Rapid evaluation of facial hyperplasia in the vertical plane. *Angle Orthod* 22:165–182, 1952.
157. Xun C, Zeng X, Wang X: Microscrew anchorage in skeletal anterior open-bite treatment. *Angle Orthod* 77:47–56, 2007.
158. Yao CC, Lee JJ, Chen HY: Maxillary molar intrusion with fixed appliances and mini-implant anchorage studied in three dimensions. *Angle Orthod* 75:754–760, 2005.

22

Asymmetric Mandibular Excess Growth Patterns

JEFFREY C. POSNICK, DMD, MD

- Hemimandibular Hyperplasia: Type I Condylar Hyperactivity
- Hemimandibular Elongation: Type II Condylar Hyperactivity
- Assessing Jaw Growth Maturity
- High Partial Condylectomy Procedure for Active Condylar Hyperactivity
- Presurgical Orthodontic Treatment
- Surgical Approach
- Complications, Informed Consent, and Patient Education
- Malocclusion after Orthodontics and Orthognathic Surgery
- Temporomandibular Disorders: The Effects of Orthodontics and Orthognathic Surgery
- Conclusions

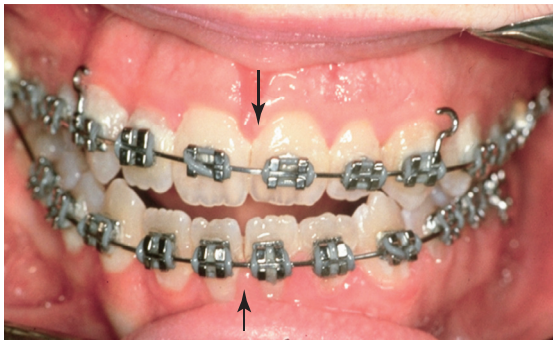
A specific pattern of dentofacial deformity that occurs after birth and that primarily affects the mandible may be referred to as an *asymmetric mandibular excess growth anomaly.** This is a useful term for name recognition, but it falls short of explaining the causes and effects of this common pattern of jaw deformity. This condition typically results in an uneven Angle Class III malocclusion, and it has been referred to by a variety of terms, including *deviated mandibular prognathism, condylar hyperplasia, mandibular lateral gnathism, osteochondroma of the mandibular condyle,* and *asymmetric Class III dentofacial deformity.* The overall effect on maxillofacial morphology also includes the secondary deformities that occur to the maxilla, the nose, the chin region, the position of the teeth, and the distortions that are visually observed in the overlying soft-tissue envelope. These effects are dependent on multiple factors, including the intensity of the mandibular hyperactivity; the patient's age when the abnormal bone growth begins; any underlying hereditary dentofacial deformity tendency; and the treatment that was previously rendered (i.e., orthodontic, dental, or surgical) before the patient's arrival to the surgeon for evaluation. These *asymmetric mandibular excess growth patterns* may be confused with other causes of maxillomandibular asymmetry (i.e., hemifacial hyperplasia, hemifacial microsomia, growth disturbance after a condyle injury during childhood).[10,27,48,49,55,93,94,124] Most misdiagnosis are easily clarified by taking an accurate history, completing a physical examination, and reviewing the patient's radiographs (Fig. 22-1).

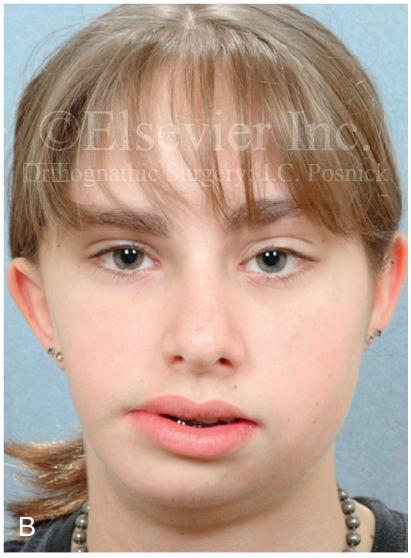
Obwegeser believes that this dentofacial deformity is caused by two different growth regulators.[23-25,68-91] The first is a more rare form, and it is clinically characterized by an increase in volume of all parts of the affected side of the mandible, without a lateral shift of the chin to the opposite side. Its primary effects end near the midline at the symphysis (Figs. 22-2 through 22-5). The second and more common form of this dentofacial deformity is characterized by an elongation of the affected side of the mandible, often with widening of the gonial angle and with clear displacement (i.e. lateral shift) of the midline of the chin and the mandibular dental midline to the opposite side of the face (Figs. 22-6 through 22-14).

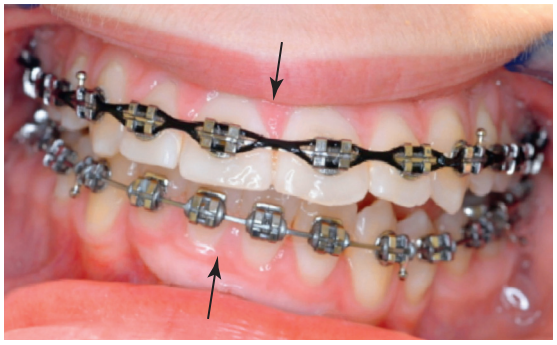
Obwegeser also stated that these two pure forms of dentofacial deformity may present as a spectrum.[23-25,68-91] He called these two pure dentofacial deformities *Type I* or *hemimandibular hyperplasia* and *Type II* or *hemimandibular elongation.* Obwegeser hypothesized that the steering mechanism for the growth of these two distinct mandibular anomalies is located entirely within the cartilaginous zone of the condylar head. This was proven to him by the fact that, whenever a very rapidly developing mandibular overgrowth (either Type I or Type II) was encountered, he was able to arrest the growth and prevent further deformity by completing a high condylar resection.

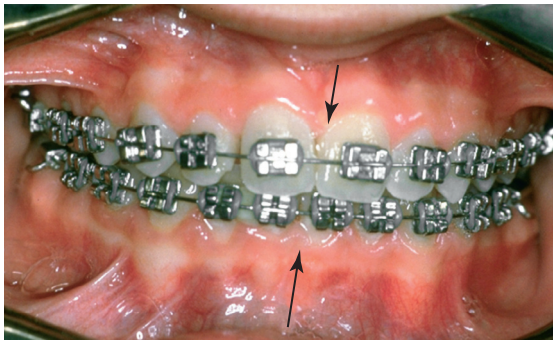
Text continued on p. 853

*References 1, 2, 4, 6, 7, 9, 11-16, 22, 28, 36, 38, 41, 47, 50, 58, 60, 62, 65-67, 100, 106, 112-114, 117, 120, 123, 125

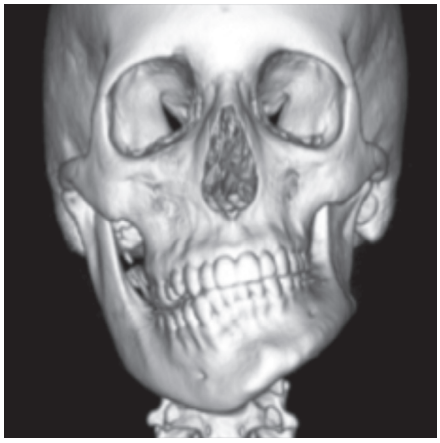


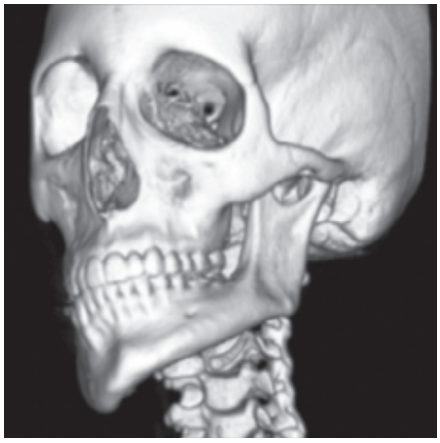


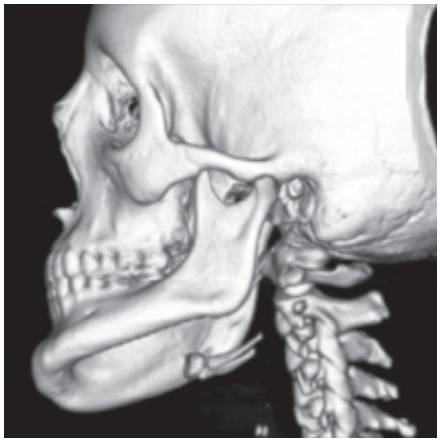




• **Figure 22-1** Asymmetric mandibular excess growth patterns are often confused with other causes of maxillomandibular asymmetry. Most misdiagnoses are easily clarified by taking an accurate patient history, completing a physical examination, and reviewing the patient's radiographs. **A,** A teenage girl with hemifacial hyperplasia of the right side is shown. This anomaly involves the overgrowth of both the soft and hard tissues on one side of the face. There is an asymmetric Class III malocclusion with a shift of the mandibular dental midline to the opposite side. **B,** Hemifacial microsomia results in the deficiency of tissues within the first and second brachial arches on one side of the face. The intraoral examination is usually consistent with an asymmetric Class II excess overjet malocclusion. This 15-year-old girl with hemifacial microsomia of the right side demonstrates these findings. There is a shift of the mandibular dental midline toward the ipsilateral side. **C,** A 12-year-old boy in the permanent dentition sustained a right condyle fracture of the mandible earlier during his life with resulting ipsilateral (right side) mandibular hypoplasia and secondary deformities of the maxilla. He presented with an asymmetric Class II deepbite malocclusion. There is a shift of the mandibular dental midline toward the ipsilateral side. The overlying soft-tissue envelope is distorted but not malformed.

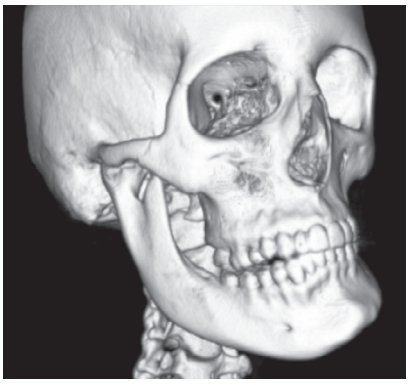


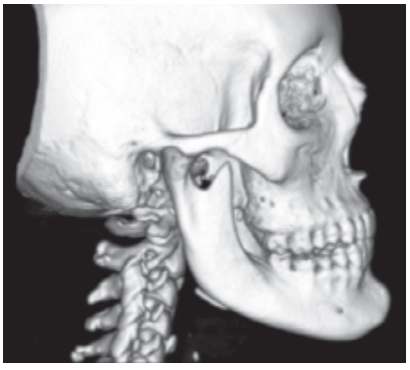


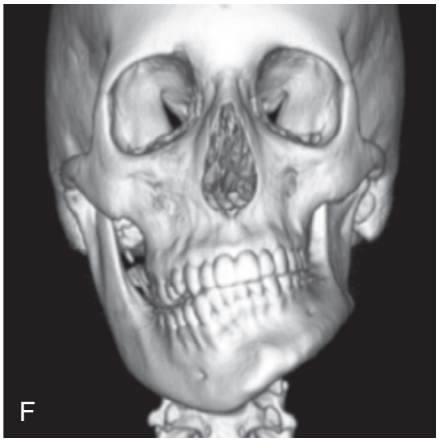


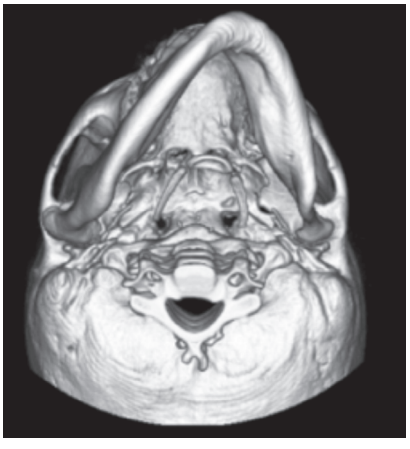
• **Figure 22-2** A teenage girl with right side hemimandibular hyperplasia. She demonstrates a typical phenotype as described within the chapter. **A,** Frontal facial and computed tomography (CT) scan views without intervention. **B,** Left facial oblique and CT scan views without intervention. **C,** Left profile and CT scan views without intervention.

Continued









• **Figure 22-2, cont'd D,** Right facial oblique and CT scan views without intervention. **E,** Right profile and CT scan views without intervention. **F,** Frontal and submental vertex CT scan views without intervention.

• **Figure 22-3** A woman in her early 20s with left side hemimandibular hyperplasia. She demonstrates a typical phenotype as described within the chapter. **A,** Frontal facial and occlusal views without intervention. **B,** Facial and posteroanterior cephalometric views without intervention.

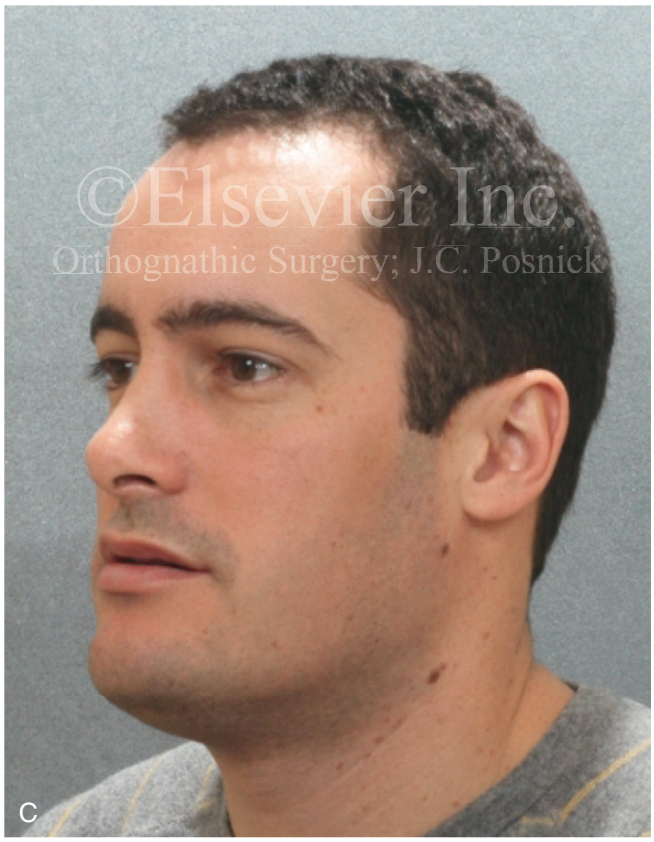
Continued

Inferior border asymmetry

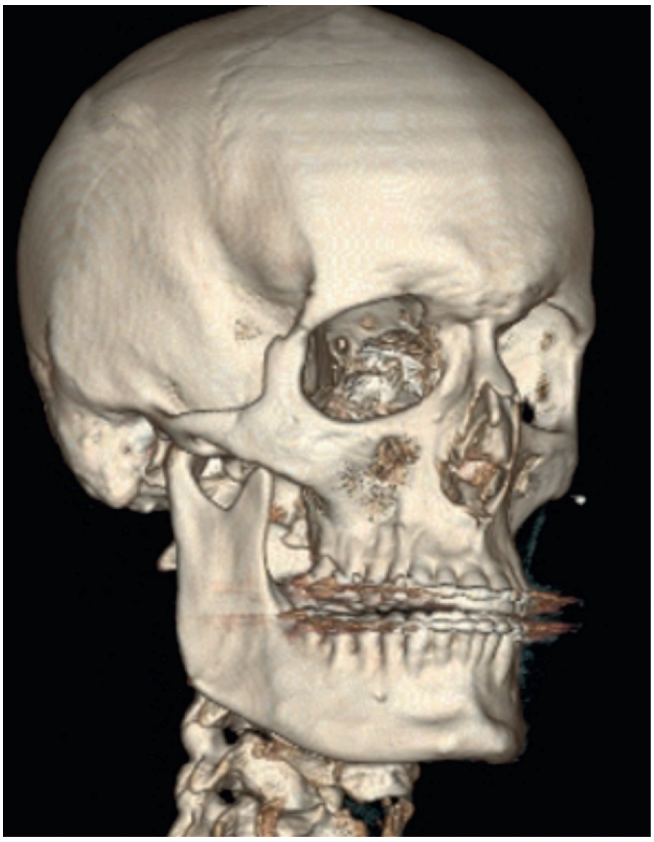
• **Figure 22-3, cont'd C,** Right profile and lateral cephalometric views without intervention. **D,** Asymmetry of the mandibular inferior borders is demonstrated in the left and right facial oblique views without intervention.

• **Figure 22-4** A man in his mid 20s with an atypical hybrid form of an asymmetric mandibular excess growth pattern on the right is shown without treatment intervention. **A,** Frontal facial view in repose. Occlusal views are also shown. The mandibular dental midline is tipped to the right. **B,** Frontal facial view with smile. A posteroanterior cephalometric radiograph is also shown.

Continued



• **Figure 22-4, cont'd C,** Left facial oblique and CT scan views without intervention. **D,** Left profile and CT scan views without intervention.



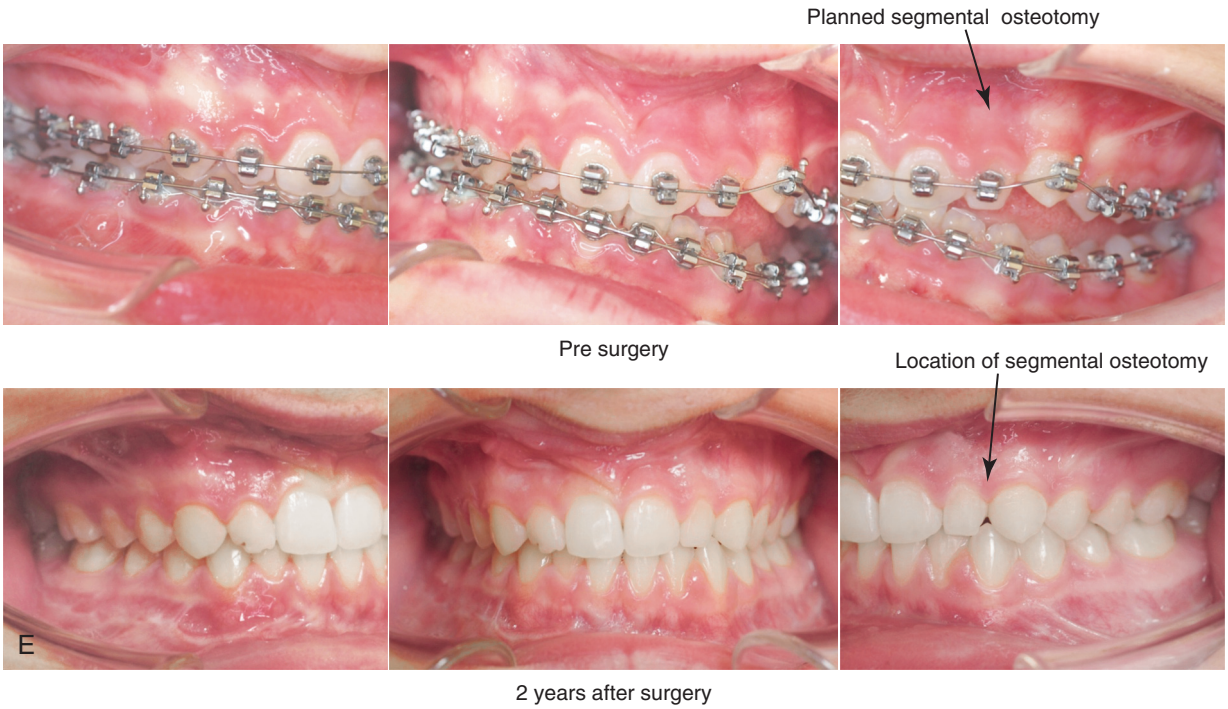
• **Figure 22-4, cont'd** **E,** Right facial oblique and CT scan views without intervention. **F,** Right profile and CT scan views without intervention.

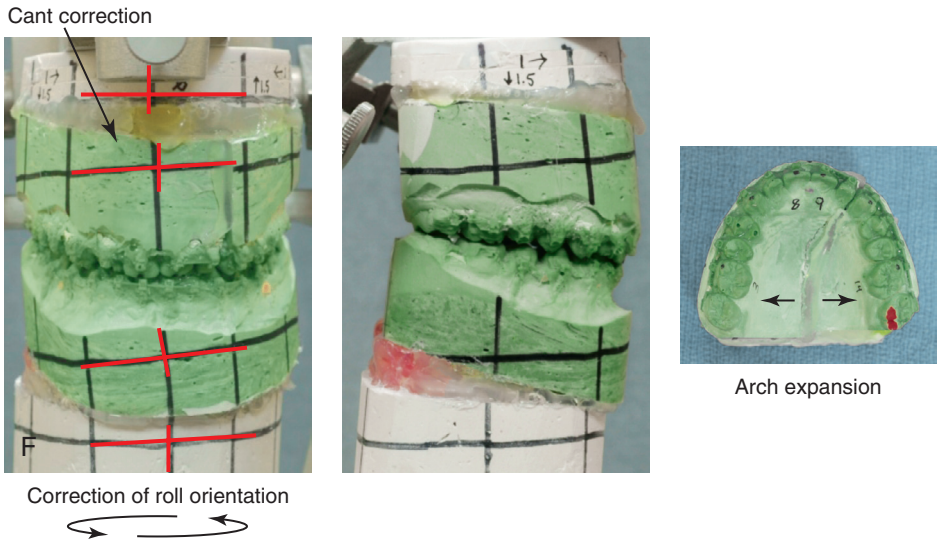
• **Figure 22-5** A 17-year-old female with hemimandibular hyperplasia of the left side and the typical facial and occlusal phenotype. She also has a lifelong history of obstructed nasal breathing. She underwent a comprehensive orthodontic and orthognathic surgical approach. Her procedures included maxillary Le Fort I osteotomy in segments (minimal advancement, cant correction, and arch expansion); bilateral sagittal split ramus osteotomies (cant correction and minimal horizontal advancement); left mandibular inferior border recontouring; osseous genioplasty (asymmetry correction); and septoplasty and inferior turbinate reduction. Note that asymmetry is present at the inferior borders before surgery and that there is much improvement afterward. **A,** Frontal views in repose before and after reconstruction. **B,** Frontal views with smile before and after reconstruction.

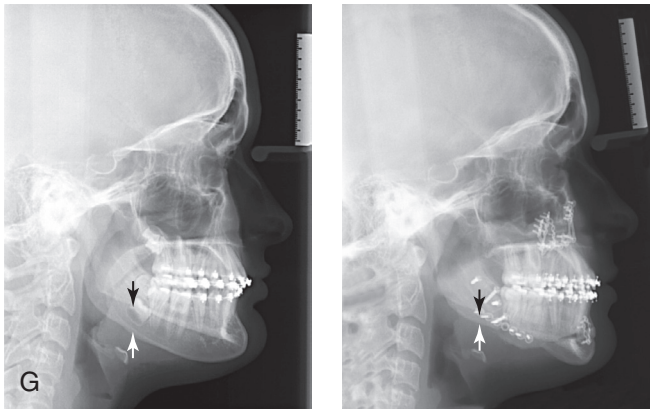
©Elsevier Inc.
Orthognathic Surgery; J.C. Posnick

• **Figure 22-5, cont'd** **C,** Left facial oblique views before and after reconstruction. **D,** Right facial oblique views before and after reconstruction.

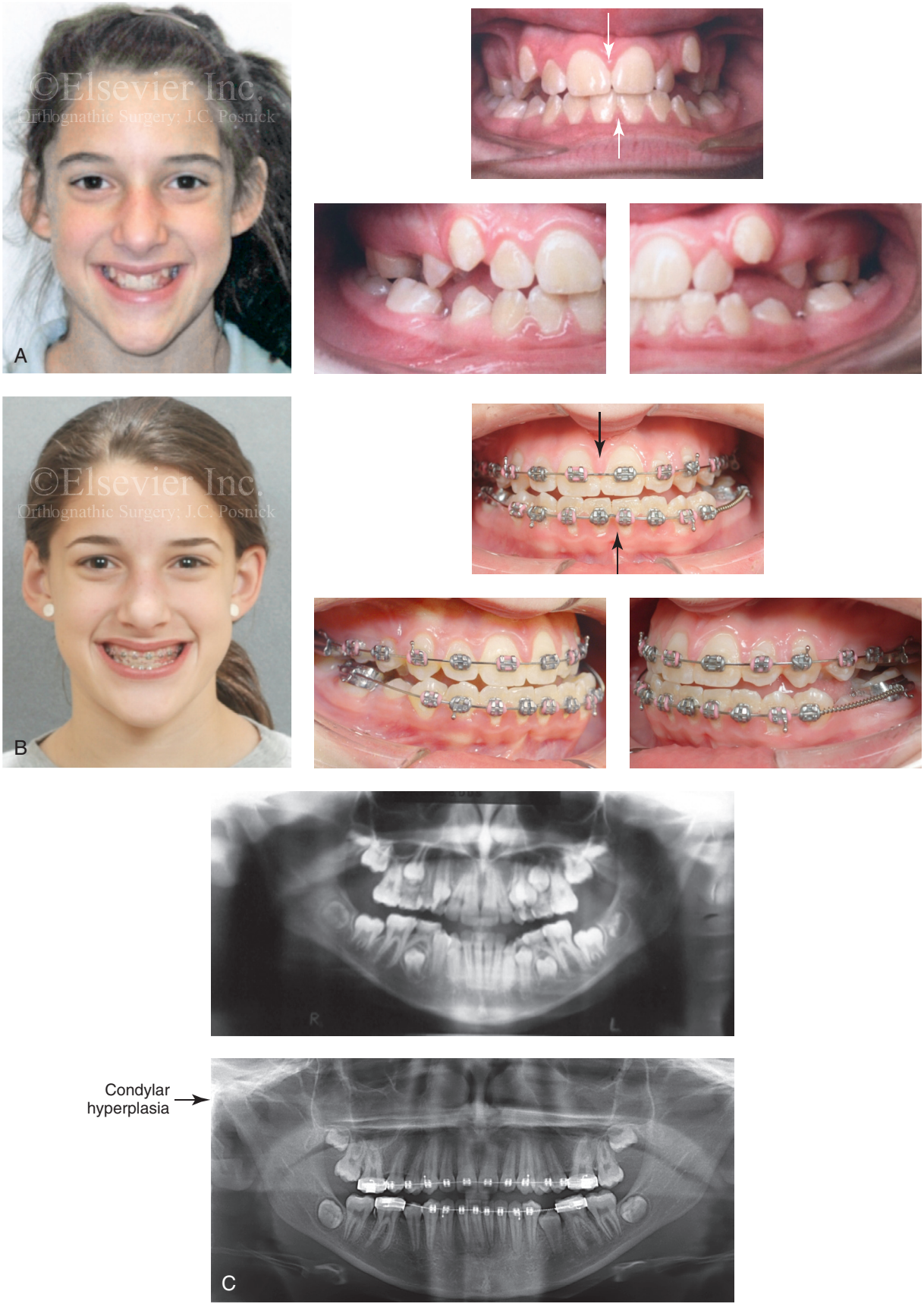
Continued







• **Figure 22-5, cont'd** **E,** Occlusal views before surgery and after reconstruction. **F,** Articulated dental models that indicate analytic surgical planning. **G,** Lateral cephalometric radiographs before and after reconstruction.



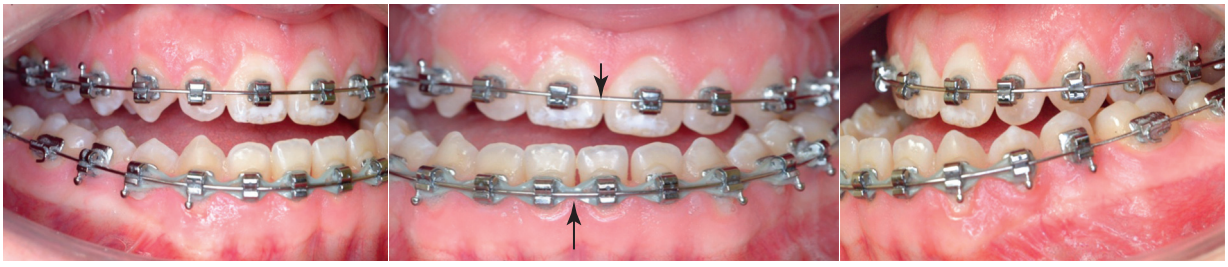
• **Figure 22-6** This is a 15-year-old girl who had been evaluated for routine orthodontic care when she was 12½ years old. At that time, she had not yet begun her menses or undergone a pubertal growth spurt. Routine orthodontics were initiated. By the time she was 15 years old (i.e., 2 years after menarche and a pubertal growth spurt), she developed an obvious asymmetric mandibular excess growth pattern due to right condylar overgrowth (Type II Hemimandibular Elongation). This resulted in both facial asymmetry and an asymmetric Angle Class III negative overjet with a posterior crossbite malocclusion. The mandibular dental midline and chin are shifted to the contralateral side. **A,** Facial and occlusal views when the patient was 12 years old. **B,** Facial and occlusal views when the patient was 15 years old. **C,** Panorex radiographs when the patient was 12 and 15 years old, respectively.

• **Figure 22-7** A 17-year-old boy with a diagnosis of left hemimandibular elongation is shown. This condition affects his chewing and speech articulation. The patient has a lifelong history of the obstruction of his nasal breathing. During his early teenage years, the patient underwent an initial phase of orthodontics, without success. He was referred for surgical evaluation and then underwent a combined redo orthodontic and surgical approach. With the relief of dental compensation, the patient's surgery included maxillary Le Fort I osteotomy in segments (horizontal advancement, cant correction, arch expansion, and clockwise rotation); bilateral sagittal split ramus osteotomies (cant correction and mandibular straightening); septoplasty and inferior turbinate reduction; and osseous genioplasty (straightening). **A,** Frontal views in repose before and after reconstruction. **B,** Frontal views with smile before and after reconstruction.

• **Figure 22-7, cont'd** **C,** Oblique facial views before and after reconstruction. **D,** Profile views before and after reconstruction.

Continued

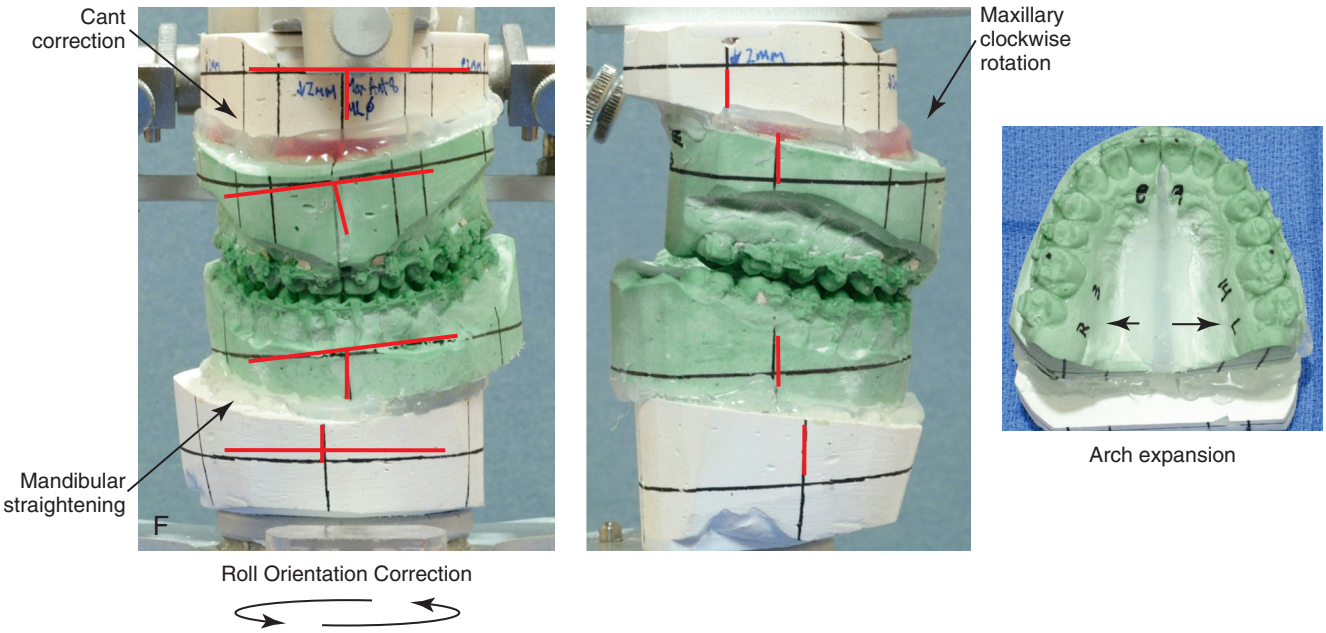
Prior to retreatment

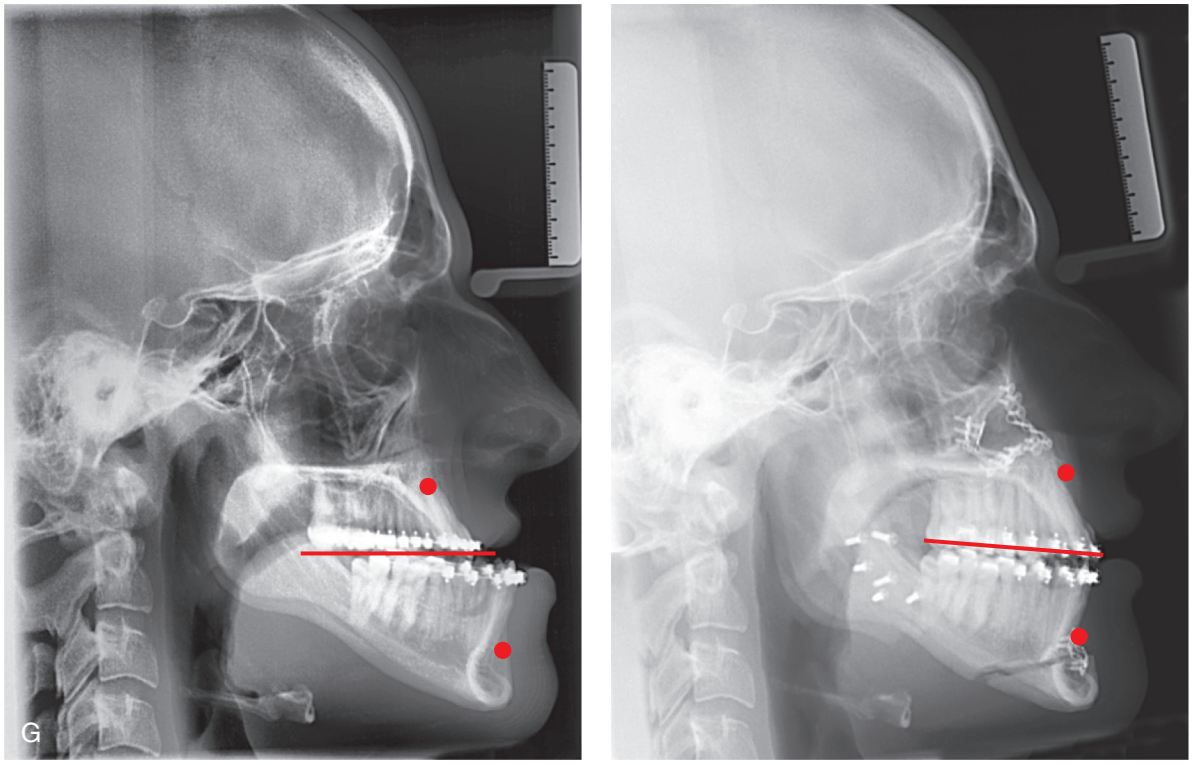


Pre surgery

After treatment

• **Figure 22-7, cont'd E,** Occlusal views before retreatment, with orthodontic decompensation, and 4 years after treatment.





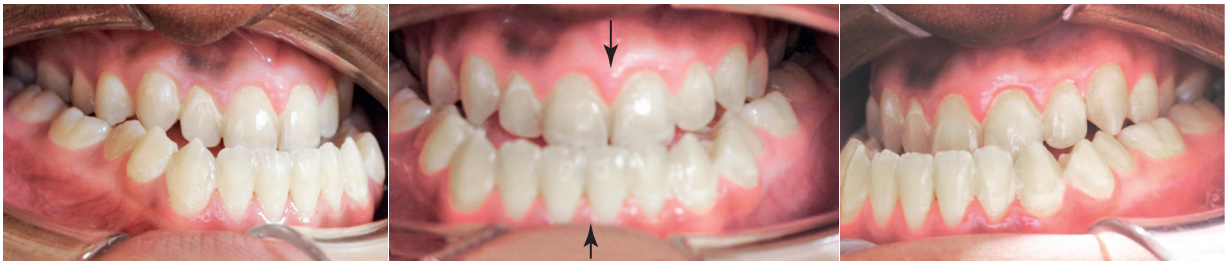
• **Figure 22-7, cont'd** **F,** Articulated dental casts that indicate analytic model planning. **G,** Lateral cephalometric radiographs before and after treatment.

©Elsevier Inc.
Orthognathic Surgery; J.C. Posnick

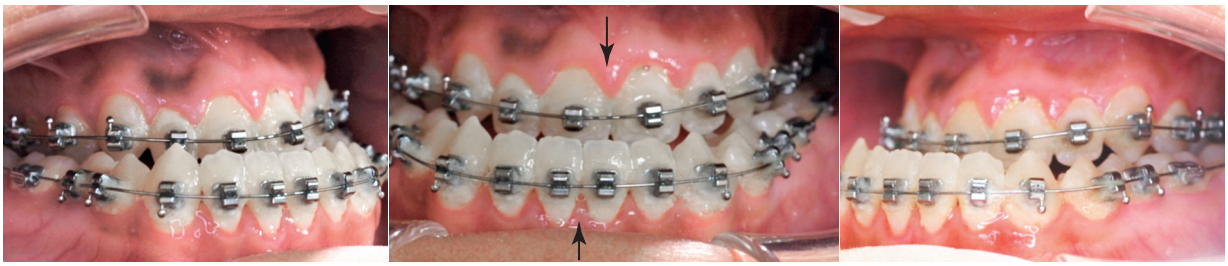
• **Figure 22-8** A 17-year-old male high school student was referred by his orthodontist for surgical evaluation. His asymmetric Class III negative overjet anterior open-bite malocclusion was consistent with hemimandibular elongation. He required redo orthodontics and jaw surgery with a non-extraction approach. The patient had a lifelong history of nasal obstruction and consistent physical findings, so intranasal procedures were also required. With orthodontic decompensation complete, the patient's surgery included maxillary Le Fort I osteotomy in segments (arch expansion, horizontal advancement, vertical lengthening, and clockwise rotation); sagittal split ramus osteotomies (minimal set-back); osseous genioplasty (vertical reduction); and septoplasty and inferior turbinate reduction. **A,** Frontal views in repose before and after treatment. **B,** Frontal views with smile before and after treatment.

• **Figure 22-8, cont'd** **C,** Oblique facial views before and after treatment. **D,** Profile views before and after treatment.

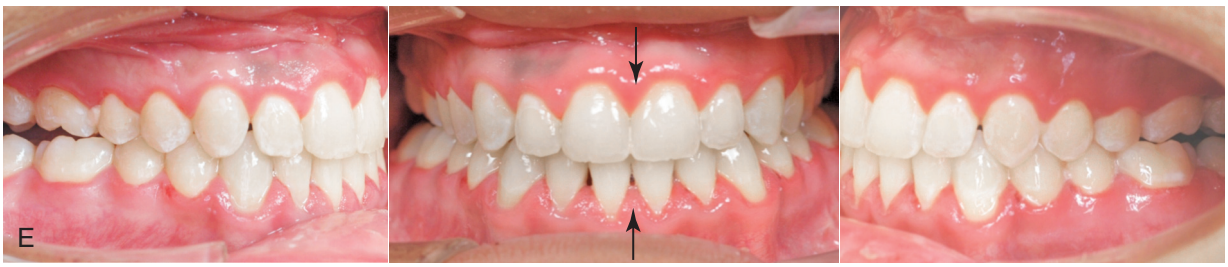
Continued



Prior to retreatment

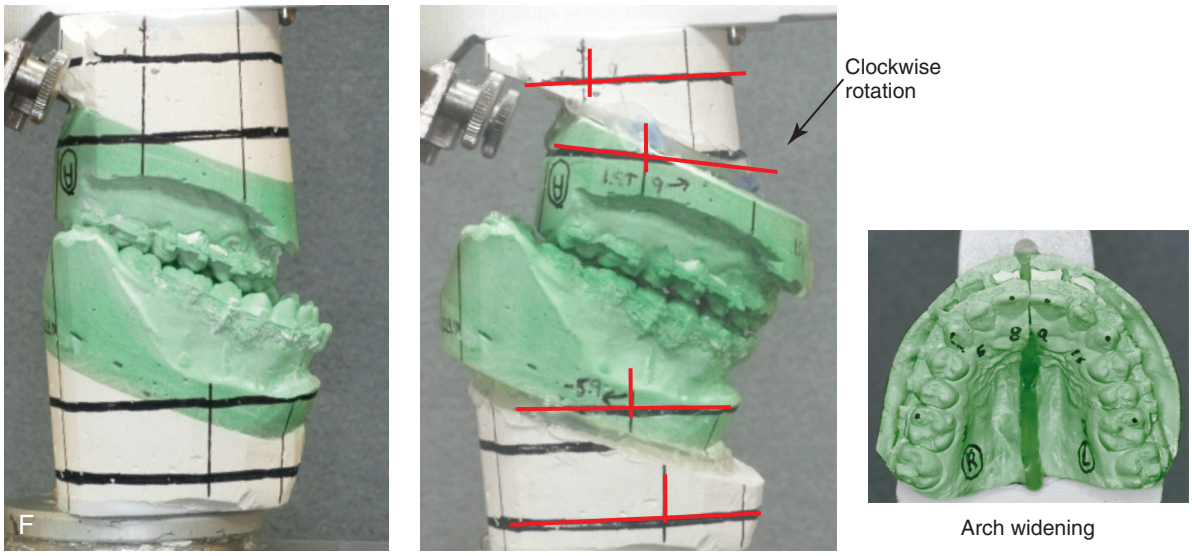


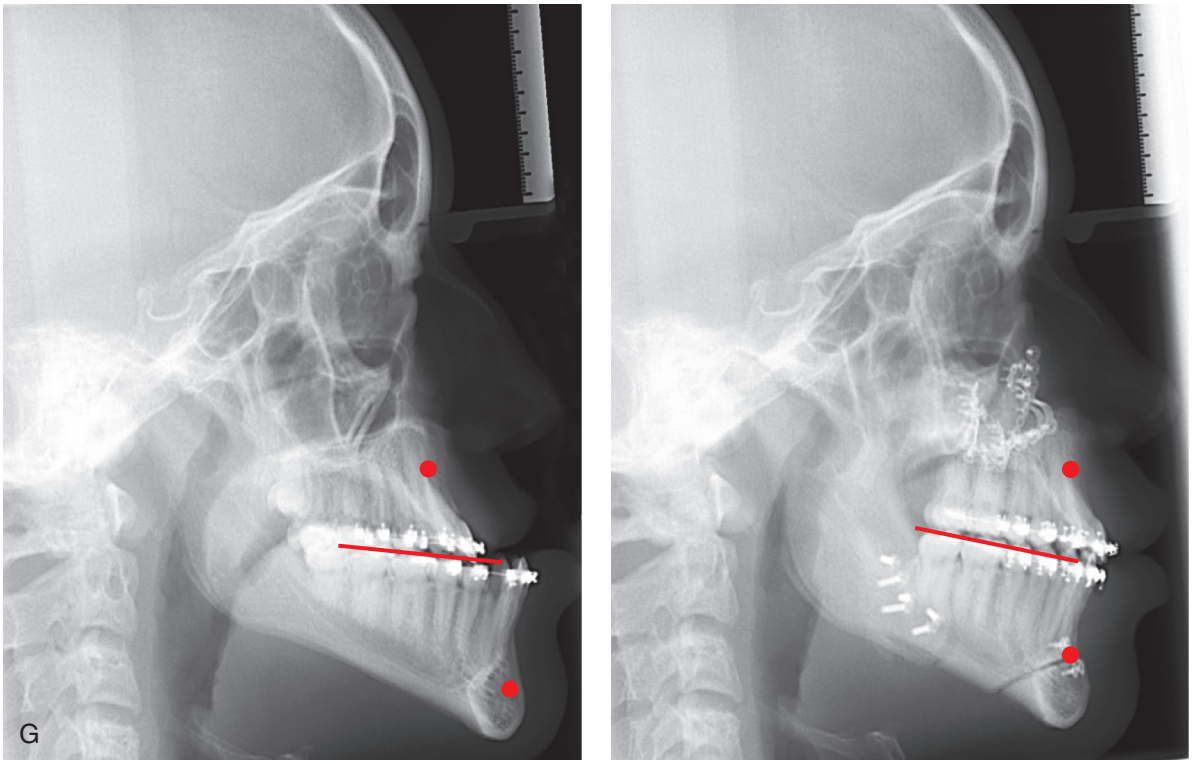
Pre surgery



After treatment

• **Figure 22-8, cont'd E,** Occlusal views before retreatment, with orthodontics in progress, and after the completion of treatment.





• **Figure 22-8, cont'd** **F,** Articulated dental casts that indicate analytic model planning. **G,** Lateral cephalometric radiographs before and after treatment. Note the improved A-point to B-point relationship that has been achieved through maxillary clockwise rotation.

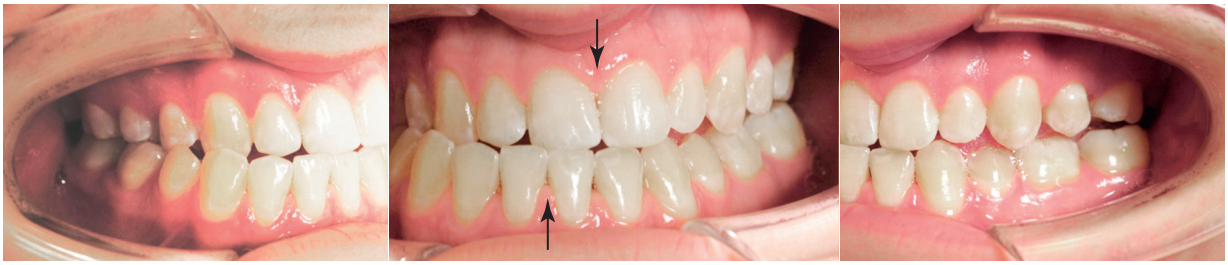
©Elsevier Inc.
Orthognathic Surgery; J.C. Posnick

• **Figure 22-9** A 20-year-old man with left side hemimandibular elongation is shown. During his teenage years, he underwent attempted growth modification and orthodontic camouflage that included the removal of three bicuspids, without success. He had a lifelong history of obstructed nasal breathing. He was referred to this surgeon for evaluation and agreed to a comprehensive redo orthodontic and orthognathic approach. With the relief of dental compensation, his surgery included maxillary Le Fort I osteotomy in segments (minimal advancement, vertical adjustment, cant correction, arch expansion, and the correction of the curve of Spee); bilateral sagittal split ramus osteotomies (mandibular straightening and asymmetric correction); osseous genioplasty (asymmetry improvement); and septoplasty and inferior turbinate reduction. **A,** Frontal views in repose before and after reconstruction. **B,** Frontal views with smile before and after reconstruction.

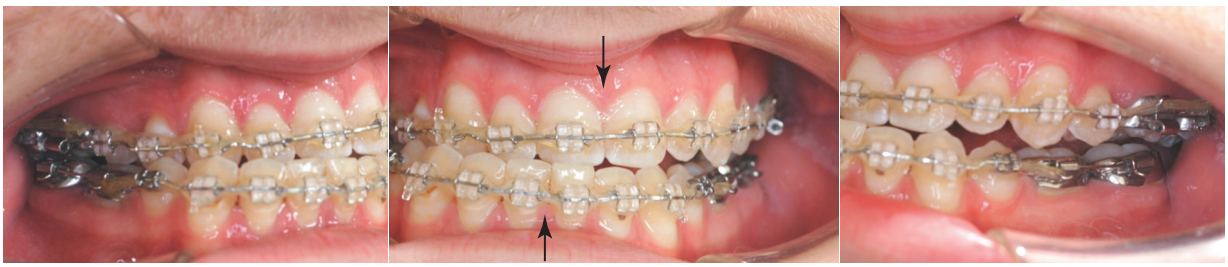
• **Figure 22-9, cont'd** **C,** Left oblique facial views before and after reconstruction. **D,** Left profile views before and after reconstruction.

Continued

• **Figure 22-9, cont'd** **E,** Right oblique facial views before and after reconstruction. **F,** Right profile views before and after reconstruction.



Prior to retreatment with 3 bicuspid extractions carried out as a teenager

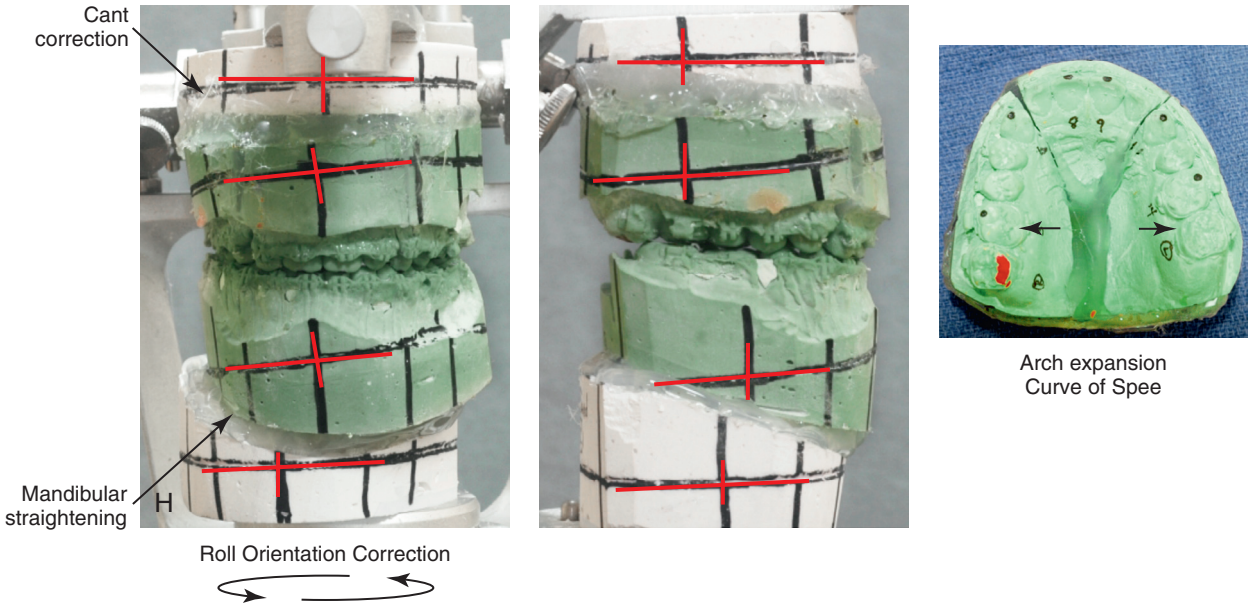


Pre surgery

After treatment

• **Figure 22-9, cont'd G,** Occlusal views before retreatment, after orthodontic decompensation, and after treatment. Note that the left mandibular second molar and the right maxillary second molar are unopposed; this is a result of the orthodontic extraction pattern.

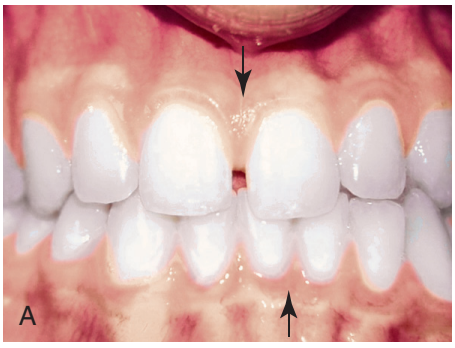
Continued



• **Figure 22-9, cont'd** **H,** Articulated dental casts that indicate analytic model planning. **I,** Panorex radiograph taken before retreatment that indicates left condyle hyperplasia and missing bicuspids in three quadrants. **J,** Lateral cephalometric radiographs before and after reconstruction.

©Elsevier Inc.
Orthognathic Surgery; J.C. Posnick

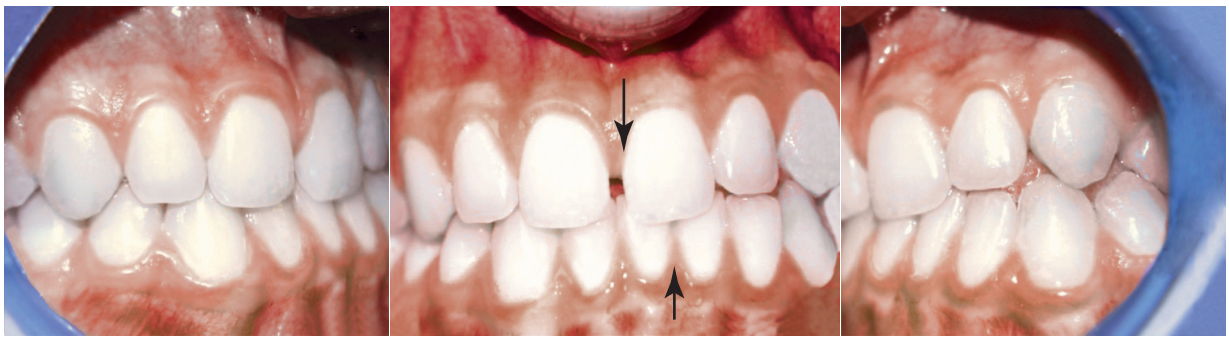
©Elsevier Inc.
Orthognathic Surgery; J.C. Posnick



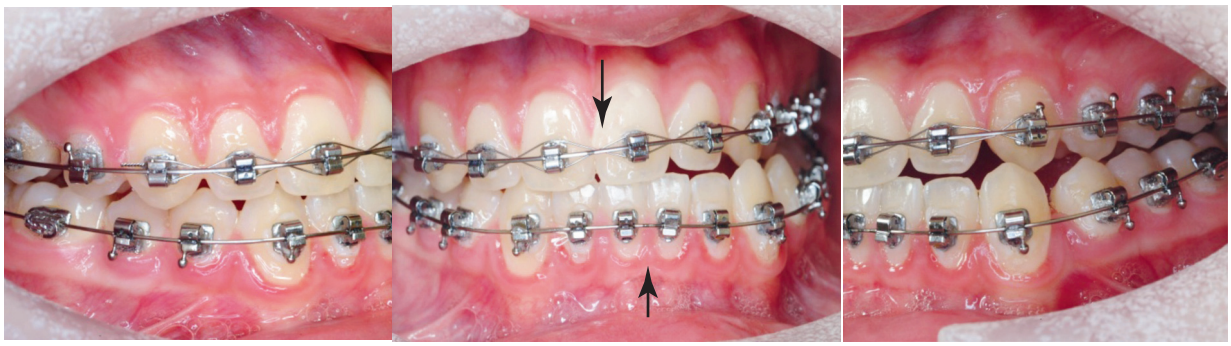
• **Figure 22-10** A teenaged boy with right side hemimandibular elongation. When he was between 10 and 14 years old, he was treated with growth modification attempts and then orthodontic camouflage. His overjet improved at the expense of the dental compensation, and the facial deformity remained. He was referred to this surgeon, who recommended stopping active treatment until the patient was skeletally mature. When he was 17 years old, he underwent redo orthodontics and an orthognathic approach. The orthodontics were carried out without extractions, which left the maxillary incisors procumbent. Clockwise rotation of the maxillomandibular complex as part of the orthognathic correction was helpful to overcome the facial disharmony. The patient's surgical procedures included maxillary Le Fort I osteotomy in segments (cant correction, horizontal advancement, clockwise rotation, arch expansion, and vertical lengthening); bilateral sagittal split ramus osteotomies (mandibular straightening and clockwise rotation); and septoplasty and inferior turbinate reduction. **A,** Facial and occlusal views when the patient was 14 years old, after failed growth modification and orthodontic camouflage.

Continued

• **Figure 22-10, cont'd B,** Facial views in repose before and after reconstruction. **C,** Frontal views with smile before and after reconstruction. **D,** Profile views before and after reconstruction.



Prior to redo orthodontics

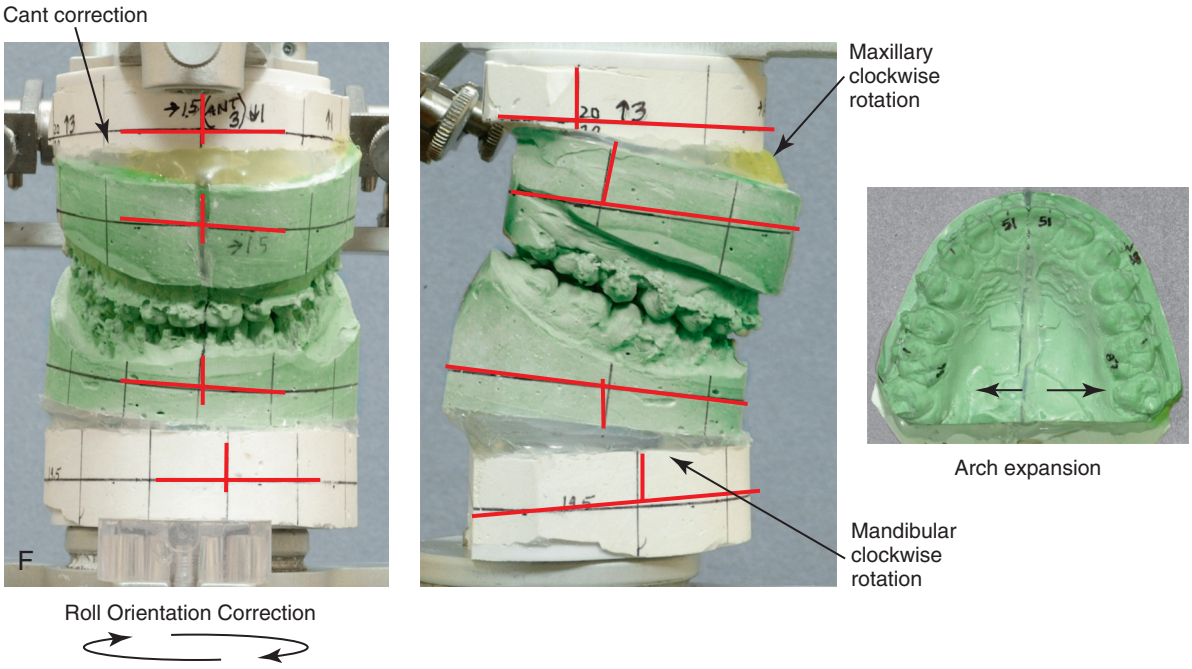


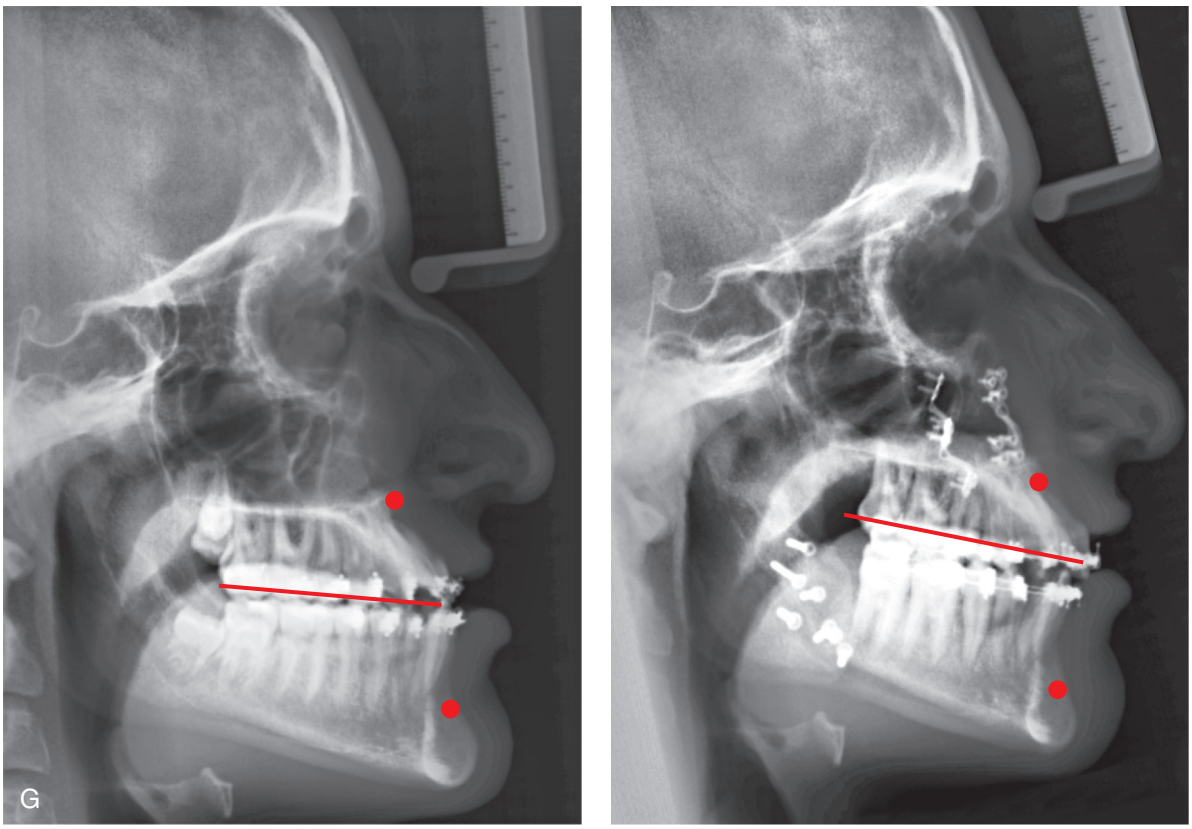
Pre surgery

After treatment

• **Figure 22-10, cont'd E,** Occlusal views when the patient was 14 years old just after the removal of orthodontics appliances, with retreatment in preparation for surgery, and after reconstruction.

Continued





• **Figure 22-10, cont'd F,** Articulated dental casts that indicate analytic model planning. **G,** Lateral cephalometric radiographs before and after reconstruction. Clockwise rotation of the maxillomandibular complex was helpful to improve the A-point to B-point relationship, despite maxillary incisor procumbancy.

©Elsevier Inc.
Orthognathic Surgery; J.C. Posnick

• **Figure 22-11** A 17-year-old girl with right side hemimandibular elongation. When she was between 10 and 14 years old, she was treated with growth modification and orthodontic camouflage techniques. She was referred to this surgeon when she was 17 years old to address her facial asymmetry and malocclusion. She underwent redo orthodontics to decompensate the arches as well as an orthognathic approach. Her surgical procedures included maxillary Le Fort I osteotomy (horizontal advancement and clockwise rotation); bilateral sagittal split ramus osteotomies (mandibular straightening); osseous genioplasty (horizontal advancement); and septoplasty and inferior turbinate reduction. **A,** Frontal views in repose before and after reconstruction. **B,** Frontal views with smile before and after reconstruction.

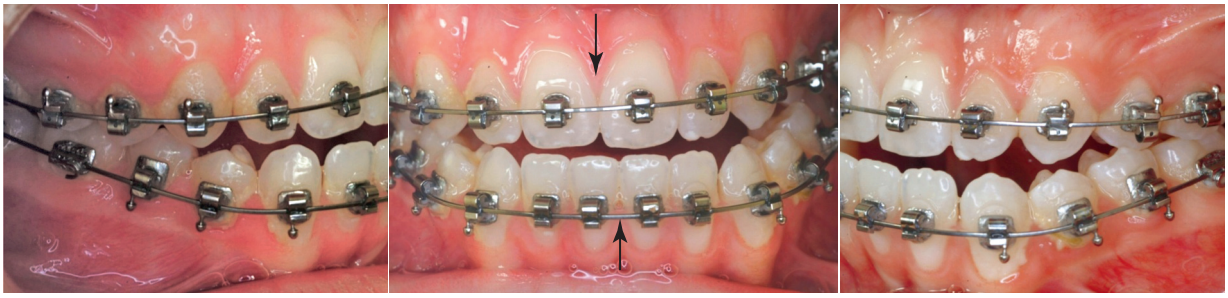
Continued

• **Figure 22-11, cont'd** **C,** Left oblique facial views before and after treatment. **D,** Left profile views before and after reconstruction.

• **Figure 22-11, cont'd E,** Right oblique views before and after reconstruction. **F,** Right profile views before and after reconstruction.

Continued

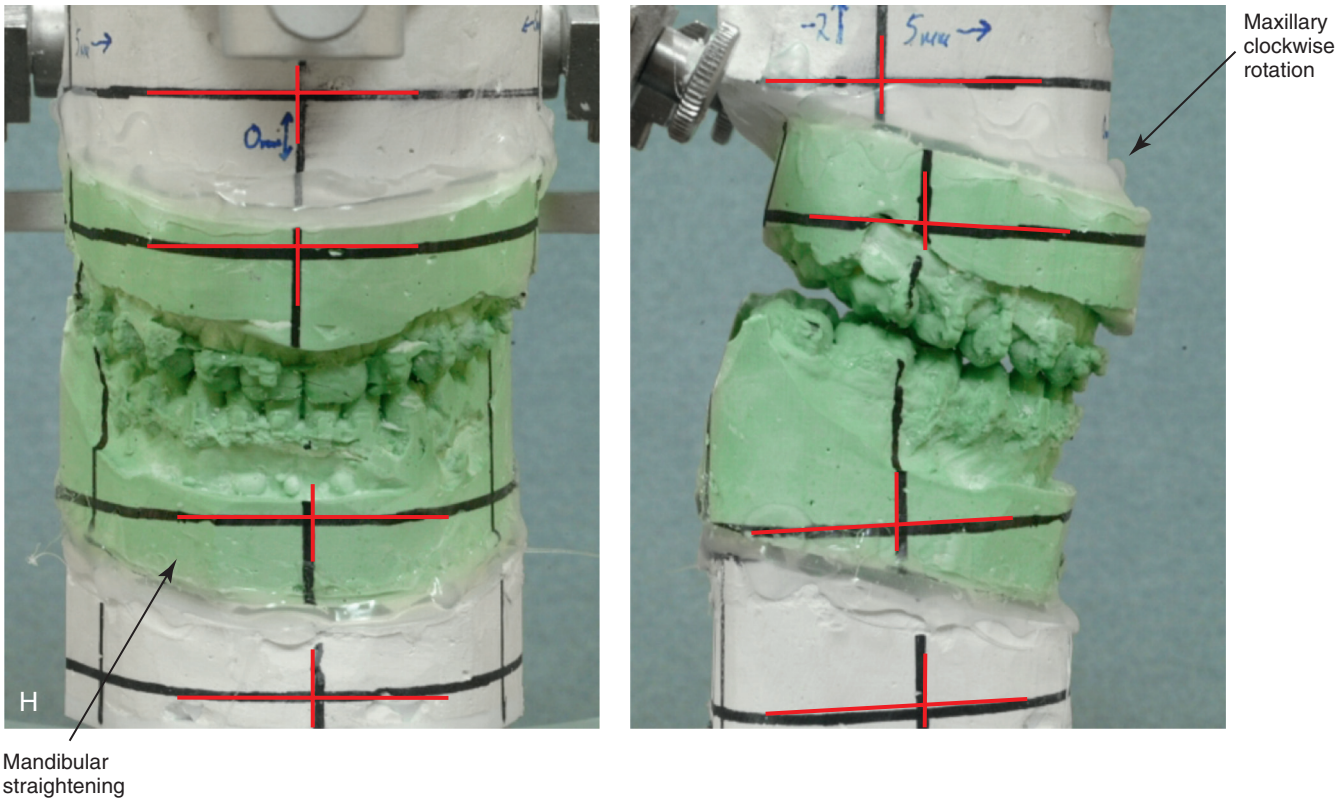
Prior to retreatment



Pre surgery

After treatment

• **Figure 22-11, cont'd G,** Occlusal views before retreatment, in preparation for surgery, and after reconstruction.

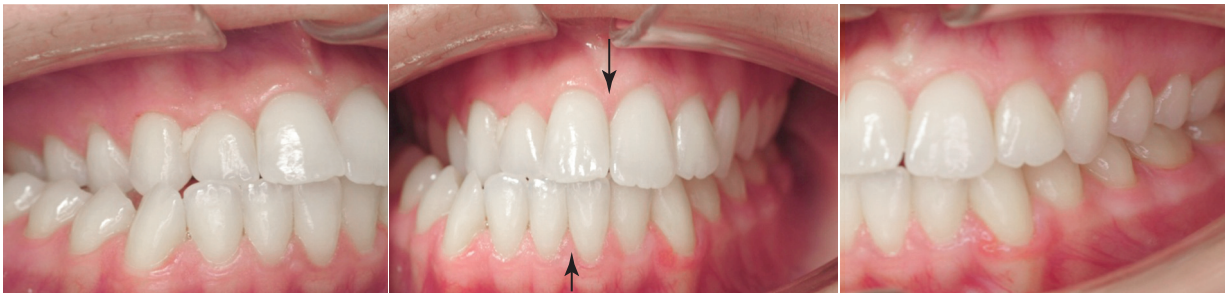


• **Figure 22-11, cont'd** **H,** Articulated dental casts that indicate analytic model planning. **I,** Lateral cephalometric radiographs before and after reconstruction.

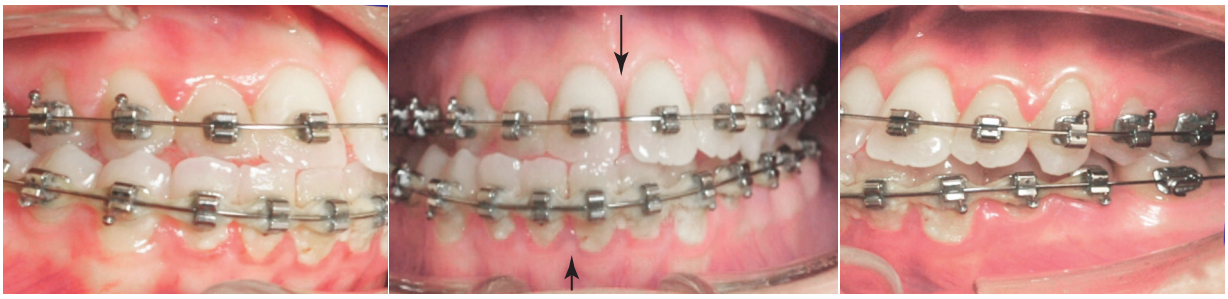
• **Figure 22-12** A 25-year-old woman with left side hemimandibular elongation. She was treated with growth modification and then orthodontic camouflage earlier in life, without success. She was referred to this surgeon and underwent redo orthodontics to decompensate the arches as well as an orthognathic approach. Lifelong nasal obstruction and intranasal examination confirmed the need for septoplasty and inferior turbinate reduction. The orthodontics were carried out without extractions, which left the maxillary incisors procumbent. Clockwise rotation of the maxilla as part of the orthognathic correction was helpful to overcome this problem and to improve the A-point to B-point relationship. Her surgical procedures included maxillary Le Fort I osteotomy in segments (cant correction, horizontal advancement, clockwise rotation, and arch expansion); bilateral sagittal split ramus osteotomies (mandibular straightening); osseous genioplasty (horizontal advancement); and septoplasty and inferior turbinate reduction. **A,** Facial views in repose before and after reconstruction. **B,** Frontal views with smile before and after reconstruction.

• **Figure 22-12, cont'd C,** Oblique facial views before and after reconstruction **D,** Profile views before and after reconstruction.

Continued



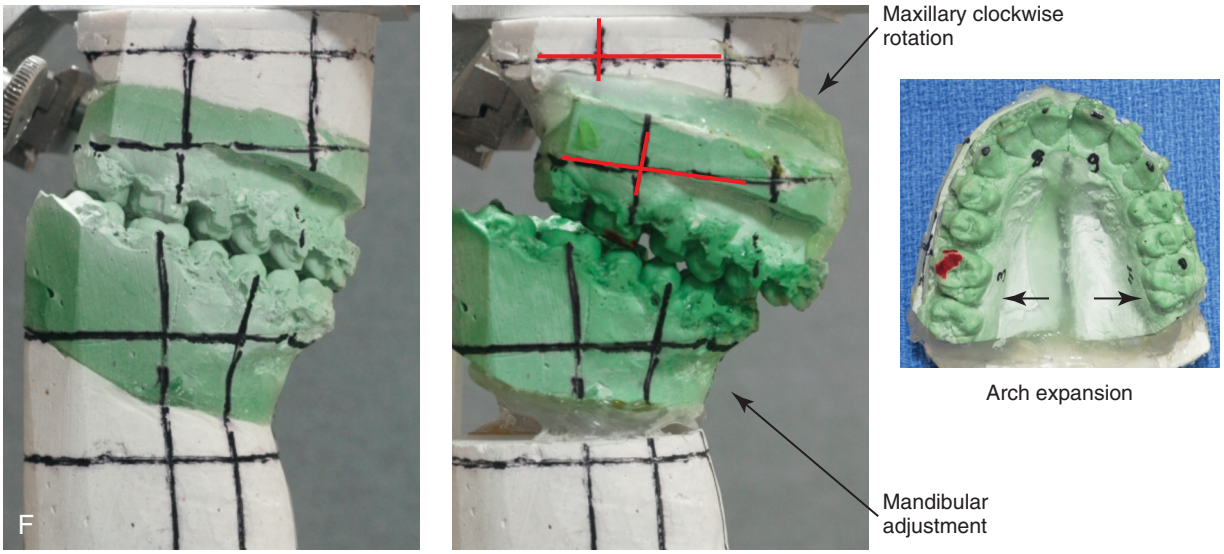
Prior to retreatment

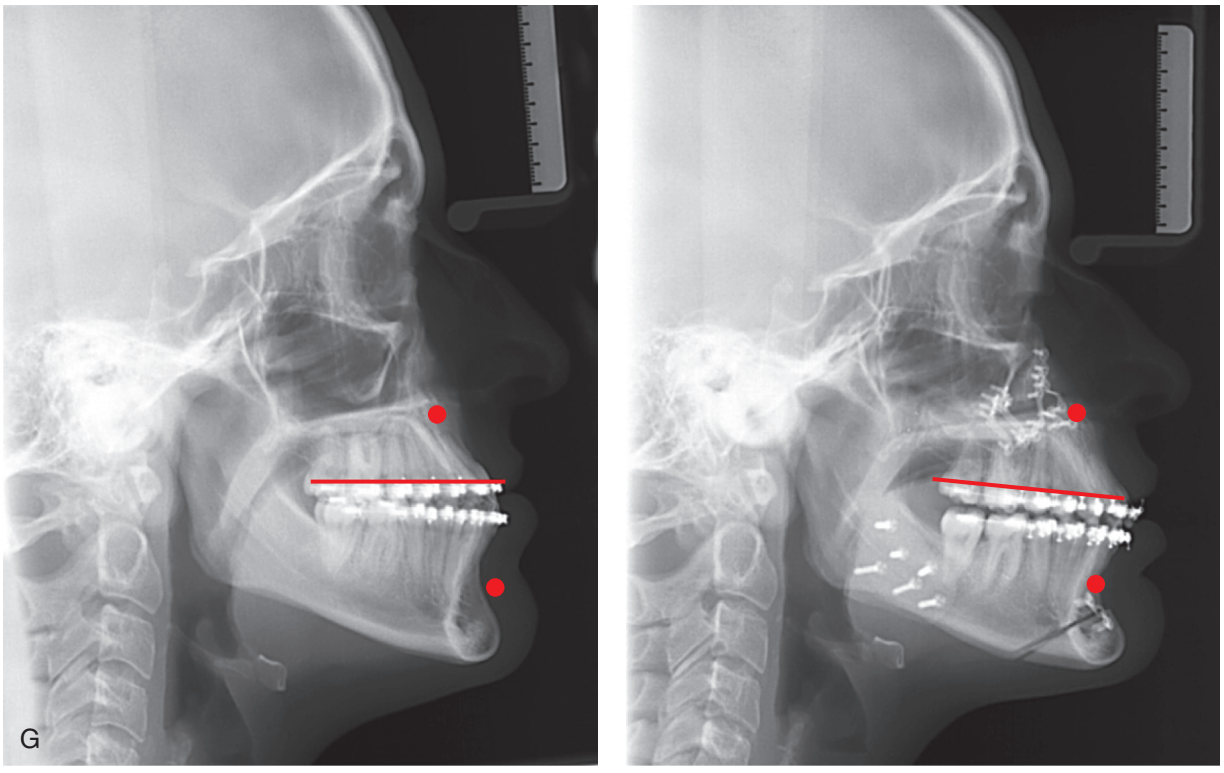


Pre surgery

After treatment

• **Figure 22-12, cont'd E,** Occlusal views before retreatment, in preparation for surgery, and after reconstruction.





• **Figure 22-12, cont'd F,** Articulated dental casts that indicate analytic model planning. **G,** Lateral cephalometric radiographs before and after reconstruction.

©Elsevier Inc.
Orthognathic Surgery; J.C. Posnick

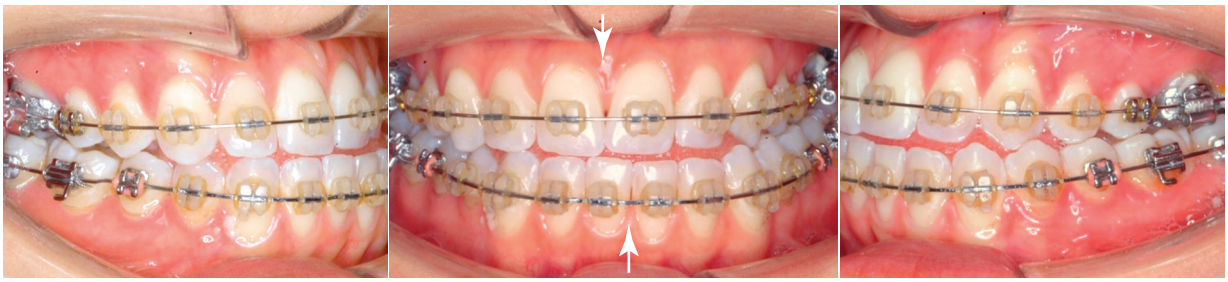
• **Figure 22-13** A 20-year-old woman with right side hemimandibular elongation. She was treated with orthodontic camouflage techniques, without success. She was referred to this surgeon and underwent an orthognathic and redo orthodontic approach. Her surgical procedures included maxillary Le Fort I osteotomy in segments (horizontal advancement, clockwise rotation, and arch expansion); bilateral sagittal split ramus osteotomies (straightening and asymmetric correction); and septoplasty and inferior turbinate reduction. **A,** Facial views in repose before and after reconstruction. **B,** Frontal views with smile before and after reconstruction.

• **Figure 22-13, cont'd** **C,** Left oblique views before and after reconstruction. **D,** Left profile views before and after reconstruction.

Continued

©Elsevier Inc.
Orthognathic Surgery; J.C. Posnick

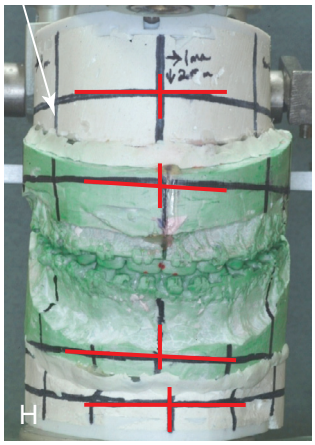
• **Figure 22-13, cont'd** **E,** Right oblique views before and after reconstruction. **F,** Right profile views before and after reconstruction.



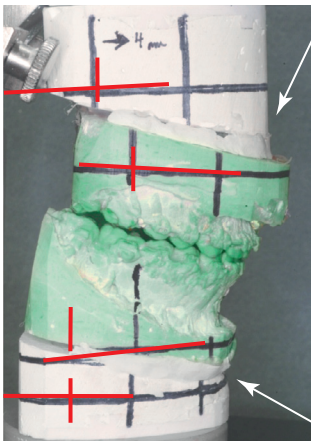
Pre surgery

After treatment

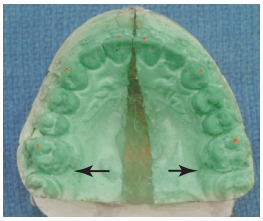
Cant correction



Roll Orientation Correction



Maxillary clockwise rotation



Arch expansion

Mandibular counter clockwise rotation

• **Figure 22-13, cont'd** **G,** Occlusal views in preparation for surgery and after reconstruction. **H,** Articulated dental casts that indicate analytic model planning. **I,** Lateral cephalometric radiographs before and after reconstruction. Clockwise rotation of the maxilla was helpful to improve the A-point to B-point relationship.

©Elsevier Inc.
Orthognathic Surgery; J.C. Posnick

• **Figure 22-14** A 17-year-old girl with left side hemimandibular elongation. Earlier in her life, she was treated with growth modification and orthodontic camouflage techniques, without success. She also has a lifelong history of nasal obstruction. She was referred to this surgeon and underwent redo orthodontics and an orthognathic approach. Her surgical procedures included maxillary Le Fort I osteotomy (cant correction and horizontal advancement); bilateral sagittal split ramus osteotomies (mandibular straightening); osseous genioplasty (minimal horizontal advancement); and septoplasty and inferior turbinate reduction. **A,** Facial views in repose before and after reconstruction. **B,** Frontal views with smile before and after reconstruction.

• **Figure 22-14, cont'd C,** Left oblique facial views before and after reconstruction. **D,** Right oblique views before and after reconstruction. **E,** Profile views before and after reconstruction.

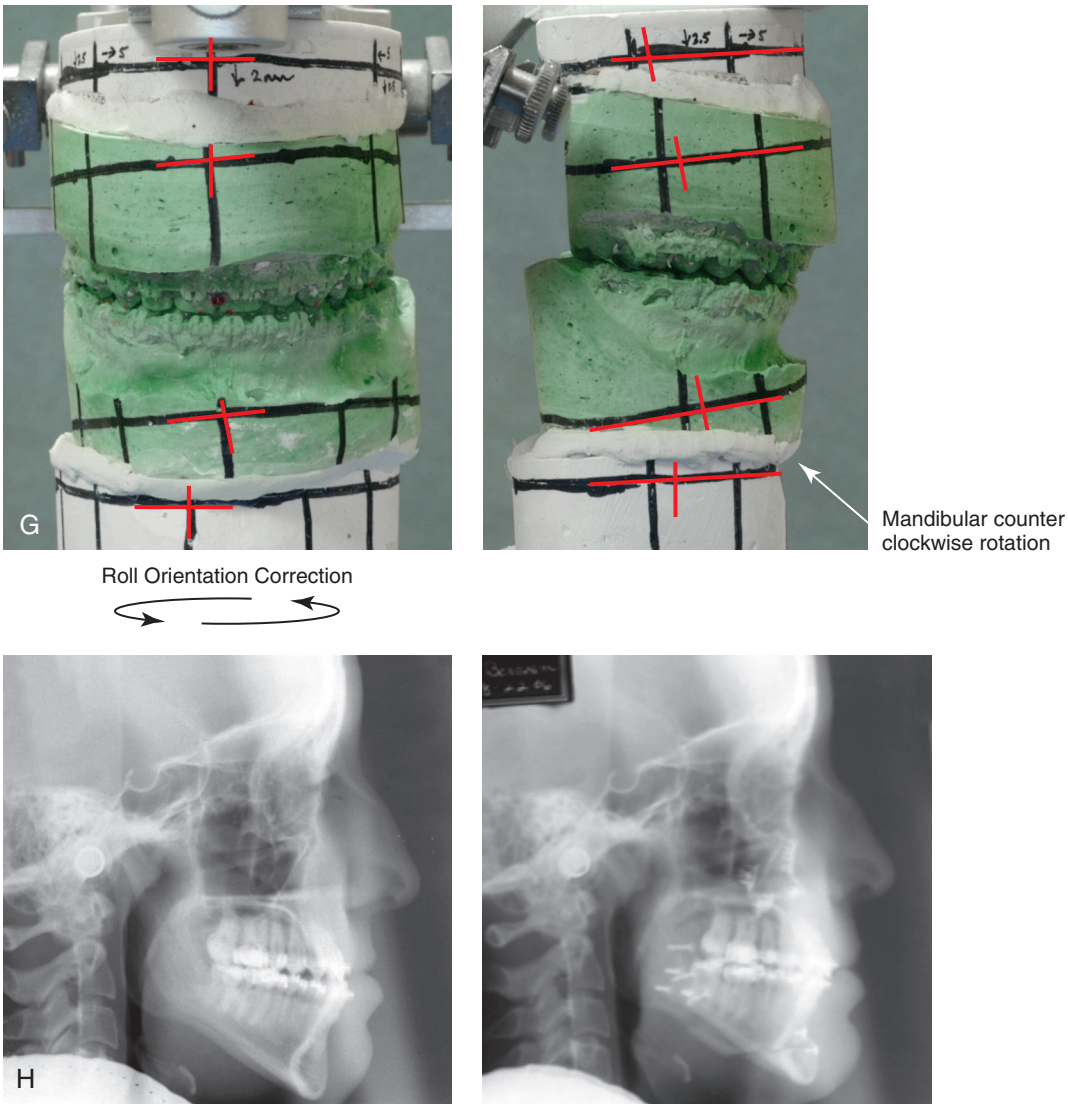
Continued

Prior to retreatment

Pre surgery

After treatment

• **Figure 22-14, cont'd F,** Occlusal views before retreatment, in preparation for surgery, and after reconstruction. Note that the maxillary and mandible dental midlines are improved but not fully corrected.



• **Figure 22-14, cont'd G,** Articulated dental casts that indicate analytic model planning. **H,** Lateral cephalometric radiographs before and after reconstruction.

Raijmakers and colleagues carried out a meta-analysis to determine gender differences when it came to the diagnosis of asymmetric mandibular excess.[101] Ten studies were reviewed, and these included a total of 275 affected patients. The meta-analysis showed a clear predominance of female patients in the study populations. The male and female patients had an equal distribution of left- and right-sided condylar overgrowth. The authors postulate that hormonal variations—especially of estrogen in women—may explain these differences. An alternative explanation put forth for this apparent female predilection is a difference in motivation between the sexes to seek the correction of their facial asymmetry (see Fig. 22-6).

Slootweg and Muller described four histologically different types of mandibular condylar hyperplasia.[116] They proposed a classification system that is based on histologic criteria, and they divided hyperplastic condyles into four types, depending on the arrangement and morphology of the various layers of the condyle. These condylar layers were the fibrous articular layer; the undifferentiated mesenchyme proliferative layer; the transitional layer; and the hypertrophic cartilage layer.

Villanueva-Alcojol and colleagues attempted to correlate demographic and clinical presentations of condylar hyperplasia with the described histopathologic features and then with the associated scintigraphic and clinical findings after treatment with high condylectomy during the active phase.[122] Interestingly, the authors were unable to find a correlation between the histologic type (as described by Slootweg and Muller[116]) and the uptake of the affected condyle on bone single photon emission computed tomography or between the patient's age and his or her histologic type.

The preferred treatment to correct the dysmorphology for all forms of condylar hyperplasia is surgical including the repositioning and recontouring of the bones in combination with orthodontics to align the teeth.* The extent of

*References 8, 17, 26, 31, 33, 37, 39, 52-54, 56, 59, 61, 96, 99, 102, 103, 111, 115, 118, 119, 121, 126

treatment depends on the presenting dysmorphology and the status of condylar growth. If ongoing condylar growth is documented, then a form of high condylectomy should be considered to arrest progressive dysmorphology.[20,21,40,42] High condylectomy (i.e., the removal of 4 mm to 5 mm of the condyle) and condylar shaving procedures (i.e., the removal of only 2 mm to 3 mm of the condyle) are options that are discussed in the literature. The documentation of cartilage islands that extend into the cancellous bone suggests that the pathology is not limited to the apparent cartilage surface. For this reason, some clinicians prefer the high condylectomy procedure, because it will also remove subcondylar bone; in theory, it will also more fully eliminate the potential of the growth center.

Hemimandibular Hyperplasia: Type I Condylar Hyperactivity

Type I hemimandibular hyperplasia is a dentofacial deformity that results in excessive growth in volume of the whole hemimandible. With this condition, there is three-dimensional enlargement of one side of the mandible, and this includes all aspects: the condyle, the condylar neck, the ramus, the body, and the symphysis. The anomaly terminates exactly at the symphysis of the mandible. In its pure form, this condition does not result in a significant shifting of the midline of the chin or of the mandibular dental midline to the contralateral side. The occlusion is rotated, but there is minimal to no crossbite. There is an increase in vertical height, but it does not involve widening of the affected side of the face. This three-dimensional enlargement of the mandible results in a notable asymmetric rotation of the face. There is visually apparent ipsilateral downward and medial projection of the ramus as well as of the angle and body of the mandible.

When the anomalous process begins before puberty, there will be more severe and characteristic secondary deformities of the maxilla, the contralateral mandible, and the positioning of the teeth. The secondary maxillary deformities follow the downward and medial growth of the ipsilateral mandible, and the occlusal plane becomes canted. The ipsilateral increase in vertical height on the affected side leads to an apparent decrease in facial height on the "normal" (contralateral) side. To the uninitiated, the facial appearance may be confused with Hemifacial Microsomia.

The natural history of this pure form of hemimandibular hyperplasia (Type I) is to progress and result in three-dimensional disproportion and asymmetry on both sides of the maxilla, the mandible, and the chin region. Fortunately, this form of asymmetric mandibular excess is rare as compared with hemimandibular elongation (Type II), which results in a less severe three-dimensional clinical deformity.

When hemimandibular hyperplasia is recognized in its active and progressive state, Obwegeser recommends a condylar shave to arrest the disease process and to prevent further progression.[23-25,68-91] Another reason why some clinicians prefer the partial condylectomy procedure is that it can be used as a method to shorten the posterior facial height. When the phenotype is characterized by primary vertical overgrowth with minimal to no horizontal deviation of the mandible, it can simultaneously be an effective way to restore the morphology. If the deformity remains active and is allowed to reach its endpoint, then the restoration of ideal facial aesthetics is problematic. The objectives of the *condylar shave procedure* are to arrest any further abnormal growth on the affected side; to prevent continued secondary deformities of the maxilla, the nose, and the contralateral side of the mandible; and to arrest any additional malpositioning of the teeth. The problem with this approach is that secondary procedures to correct the facial skeleton are still required in most cases. It remains unclear just how much of a deformity will be aborted by an open-joint procedure.

Practically speaking, however, a condylar procedure (e.g., condylar shave, high condylectomy) is not generally required, because growth has usually ceased by the time of diagnosis and surgical referral. This can be surmised by a review of the patient's previous records (e.g., radiographs, facial and occlusal photographs, dental models), or it can be confirmed by a bone scan. Definitive treatment of the dentofacial deformity through orthognathic and recontouring surgery is the usual approach once growth maturity and condylar arrest have occurred.

Functional Aspects

- Articulation errors in speech may result if a significant malocclusion is present (see Chapter 8).
- Predictable chewing and swallowing difficulties will result if a significant posterior open-bite malocclusion is present (see Chapter 8).
- The ability to comfortably close the lips together and their position at rest may be negatively affected depending on the extent of jaw malposition and dental malocclusion.
- Depending on the extent of deformity, the individual with asymmetric mandibular excess is likely to experience self-esteem and body-image issues (see Chapter 7).
- Nasal airway obstruction as a result of a deviated septum and turbinate enlargement with increased nasal airway resistance may be present and should be ruled out (see Chapter 10).
- Detrimental long-term effects on the dentition and the periodontium caused by traumatic malocclusion, the displacement of teeth outside of the alveolar housing, or previous camouflage orthodontic treatment may occur (see Chapter 6).
- A higher-than-average incidence of TMD has been reported in this form of dentofacial deformity (see Chapter 9).

Clinical Characteristics

- This deformity should not be confused with hemifacial microsomia, because the soft-tissue envelope (including

the external ear) is not hypoplastic on the affected side of the face.
- There is increased vertical height of the affected (ipsilateral) side of the face.
- The medial and downward shift of the ipsilateral mandible is most notable at the angle.
- There is a relative lateral and upward position of the contralateral mandible.
- The overall face has a "rotated" appearance.
- Mouth opening is without restriction.
- The ipsilateral corner of mouth is inferior but not posteriorly positioned.
- The cheek appears flat on the ipsilateral side.
- The width of the face on the ipsilateral side appears to be decreased.
- The maxilla is generally canted down on the ipsilateral side.

Dental Findings

- The crowns of the teeth on the contralateral side of the mandible tilt toward the ipsilateral side. They do so as a dental compensation to maintain occlusion with the opposing maxillary teeth, thereby avoiding crossbite.
- Lingual tilting of the mandibular alveolar ridge on the contralateral side is a secondary deformation as the (contralateral) lower jaw shifts lateral and assumes a superior position.
- The dental arches appear "twisted" by the slow process of three-dimensional growth distortion of the ipsilateral mandibular and the secondary deformations that occur in the contralateral side of the mandible and whole maxilla.
- If the ipsilateral excess mandibular growth is rapid, then the maxilla cannot respond at the same rate; this results in an ipsilateral posterior open bite.
- If the rate of ipsilateral excess mandibular growth is relatively slow, then maxillary dental hypereruption with secondary canting and the maintenance of occlusion are more likely.
- In most cases, the maxillary and mandibular dental midlines will closely match.

Radiographic Findings

- There is three-dimensional enlargement of the ipsilateral mandible, which terminates at the symphysis. The enlarged (ipsilateral) condyle also becomes "classically" deformed.
- The overall height of the body of the mandible is greater on the ipsilateral side.
- The inferior border and angle of the mandible are rounded on the ipsilateral side.
- Increased alveolar height between the inferior border and the root apices on the ipsilateral side is expected.
- The mandibular canal will be displaced downward on the ipsilateral side, often in close proximity to the inferior border.

Hemimandibular Elongation: Type II Condylar Hyperactivity

The dentofacial deformity called Type II *hemimandibular elongation* results in excessive growth on one side of the mandible without an overall three-dimensional increase in the volume of the whole affected side as seen with a Type I condition (i.e., hemimandibular hyperplasia; see Figs. 22-6 through 22-15). At the same time, hemimandibular elongation is not limited to just one aspect of the mandible. It results in a relative elongation (i.e. lateral shift) of all components of the ipsilateral lower jaw, including the condyle, the condylar neck, the ascending ramus, and the body. The condylar neck will be elongated and narrow, but the condylar head (i.e., the articulation of the temporomandibular joint [TMJ]) appears to be relatively normal in size and shape.

Obwegeser recognizes two distinct variations within the Type II hemimandibular elongation form of condylar hyperactivity.[23,24,25,68-91] In the first subgroup (i.e., the *"slender type"*), there is an elongated hemimandible on the affected side as well as a morphologic "stretching" of the angle of the mandible. The distinguishing morphologic feature is an obtuse gonial angle as compared with the contralateral side. The second subgroup (i.e., the *"non-slender type"*) has an angle of the mandible that looks very much like the "normal" contralateral side and yet the whole (ipsilateral) mandible is clearly elongated.

The clinical features that are always present with Type II hemimandibular elongation—and therefore useful findings that differentiate it from Type I hemimandibular hyperplasia—include a visual lateral shift of the chin midline and the mandibular dental midline to the contralateral side; a posterior crossbite on the contralateral side; and no significant increase in the posterior facial height from side to side. The most striking features of Type II hemimandibular elongation include the deviation of the chin and the mandibular dental midline to the opposite side with a posterior crossbite on the contralateral side and a class III molar relationship on the ipsilateral side. As stated, there will be no significant difference in the posterior facial height between the two sides of the face, but canting that has been recognized in the maxilla and mandible is likely to be present and will benefit from surgical correction.

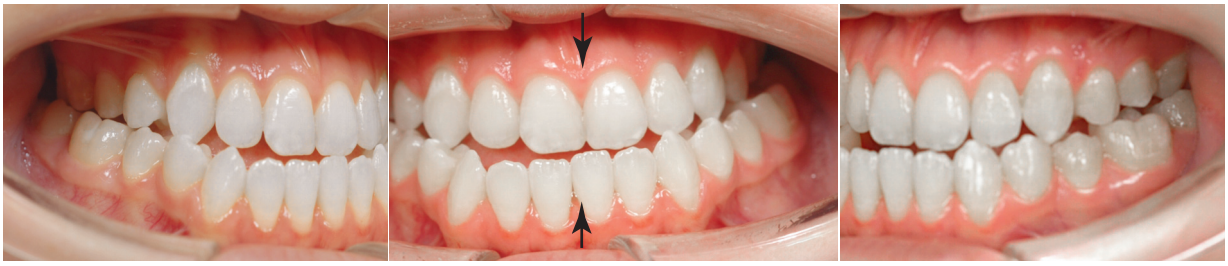
To review, *hemimandibular elongation* is the preferred term for what has classically been referred to as "asymmetric mandibular excess." Interestingly, the deformity has usually progressed and done its damage by the time of clinical presentation to the surgeon. For this reason, a condylar shave is rarely warranted. In general, these deformities can be reliably managed with standard orthognathic techniques carried out at skeletal maturity in conjunction with effective

©Elsevier Inc.
Orthognathic Surgery; J.C. Posnick

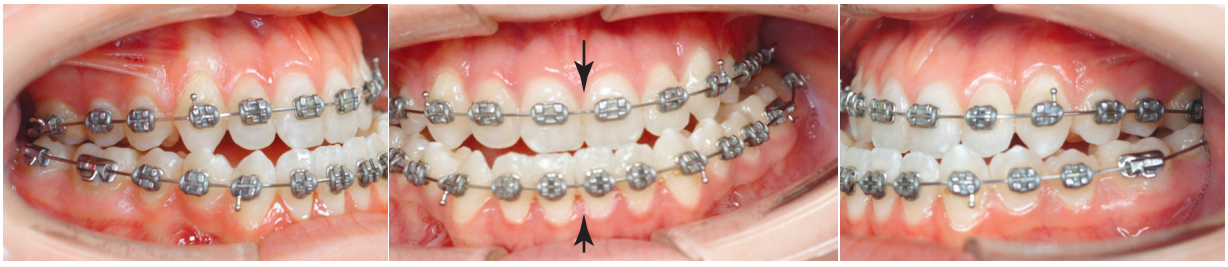
• **Figure 22-15** A 21-year-old woman with a diagnosis of left hemimandibular elongation; this affects her chewing and speech articulation. She also has a lifelong history of obstructed nasal breathing. During her early teenage years, the patient underwent an initial phase of orthodontics, without success. She was referred for surgical evaluation and then underwent a combined redo orthodontic and surgical approach. With the relief of dental compensation, the patient's surgery included maxillary Le Fort I osteotomy in segments (horizontal advancement, cant correction, arch expansion, and clockwise rotation); bilateral sagittal split ramus osteotomies (cant correction and mandibular straightening); septoplasty and inferior turbinate reduction; and osseous genioplasty (straightening). **A,** Frontal views in repose before and after reconstruction. **B,** Frontal views with smile before and after reconstruction.

• **Figure 22-15, cont'd C,** Oblique facial views before and after reconstruction. **D,** Profile views before and after reconstruction.

Continued



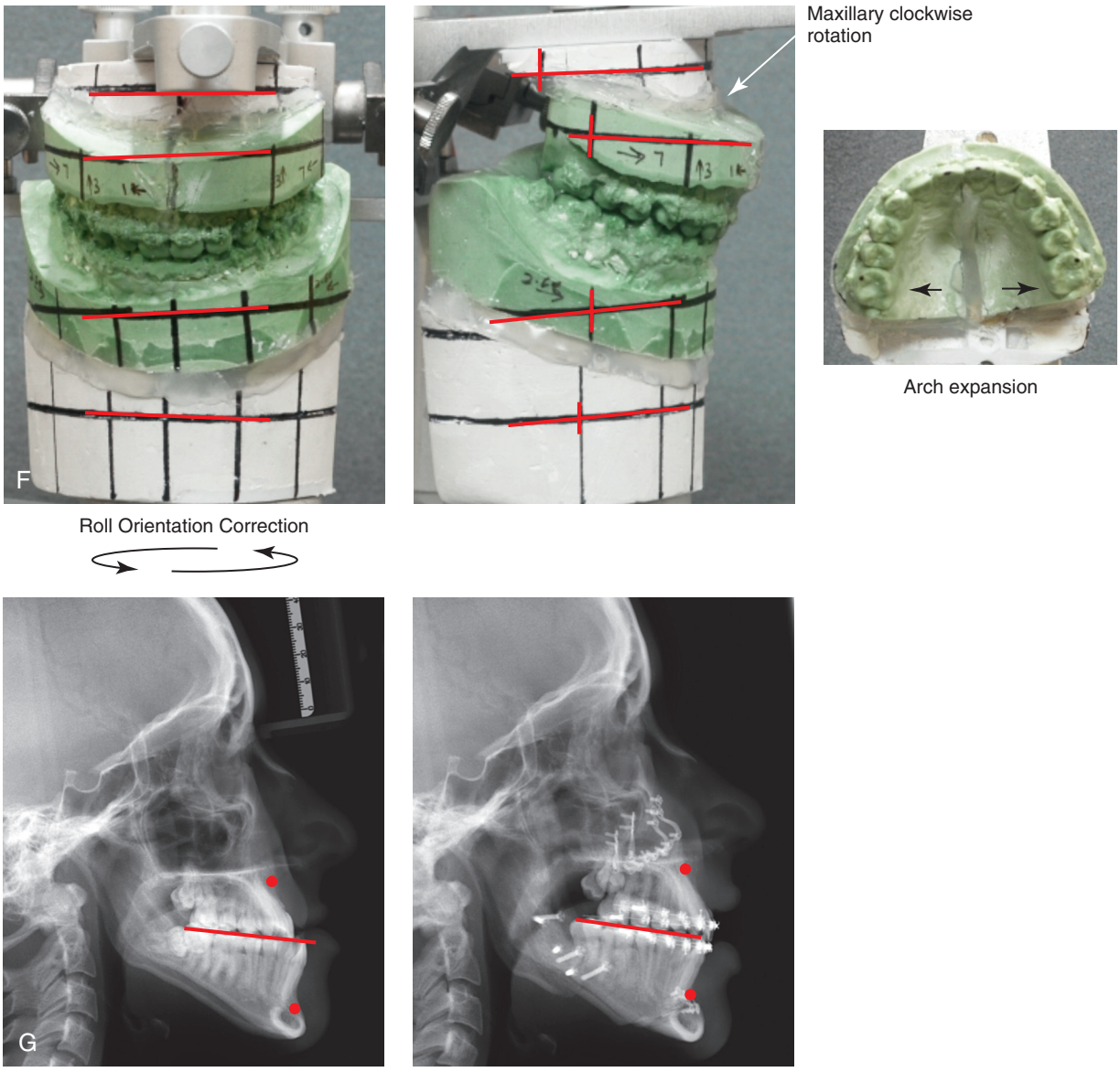
Prior to retreatment



Pre surgery

After treatment

• **Figure 22-15, cont'd E,** Occlusal views before retreatment, with orthodontic decompensation, and after treatment.



• **Figure 22-15, cont'd F,** Articulated dental casts that indicate analytic model planning. **G,** Lateral cephalometric radiographs before and after treatment. Note that the clockwise rotation of the maxilla was helpful to improve the A-point to B-point relationship as viewed in profile.

orthodontics to improve head and neck function and to enhance facial aesthetics (Fig. 22-6 through Fig. 22-14).

Functional Aspects

- Articulation errors in speech may result if a significant malocclusion is present (see Chapter 8).
- Chewing and swallowing difficulties resulting from the presenting malocclusion are typical (see Chapter 8).
- The ability to comfortably close the lips together and their position at rest will be negatively affected by the extent of jaw malposition and dental malocclusion.
- The individual with this jaw growth pattern is likely to experience self-esteem and body image issues directly related to the presenting facial asymmetry (see Chapter 7).
- Nasal airway obstruction as a result of a deviated septum and enlarged inferior turbinates with increased nasal airway resistance is likely present and should be ruled out (see Chapter 10).
- Detrimental long-term effects on the dentition and periodontium as a result of traumatic malocclusion, the displacement of the teeth outside of the alveolar housing, or previous camouflage orthodontic treatment may occur (see chapter 5 & 6).
- A higher-than-average incidence of temporomandibular disorder (TMD) has been reported with this anomalous pattern of dentofacial growth. Joint noise and

discomfort on the ipsilateral side are not infrequent (see Chapter 9).

Clinical Characteristics

- The pogonion of the mandible and soft-tissue chin prominence are shifted to the contralateral side.
- The lower lip is in front of (i.e., anterior to) the upper lip; this is the reverse of normal.
- Vertical mouth opening is usually undisturbed.
- The ipsilateral corner of the mouth is usually lower and more anterior than the contralateral side (i.e., canting is present).
- The profile line may be harmonious, but it more likely gives the impression of mandibular prognathism.
- The soft-tissue cheek region appears fuller on the contralateral side.
- The prominence of the cheekbone appears to be deficient on the ipsilateral side.
- There is no apparent vertical height difference between the two sides of the face.

Dental Findings

- The ipsilateral molars are in Class III malocclusion.
- The contralateral molars may be in Class I or Class II malocclusion.
- A posterior crossbite on the contralateral side is typical.
- The mandibular dental midline is shifted to the contralateral side.
- Tilting of the lower anterior teeth with the incisal surfaces toward the ipsilateral side is typical.
- Retroinclination of the lower incisors is generally seen.
- Lingual inclination of the contralateral mandibular posterior teeth is typical.
- The maxilla is often canted downward and forward on the ipsilateral side.

Radiographic Findings

- Ascending ramus asymmetry from side to side is frequent.
- A thick cortical plate along the inferior border of the mandible on the ipsilateral side is frequent.
- The ipsilateral mandibular canal may appear to be lower positioned (i.e., closer to the inferior border), better defined (i.e., encased in cortical bone), and in close proximity to the roots of the teeth as compared with the contralateral side.
- The overall visual appearance and the measurable length of the mandible indicate elongation on the ipsilateral side.
- The ipsilateral angle of the mandible may be more obtuse (i.e., slender) as compared with the normal (contralateral) side.
- The ipsilateral condylar neck will be long, but the diameter is typically not increased.
- When viewed on a posteroanterior cephalometric radiograph, canting of the maxillary occlusal plane may be seen (i.e., downward and forward on the ipsilateral side).
- When viewed on a posteroanterior cephalometric radiograph, canting of mandibular plane is typically seen (i.e., downward and forward on the ipsilateral side).

Assessing Jaw Growth Maturity

Skeletal scintigraphy with a technetium bone scan is a useful method for the assessment of difficult growth-related diagnostic problems of the mandibular condyle.[3,29,34,35,57,63,92,95,104] For patients with asymmetric mandibular excess, the state of residual growth may be clarified by the unequal ratio of uptake of technetium in each condylar head. Alternatively, an equal ratio of uptake shows that asymmetric growth has likely ceased. In difficult diagnostic cases, normal versus abnormal condylar growth can be further assessed by comparing the ratio of the uptake with the tabulated normative standards developed by Kaban and colleagues.[18,19,43-46] The cessation of mandibular growth can be more or less predicted when the uptake ratios approach 0.70, which is the adult norm. Skeletal scintigraphy (i.e., bone scan) may be useful during the making of decisions that involve the timing and extent of treatment when residual condylar growth remains uncertain. After the cessation of growth, there will be no advantage to an open-joint procedure. In this case, the correction of the dentofacial deformity with the use of standard orthognathic techniques is indicated.[5,107-109]

High Partial Condylectomy Procedure for Active Condylar Hyperactivity

The successful treatment of asymmetric mandibular excess requires the cessation of growth before definitive surgical correction. Knowledge of whether the condition is still active is assessed at the condylar site.[5,107-109] For the patient with *hemimandibular elongation,* the deformity has usually progressed and done its damage by the time of clinical presentation to the surgeon. In general, the presenting jaw deformities are best managed with standard orthognathic techniques that are carried out at skeletal maturity to improve both head and neck function and to enhance facial aesthetics. Alternatively, the *hemimandibular hyperplasia* deformity may be more gradual in its progression. In this case, if the condition is allowed to reach its endpoint, it may not be possible to achieve an ideal symmetric facial aesthetic result. A condylar shave procedure may be beneficial for the subgroup of patients with Type I hemimandibular hyperplasia to arrest ongoing abnormal growth on the affected

side; to prevent further secondary deformities of the maxilla, the nose, and the contralateral side of the mandible; and to arrest the malpositioning of the teeth.[20,21,40,42]

Some surgeons are relatively quick to suggest a high condylectomy or a condylar shave, because they feel that little harm will be done and that such a procedure has the potential to limit the extent of orthognathic surgery that will be required. Most surgeons remain hesitant when it comes to performing a direct condylar procedure, because they recognize the law of unintended consequences and the potential for the deterioration of TMJ function. In general, I agree with this conservative approach when considering an open condylar procedure.

Lippold and colleagues as well as other researchers have suggested that minimal harm occurs from a "high condylar shave."[51] Until recently, critical reviews of an invasive TMJ approach have not been forthcoming. Saridin and colleagues completed an evaluation of temporomandibular function after high partial condylectomy in a consecutive series of individuals with unilateral condylar hyperplasia (N = 33).[110] The study group included individuals with unilateral condylar hyperplasia whose conditions were considered to be active; in other words, the mandibular asymmetry appeared to be clinically progressive, and the bone scan was positive. The subjects then underwent high condylectomies (N = 33). The patients were compared with a control group that was matched for age and gender (N = 31). All patients and all members of the control group filled out a history questionnaire and underwent a clinical examination in accordance with the research diagnostic criteria for temporomandibular disorders. The patients were studied for an average of 4.3 years after the condylectomy was carried out (range, 0.6 to 13.3 years). The results of the study indicate that patients with unilateral condylar hyperplasia who undergo condylectomy in an attempt to arrest progressive mandibular asymmetry will have more joint-related temporomandibular problems and a higher persistence of long-term pain as compared with age- and gender-matched controls.

Presurgical Orthodontic Treatment

Among patients who are affected by these mandibular excess growth patterns, orthodontic maneuvers that were carried out earlier during their lives to modify growth or as camouflage approaches to neutralize the occlusion should be overcome with further orthodontic mechanics before orthognathic surgery. Frequently seen scenarios include the following: (1) orthodontic overexpansion of the maxillary arch to correct crossbites; (2) hypereruption of the molars on one side and intrusion on the other side to correct canting; (3) lingual tipping of the mandibular molar or premolar crowns on the contralateral side and buccal tipping of the molar or premolar crowns on the ipsilateral side; and (4) retroclining of the lower anterior teeth to correct the overbite. All of these orthodontic techniques may have been tried with the hope of neutralizing the occlusion and avoiding surgery. These camouflage orthodontic mechanics hamper the preferred surgical positioning of the jaws to achieve maximum facial harmony, and they will also leave the occlusion prone to late relapse and the dentition susceptible to long-term periodontal breakdown (see Chapter 5).

In preparation for an orthognathic correction, the orthodontic treatment of each jaw should achieve the correct positioning of the teeth within the respective alveolar process (see Chapter 5). If this is accomplished, the clinicians can best achieve maximum mirror-image skeletal symmetry, Euclidian facial proportions, and a long-term stable occlusion with periodontal health (see Chapter 6).

Surgical Approach

The Influence of Chin Asymmetry on Perceived Facial Attractiveness

Naini and colleagues undertook an investigation with the objective of assessing how severe an asymmetry at the chin point would have to be to influence perceived attractiveness.[64] Idealized two-dimensional frontal facial views were created with the use of computer software in which the chin point was altered from side to side in 5-mm increments from 0 mm to 25 mm. These images were then rated on a seven-point Likert scale by a preselected group of observers that included orthognathic patients before treatment, clinicians, and laypeople (n = 185 observers). The study confirmed that, in the frontal view, chin asymmetries of 10 mm or greater are perceived as being significant, whereas those of 5 mm or less largely go unnoticed. The greater the degree of asymmetry (i.e., >10 mm), the more noticed it will be by an observer and the greater the patient's desire for correction. Both clinician and patient ratings were similar and found to be more critical than the ratings given by laypeople. The images that were considered the most attractive showed perfect bilateral symmetry of the chin region. The lowest-rated images showed the highest degrees of asymmetry of the chin. Combined vertical and horizontal asymmetries were rated slightly worse than horizontal asymmetry alone.

Hemimandibular Hyperplasia (Type I)

If the *hemimandibular hyperplasia deformity* remains active and is allowed to progress to its endpoint, it may not be possible to achieve an ideal symmetric facial aesthetic result. For this reason, a high condylar shave to arrest further abnormal growth on the affected side and to prevent ongoing secondary deformities (i.e., of the maxilla, nose, and contralateral side of the mandible; malpositioning of the teeth) should be considered if condylar hyperactivity remains, as described previously.[20,21,40,42]

Details of the definitive maxillofacial reconstruction will depend on the patient-specific morphologic findings (see

Figs. 22-2 through 22-5).[30,32,97,98,105] A wide spectrum of three-dimensional maxillofacial distortions caused by the primary anomaly and the secondary deformities is expected. In addition to standard techniques (i.e., Le Fort I osteotomy in segments, sagittal split ramus osteotomies of the mandible, osteotomy of the chin), mandibular inferior border recontouring will often be beneficial (see Chapter 12). The recontouring includes inferior border resection and rotary drill bur modification from the angle of the mandible to the chin on the ipsilateral side, as indicated. Inferior border augmentation on the contralateral side is not usually recommended because, over time, visible irregularities on the grafted side can easily occur after partial graft resorption. Segmentation of the maxilla (i.e., into two or three pieces) is also generally required to correct the cant and the arch form.

Hemimandibular Elongation (Type II)

The *deformities of hemimandibular elongation* are best managed with standard orthognathic techniques carried out at skeletal maturity to improve both head and neck function and to enhance facial aesthetics (see Figs. 22-6 through 22-14). This will include, at a minimum, sagittal split ramus osteotomies of the mandible. If the maxilla is secondarily deformed (i.e., canted, with a shift of the dental midline and an altered arch form), a Le Fort I osteotomy with three-dimensional repositioning is also required. An oblique osteotomy of the chin will likely further improve facial symmetry and proportions (see Chapter 12). The need for a high condylar shave to arrest growth remains limited, and this procedure is only considered in the presence of documented progressive active disease at the time of diagnosis.

Complications, Informed Consent, and Patient Education

Orthodontists and surgeons are frequently asked to evaluate jaw disharmonies and malpositioned teeth and then to attempt to alter nature's events. With an understanding of the natural progression of the uncorrected asymmetric mandibular excess growth anomaly and with knowledge of how specific interventions will affect head and neck function and facial aesthetics, predictable results are generally achieved. Complications and patient dissatisfaction will always occur in a small percentage of individuals. In the setting of asymmetric mandibular excess, obtaining the desired occlusion is more or less predictable. The expected outcome is an improvement in the facial asymmetry with persistent minor discrepancies. The clinician's ability to communicate the potential risks, complications, alternative approaches, and expected results of the recommended treatment to the patient and the patient's family is essential to the achievement of a favorable outcome. When the patient, the family, and all treating clinicians are fully informed, shared responsibility for the treatment result is more likely (see Chapter 16).

Malocclusion after Orthodontics and Orthognathic Surgery

There are inherent biologic challenges to the orthodontic movement of the teeth within the alveolar process and to the surgical repositioning of the jaws or jaw segments in the individual with an asymmetric mandibular excess growth anomaly. This includes the technical aspects of the repositioning, the initial healing requirements, and the difficulties associated with the long-term maintenance of the mechanically relocated teeth, jaws, and jaw segments. When following sound biomechanical and aesthetic principles, clinicians carry out orthodontic treatments and surgical procedures; in most cases, the results are effectively maintained. Potential intraoperative technical difficulties associated with sagittal split ramus osteotomies of the mandible and maxillary Le Fort I osteotomy can result in immediate early malocclusion, but these problems are generally preventable (see Chapter 17). In the teenager, timing surgery to limit concern about ongoing mandibular growth afterward is more problematic with asymmetric mandibular excess than with most other patterns of dentofacial deformity; this is discussed in more detail earlier in this chapter. All of these factors must be taken into account to achieve and then maintain the desired results.

Temporomandibular Disorders: The Effects of Orthodontics and Orthognathic Surgery

It is estimated that an average of 32% of the population report at least one symptom of TMD, whereas an average of 55% demonstrate at least one clinical sign. TMD includes various symptoms and signs of the TMJs, the masticatory muscles, and the related structures. The symptoms and signs may include a spectrum of referred head and neck pain; joint noise (e.g., popping, clicking, crepitus); reduced or altered mandibular movements caused by muscle spasm or disc displacement with or without reduction; condylar head erosion; and pain on direct palpation of either of the TMJs or the masticatory muscles. Occlusal factors (i.e., degrees of malocclusion) are often claimed to be associated with TMD. Asymmetric mandibular excess is associated with TMD more frequently than many other dentofacial deformities. The correction of the presenting jaw deformity and malocclusion through effective orthodontics and orthognathic surgery may relieve symptoms and signs of TMD in some patients, but this is not predictable for each individual patient. As stated earlier in this chapter, Saridin and colleagues found that the completion of a partial condylectomy on the ipsilateral side was associated with a higher incidence of TMD (see Chapter 9).[110] This should give the

clinician pause before he or she agrees to perform a condylectomy procedure.

Conclusions

Obwegeser believes that *asymmetric mandibular excess growth anomalies* are caused by two different growth regulators that are located entirely within the cartilaginous bony surface of the condylar head. This results in two different basic patterns of clinical presentation. The first, *hemimandibular hyperplasia,* is a more rare form that is characterized by an increase in the volume of all parts of the affected side of the mandible, with secondary deformities of all adjacent skeletal structures. If hemimandibular hyperplasia remains active and is allowed to reach its end deformity, the achievement of ideal facial symmetry is less likely. For this reason, if this condition is diagnosed during its active phase, a high condylar shave maybe advisable to prevent its progression. The second form of presentation, *hemimandibular elongation,* is more common; it is characterized by the elongation of one side of the mandible, and it results in the displacement of the midline of the chin and the dentition to the contralateral side. In most cases, hemimandibular elongation is best managed with standard orthognathic techniques carried out at skeletal maturity to improve both head and neck function and to enhance facial aesthetics.

References

1. Adams SS: A case of hemihypertrophy (giant growth). *Arch Pediatr* 2:901, 1894.
2. Angle EH: *Treatment of malocclusion of the teeth: Angle's system*, ed 7, Philadelphia, 1907, SS White.
3. Beirne OR, Leake DL: Technetium-99m pyrophosphate uptake in a case of unilateral condylar hyperplasia. *J Oral Surg* 38:385, 1980.
4. Benoist M, LePesteur J: Considerations sure le bilan preoperatoire des laterognathies mandibulaires. *Rev Stomatol* 77:96, 1976.
5. Björk A: Variations in the growth pattern of the human mandible: Longitudinal radiographic study by the implant method. *J Dent Res Suppl* 42:400–411, 1963.
6. Bloomquist K, Hogeman KE: Benign unilateral hyperplasia of mandibular condyle: Report of eight cases. *Acta Chir Scand* 126:414, 1963.
7. de Bont LG, Blankestijn J, Panders AK, Vermey A: Unilateral condylar hyperplasia combined with synovial chondromatosis of the temporomandibular joint: Report of a case. *J Maxillofac Surg* 13:32–36, 1985.
8. Brami S, Lamarche JP, Souyris F: Treatment of facial asymmetries by one-stage maxillary and mandibular bilateral osteotomies. *Int J Oral Surg* 3:239, 1974.
9. Bruce RA, Hayward JR: Condylar hyperplasia and mandibular asymmetry: A review. *J Oral Surg* 26:281–290, 1968.
10. Buchner A, David R, Temkin D: Unilateral enlargement of the masseter muscle. *Int J Oral Surg* 8:140, 1979.
11. Burchfield D, Escobar V: Familial facial asymmetry (autosomal dominant hemihypertrophy?) *Oral Surg Oral Med Oral Pathol* 50:321, 1980.
12. Norman JE, Painter DM: Hyperplasia of the mandibular condyle. A historical review of important early cases with a presentation and analysis of twelve patients. *J Maxillofac Surg* 8:161–175, 1980.
13. Cernea P: Unilateral hypertrophy of the mandibular condyle. *Trans Int Conf Oral Surg* 255–257, 1967.
14. Chen YR, Bendor-Samuel RL, Huang C-S: Hemimandibular hyperplasia. *Plast Reconstr Surg* 97:730, 1996.
15. Chen YR, Lo LJ, Kyutoku S, et al: Facial midline and symmetry: Modified face bow. *Plast Reconstr Surg* 90:126, 1992.
16. Chierici G, Harvold EP, Dawson WJ: Primate experiments on facial asymmetry. *J Dent Res* 49:847, 1970.
17. Choung PH: Surgical correction of asymmetric mandibular excess. *Taehan Chikkwa Uisa Hyophoe Chi* 23:1057, 1985.
18. Cisneros GJ: The use of bone seeking radiopharmaceutical uptake in the assessment of facial growth and development [thesis], 1982, Harvard University.
19. Cisneros GJ, Kaban LB: Computerized skeletal scintigraphy for assessment of mandibular asymmetry. *J Oral Maxillofac Surg* 42:513–520, 1984.
20. Delaire J, Gaillard A, Tulasne JF: La place de la condylectomie dans le traitment des hypercondyles. *Rev Stomatol Chir Maxillofac* 84:11–18, 1983.
21. Delaire J: Le role du condyle la croissance de la machoire inferieur et dans l'equilibie de la face. *Rev Stomatol Chir Maxillofac* 91:179–192, 1990.
22. Dingman RO, Grabb WC: Mandibular laterognathism. *Plast Reconstr Surg* 31:563, 1963.
23. Egyedi P, Obwegeser H: Zur operativen zungenverkleinerung. *Dtsch Zahn Mund Kiefrehlkd* 41:16–25, 1964.
24. Egyedi P: Etiology of condylar hyperplasia. *Aust Dent J* 14:12–17, 1969.
25. Egyedi P: Hyperplasia van de condylus mandibulae. *Ned Tijdschr Geneeskd* 118:1593, 1974.
26. Ferguson JW: Definitive surgical correction of the deformity resulting from hemimandibular hyperplasia. *J Craniomaxillofac Surg* 33:150–157, 2005.
27. Forsberg CT, Burstone CJ, Hanley KJ: Diagnosis and treatment planning of skeletal asymmetry with the submental-vertical radiograph. *Am J Orthod Dentofacial Orthop* 85:224, 1984.
28. Gordeeff A, Mercier J, Delaire J: L'hypercondyle mandibulaire. Ses different aspects cliniques et son traitement. *Acta Stomatol Belg* 85:269–276, 1988.
29. Gray RJM, Sloan P, Quale AA, Carter DH: Histopathological and scintigraphic features of condylar hyperplasia. *Int J Oral Maxillofac Surg* 19:65–71, 1990.
30. Grummons DC, Kappeyne van de Coppello MA: A frontal asymmetry analysis. *J Clin Orthod* 21:448, 1987.
31. Grunert J, Krenhal CH, Furtenbach M: Vergleich myofuntioneller befunde bei patienten mit und ohne rezidiv nach progenie-operationen. *Z Stomatol* 87:261–271, 1990.
32. Hall HD: An improved method for treatment of facial asymmetry secondary to jaw deformity. *J Oral Maxillofac Surg* 42:673, 1984.
33. Hampf GA, Tasanen A, Nordling S: Surgery in mandibular condylar hyperplasia. *J Maxillofac Surg* 13:74–78, 1985.
34. Harris SA, Quayle AA, Testa HJ: Radionuclide bone scanning in the diagnosis and management of condylar hyperplasia. *Nucl Med Commun* 5:373, 1984.
35. Henderson MJ, Wastie ML, Bromige M, et al: Technetium-99m bone scintigraphy and mandibular condylar hyperplasia. *Clin Radiol* 41:411, 1990.
36. Hinds ED, Reid LC, Burch RJ: Classification and management of mandibular asymmetry. *Am J Surg* 100:825, 1960.
37. Hovinga J, Kraal ER, Roorda LAM: Difficulties in and indications for the treatment of facial asymmetry. *Int J Oral Surg* 3:234, 1974.
38. Hyckel P, Erler U, Muller P: Unilateral hyperplasia of the TMJ condyle [German]. *Dtsch Z Mund Kiefer Gesichtschir* 15:42–50, 1991.
39. Jacobs FJ, Rafel SS, Weiss B: Correction of asymmetric mandibular prognathism by unilateral osteotomy. *J Oral Surg* 11:42, 1953.

40. Jaquemaire D, Delaire J: L'hypercondylie mandibulaire. *Rev Stomatol Chir Maxillofac* 84:5–10, 1983.
41. Jonck LM: Facial asymmetry and condylar hyperplasia. *J Oral Surg* 40:567, 1975.
42. Jonck LM: Condylar hyperplasia: A case for early treatment. *Int J Oral Surg* 10:154, 1981.
43. Kaban LB, Cisneros GJ, Heymann S, Treves ST: Assessment of mandibular growth by skeletal scintigraphy. *J Oral Maxillofac Surg* 40:18–22, 1982.
44. Kaban LB: Skeletal scintigraphy for assessment of mandibular growth and asymmetry. In Treves ST, editor: *Pediatric nuclear medicine*, New York, 1985, Springer-Verlag, p 49.
45. Kaban LB: Congenital and acquired growth abnormalities of the temporomandibular joint. In Keith DA, editor: *Surgery of the temporomandibular joint*, London, 1992, Blackwell Scientific, pp 84–115.
46. Kaban LB, Treves ST, Pogrel MA, Hattner RS: Skeletal scintigraphy for assessment of mandibular growth and asymmetry. In Treves ST, editor: *Pediatric nuclear medicine*, ed 2, New York, Berlin, Heidelberg, 1995, Springer, pp 316–327.
47. Kaneda T, Torii S, Yamashita T, et al: Giant osteochondroma of the mandibular condyle. *J Oral Maxillofac Surg* 40:818, 1982.
48. Keller E, Jackson IT, Marsh WR, et al: Mandibular asymmetry associated with congenital muscular torticollis. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 61:216, 1986.
49. Kessel LJ: Condylar hyperplasia: A case report. *Br J Oral Surg* 7:124, 1969
50. Lineaweaver W, Vargervik K, Tomer BS, Ousterhout DK: Posttraumatic condylar hyperplasia. *Ann Plast Surg* 22:163–172, 1989.
51. Lippold C, Kruse-Losler B, Danesh G, et al: Treatment of hemimandibular hyperplasia: The biological basis of condylectomy. *Br J Oral Maxillofac Surg* 45:353–360, 2007.
52. Luhr HG: Zur stabilen osteosynthese bei unterkiefer–frakturen. *Dtsch Zahnarztl Z* 23:754, 1968.
53. Luhr HG: Skelettverlagernde operationen zur harmonisierung des gesichtsprofils—probleme der stabilen fixation von osteotomiesegmenten. In von Pfeifer G, editor: *Die asthetik von form und funktion in der plastischen und wiederherstellungschirurgie*, Berlin, 1985, Springer-Verlag, p 87.
54. MacIntosh RB: Treatment of condylar hyperplasia of the mandible using unilateral ramus osteotomies [discussion]. *J Oral Maxillofac Surg* 54:1169, 1996.
55. Magnusson T, Ahlborg G, Svartz K: Function of the masticatory system in 20 patients with mandibular hypo- or hyperplasia after correction by a sagittal split osteotomy. *Int J Oral Maxillofac Surg* 19:289, 1990.
56. Martis C, Raraboula I, Zazaridis N: Severe unilateral condylar hyperplasia corrected by modified sagittal split osteotomy: Report of a case. *J Oral Surg* 37:835, 1979.
57. Matteson SR, Proffit WR, Terry BC, et al: Bone scanning with 99 m technetium phosphate to assess condylar hyperplasia. Report of two cases. *Oral Surg Oral Med Oral Pathol* 60:356–367, 1985.
58. Mercier P: Mandibular asymmetry: Proposed classification and an analysis of two cases. *J Can Dent Assoc* 35:146–153, 1969.
59. Mizuno A, Motegi K: Treatment of an asymmetric mandibular prognathism in an acromegalic patient. *J Oral Maxillofac Surg* 46:314, 1988.
60. Mizuno A, Nakamura T, Motegi K, Shirasawa H: Osteochondroma of the mandibular condyle: Report of a case and review of the Japanese literature. *Int J Oral Surg* 12:221, 1983.
61. Motamedi MHK: Treatment of condylar hyperplasia of the mandible using unilateral ramus osteotomies. *J Oral Maxillofac Surg* 54:1161, 1996.
62. Müller H: Unilateral condylar hyperplasia and acromegaly. *J Maxillofac Surg* 7:73–76, 1979.
63. Murray IP, Ford JC: Tc-99m medronate scintigraphy in mandibular condylar hyperplasia. *Clin Nucl Med* 7:474, 1982.
64. Naini FB, Donaldson AA, McDonald F, et al: Assessing the influence of asymmetry affecting the mandible and chin point on perceived attractiveness in the orthognathic patient, clinician, and layperson. *J Oral Maxillofac Surg* 70:192–206, 2012.
65. Nitzan DW, Katsnelson A, Bermanis I, et al: The clinical characteristics of condylar hyperplasia: Experience with 61 patients. *J Oral Maxillofac Surg* 66(2):312–318, 2008.
66. Norman JE, Painter DM: Hyperplasia of the mandibular condyle: A historical review of important early cases with a presentation and analysis of twelve patients. *J Maxillofac Surg* 8:161, 1980.
67. Oberg T, Fajers CM, Lysell G, Friberg U: Unilateral hyperplasia of the mandibular condyle process. A histological, microradiographic and autoradiographic examination of one case. *Acta Odontol Scand* 20:485–504, 1962.
68. Obwegeser HL: Ueber eine einfache methode der freihandigen drahtschienung von kieferbruchen. *Oesterr Z Stomatol* 49:652–670, 1952.
69. Obwegeser HL: In Trauner R, Obwegeser H, editors: Zur operationstechnik bei der progenie and anderen underkieferanomalien. *Dtsch Zahn Mund Kieferhlkd* 23:11–26, 1955.
70. Obwegeser HL: Surgical procedures to correct mandibular prognathism and reshaping of the chin. In Trauner R, Obwegeser H, editors: The surgical correction of mandibular prognathism and retrognathia with consideration of genioplasty. *Oral Surg Oral Med Oral Pathol* 10:677–689, 1957.
71. Obwegeser HL: Die Kinnvergrosserung. *Oesterr Z Stomatol* 55:535–541, 1958.
72. Obwegeser HL: Cirugia del "modex apertus." *Rev Asoc Odont Argent* 50:430–441, 1962.
73. Obwegeser HL, Egyedi P: Zur operativen zungenverkleinerung. *Dtsch Zahn Mund Kieferhlkd* 41:16–25, 1964.
74. Obwegeser HL: Der offene biss in chirurgischer sicht. *Schweitz Monatschr Zahnhlkd* 74:668–687, 1964.
75. Obwegeser HL: Zur behandlung der veralteten oberkieferfracturen. *Fortschr Kiefer Gesichtschir* 12:126–131, 1967.
76. Obwegeser HL: Die bewegung des unteren alveolarforstatzes zur korrektur von kieferstellungsanomalien: Part 1. *Dtsch Z Zeitschrift* 23:1075–1084, 1968.
77. Obwegeser HL: Operative Behandlung der zahnlosen progenie ohne intermaxillare fixation. *Schweiz Monatschr Zahnhlkd* 78:416–425, 1968.
78. Obwegeser HL: Die bewegung des unteren alveolarforstatzes zur korrektur von kieferstellungsanomalien: Part 2. *Dtsch Z Zeitschrift* 24:5–15, 1969.
79. Obwegeser HL: Forstchritte und schwerpunkte der orthopadischen kiefer- und gesichtschirurgie. In Schuchardt K, Pfeifer G, editors: *Fortschritte der kiefer-u gesichtschirurgie*, Vol 21, Stuttgart, 1971, Thieme, pp 54–60.
80. Obwegeser HL: Mandibular surgery. *Br J Oral Surg* 10:251–253, 1973.
81. Obwegeser HL: Kieferstellungskorrekturen beim erwachsenen spaltpatienten. *Dtsch Z Zeitschrift* 28:688–694, 1973.
82. Obwegeser HL: Grundsatzliches zur korrekturplanung von kiefer- und gesichtsanomalien aus chirurgischer sicht. In Schuchardt K, Schwenzer N, editors: *Fortschr kiefer gesichtschir*, Vol 26, Stuttgart, 1981, Thieme, pp 9–12.
83. Obwegeser HL, Farmand M: Unsere behandlung der kiefergelenksankylose. *Schweiz Monatschr Zahnhlkd* 92:569–580, 1982.
84. Obwegeser HL, Maked MS: Hemimandibular hyperplasia/hemimandibular elongation. *J Maxillofac Surg* 14:183, 1986.
85. Obwegeser HL, Makek M: Hemimandibulare hyperplasie-hemimandibulare elongation. *Zahnnarztl Med Schriftum, Munchen* 18:273–313, 1986.
86. Obwegeser HL, Marentette L: Profile planning based on alterations in the position of the bases of the facial thirds. *J Oral Maxillofac Surg* 44:302–311, 1986.
87. Obwegeser HL, Mekek M: Hemimandibular hyperplasia-hemimandibular elongation. *J Maxillofac Surg* 14:183–208, 1986.
88. Obwegeser HL: Chirurgische behandlungsmoglichkeiten von sekundardeformierugen bei spaltpatienten. *Fortschr Kieferorthop* 49:272–296, 1988.
89. Obwegeser JA: Eine neue operationsmethode zur osteotomie des gesamten

unterkiefer-alveolarfortsatzes. *Osterr Z Stomal* 85:35–46, 1988.
90. Obwegeser HL: Descriptive terminology for jaw anomalies. *Oral Surg Oral Med Oral Pathol* 75:138–140, 1993.
91. Obwegeser HL: *Mandibular growth anomalies: Terminology-aetiology-diagnosis-treatment*, Berlin, Heidelberg, New York, 2001, Springer-Verlag.
92. Paulus G, Hirschfelder U, Festel H: Computergestutzte quantitative skelettszintigraphie bei der therapieplanung der condylaren hyperplasie. *Forstchr Kiefer Gesichtschir* 32:180–181, 1987.
93. Perko M: Hipertrofija masetera. *J Chir Maxillofac Plast* 4:35–37, 1963.
94. Persson M: Mandibular asymmetry of hereditary origin. *Am J Orthod* 63:1–11, 1973.
95. Pogrel MA: Quantitative assessment of isotope activity in the temporomandibular joint regions as a means of assessing unilateral condylar hypertrophy. *Oral Surg Oral Med Oral Pathol* 60:15–17, 1985.
96. Posnick JC: Asymmetic mandibular prognathism with or without maxillary deformity. In Posnick JC, editor: Craniofacial and maxillofacial surgery in children and young adults, Vol 40, Philadelphia, 2000, WB Saunders Co, pp 1057–1080.
97. Posnick JC, Fantuzzo J, Orchin J: Deliberate operative rotation of the maxillo-mandibular complex to alter the A-point to B-point relationship for enhanced facial esthetics. *J Oral Maxillofac Surg* 64:1687–1695, 2006.
98. Posnick JC, Fantuzzo JJ, Troost, T: Simultaneous intranasal procedures to improve chronic obstructive nasal breathing in patients undergoing maxillary (Le Fort I) osteotomy. *J Oral Maxillofac Surg* 65:2273–2281, 2007.
99. Posnick JC, Agnihotri N: Managing chronic nasal airway obstruction at the time of orthognathic surgery: A twofer. *J Oral Maxillofac Surg* 69:695–701, 2011.
100. Proffit WR, Fields HW, Moray LJ: Prevalence of malocclusion and orthodontic treatment need in the United States: estimates from the N-HANES III survey. *Int J Adult Orthodon Orthognath Surg* 13:97–106, 1998.
101. Raijmakers PG, Karssemakers LHE, Tuinzing DB: Female predominance and effect of gender on unilateral condylar hyperplasia: A review and meta-analysis. *J Oral Maxillofac Surg* 70:e72–e76, 2012.
102. Reyneke J, Masireol C: Condylar hyperplasia: An alternative method of treatment. *J Dent Assoc S Afr* 34:335, 1979.
103. Reyneke JP, Evans WG: Surgical manipulation of the occlusal plane. *Int J Adult Orthodon Orthognath Surg* 5:99–110, 1990.
104. Robinson PD, Harris K, Coghlan KC, Altman K: Bone scans and the timing of treatment for condylar hyperplasia. *Int J Oral Maxillofac Surg* 19:243–246, 1990.
105. Rosen H: Aesthetics in facial skeletal surgery. *Perspect Plast Surg* 6:1, 1993.
106. Rubenstein LK, Campbell RL: Acquired unilateral condylar hyperplasia and facial asymmetry: Report of case. *J Dent Child* 52:114, 1985.
107. Rushton MA: Growth at the mandibular condyle in relation to some deformities. *Br Dent J* 76:57, 1944.
108. Rushton MA: Unilateral hyperplasia of the mandibular condyle. *Proc R Soc Med* 39:431–438, 1946.
109. Rushton MA: Unilateral hyperplasia of the jaws in the young. *Int Dent J* 2:50, 1951.
110. Sarindin CP, Raijmakers P, Stootweg PJ, et al: Unilateral condylar hyperactivity: A histopathologic analysis of 47 patients. *J Oral Maxillofac Surg* 68:47–53, 2010.
111. Scholssmann A: Cited by G Perthes: Operative korrektur der progenie. *Zentralbl Chir* 49:1540–1541, 1922.
112. Schultz LW, Varizani SJ, Bolden TE: Unilateral hyperplasia and exostosis of the mandibular condyle: A clinical syndrome. *Oral Surg Oral Med Oral Pathol* 13:387–395, 1960.
113. Severt TR, Proffit WR: The prevalence of facial asymmetry in the dentofacial deformities population at the University of North Carolina. *Int J Adult Orthodon Orthognath Surg* 12:171–176, 1997.
114. Simon GT, Kendrick RW, Whitlock RI: Osteochondroma of the mandibular condyle: Case report and its management. *Oral Surg Oral Med Oral Pathol* 43:18, 1977.
115. Skaloud F: New surgical method for correction of prognathism of the mandible. *Oral Surg Oral Med Oral Pathol* 4:689–694, 1951.
116. Slootweg PJ, Muller H: Condylar hyperplasia. A clinico-pathological analysis of 22 cases. *J Maxillofac Surg* 14:209–214, 1986.
117. Smylski PT: Facial asymmetry and the oral surgeon. *J Otolaryngol* 5:177, 1976.
118. Steinhauser E: Kondylektomie oder korrektive osteotomie bei der kondylaren hyperplasie. *Fortschr Kiefer Gesichtschir* 25:132–135, 1980.
119. Steinhauser EW: Aesthetische und funktionielle korrektur der durch condylare hyperplasie verursachten gesichtsasymmetrie. In Pfeifer G, editor: *Die aesthetik von form und funktion in der plastichen und widerherstellungschirurgie*, Berlin, Heidelberg, New York, 1985, Springer, pp 103–109.
120. Tarsitano JJ, Wooten JW: The asymmetrical mandible. *J Oral Surg* 28:832, 1970.
121. Trauner R, Obwegeser H: Zur operationstechnik bei der progenie und anderen unterkieferanomalien. *Dtsch Zahn Mund Kieferhlkd* 23:1–26, 1955.
122. Villanueva-Alcojol L, Monje F, Gonzales-Garcia R: Hyperplasia of the mandibular condyle: Clinical, histopathologic, and treatment considerations in a series of 36 patients. *J Oral Maxillofac Surg* 69:447–455, 2011.
123. Wang-Norderud R, Ragab RR: Unilateral condylar hyperplasia and the associated deformity of facial asymmetry. *Scand J Plast Reconstr Surg* 11:91, 1977.
124. Windmaier W: Die chirurgische behandlung der postoperativen kieferdeformierungen nach lippen-kiefer-gaumenspalten-operationen. *Dtsch Zahn Mund Kieferhlkd* 33:23–35, 1960.
125. Wisth PJ: Mandibular function and dysfunction in patients with mandibular prognathism. *Am J Orthod* 85:193–198, 1984.
126. Wunderer S: Die prognathie-operation mittels frontal gestieltem maxillofragment. *Oesterr Z Stomatol* 59:98–102, 1962.

23

Short Face Growth Patterns: Maxillomandibular Deficiency

JEFFREY C. POSNICK, DMD, MD

- Functional Aspects
- Clinical Characteristics
- Dental Findings
- Radiographic Findings
- Treatment Indications and Objectives
- The Role of Growth Modification in the Preadolescent Patient
- Definitive Reconstruction
- Postsurgical Orthodontic Maintenance and Detailing
- Complications, Informed Consent, and Patient Education
- Malocclusion after Orthodontics and Orthognathic Surgery
- Temporomandibular Disorders: The Effects of Orthodontics and Orthognathic Surgery
- Conclusions

A frequently seen pattern of facial disharmony has the common denominator of a significant degree of vertical and horizontal (sagittal) deficiency in the maxilla and mandible. Measuring the ratio of the lower facial height (i.e., the subnasion to the menton) to the upper facial height (i.e., the glabella to the subnasion) will confirm that the lower height is diminished. The height of the alveolar process in both jaws is reduced.[23] The visual presentation of the face includes an apparent redundancy of the soft-tissue envelope of the lower face, thereby creating "chubby cheeks" in the adolescent and a prematurely aged look by early adulthood.

Clinical examination of the individual with a short face growth pattern will generally confirm a pseudoprominence of the nose; an acute nasolabial angle; a measurable and visually perceived short lower facial height with a disproportionate facial width-to-height ratio; and either a prominent pogonion (more frequent with a Class I deep bite) or a weak chin (more common with a Class II excess overjet).[3,10,11,20,21,35,39,40] Normally, there is a 1 : 2 relationship between the distance from the base of the nose to the upper lip and the distance from the lower lip to the chin. In the individual with a short face growth pattern, the relationship is closer to 1 : 1.[15,42,43,56] The labiomental sulcus is deep and the lower lip is everted, thereby further confirming the degree of mandibular deficiency. The maxillary incisors are often completely covered by the upper lip, which creates an edentulous appearance in repose and an unfavorable "no gingiva" smile. With the teeth in occlusion, the oral aperture appears closed and the upper lip projects forward, often in front of procumbent incisors. Individuals are frequently described as having a sad or unhappy look in repose as a result of the downturned oral commissures and the closed aperture. The reduced lower facial height causes the everted and prominent lips that would otherwise have appropriate shape and volume if the facial height and horizontal projection of the jaws were restored. In the profile view, the nose appears long and prominent. There is often a lateral flaring of the nostrils with a "hanging columella."

Classically, there is a deep bite with either marked anterior crowding or excessive flaring with less dental crowding.[34] There may be a Class II malocclusion with greater than average overjet.[50,51] If so, the mandible has greater deficiency than the maxilla, and the pogonion will lack prominence. Individuals with Class I deep bite without excess overjet are more likely to have a retropositioned A-point as compared with the B-point (i.e., a counterclockwise rotated maxillomandibular complex) and a prominent pogonion. The maxillary and mandibular planes are often nearly horizontal, which results in a "square jaw" facial appearance.

Functional Aspects

- Articulation errors in speech and altered swallowing mechanisms that result from the small jaws, the Class II

deep-bite malocclusion, and the limited intraoral volume for the tongue are expected (see Chapter 8).
- Difficulties with chewing and with lateral and protrusive mandibular movements as a result of the Class II deep-bite malocclusion are expected (see Chapter 8).
- A spectrum of upper airway sites of obstruction often exists with the short face growth pattern. This includes obstructions at the intranasal, palatopharyngeal (retropalatal), and glossopharyngeal (retroglossal) locations. The incidence of obstructive sleep apnea is increased in this patient population and should be ruled out (see Chapter 10 & 26).
- Detrimental long-term effects on the dentition and the periodontium as a result of any traumatic malocclusion, the crowding of the teeth in the limited alveolar housing, and previous camouflage orthodontic treatment may be seen (see Chapter 5 & 6).
- A higher incidence of temporomandibular disorders in the form of masticatory muscle discomfort as a result of the short facial height and the deep-bite malocclusion has been reported (see Chapter 9).

Clinical Characteristics

- Absence of maxillary tooth show beneath a relaxed upper lip
- Limited tooth and no gingiva show with broad smile
- Acute nasolabial angle
- Depressed pyriform rims
- An apparent fullness of the cheeks and a squareness of the overall face
- An apparent prominent and elongated nose when viewed in profile
- Procumbent everted lips, turned down corners of the mouth, and a closed aperture
- An excessively deepened and elongated labiomental fold
- Soft-tissue bunching that eventually results in perioral creases (i.e., marionette lines)
- Early and deep jowl formation by middle age
- Premature loss of any semblance of an attractive neck–jaw angle

Dental Findings

- Class II excess overjet malocclusion is common, but a Class I deep bite is also frequent.
- A deep impinging bite with palatal irritation is a frequent finding.
- Dental crowding with flaring of the incisors in each arch is also a frequent finding.

Radiographic Findings

- The total anterior facial height is decreased (i.e., from the nasion to the menton).
- The lower anterior facial height is short (i.e., from the subnasion to the menton).
- Horizontal retrusion is measurable at the A-point and the B-point in both jaws.
- Disproportion in the ratio of the facial width to the facial height is measurable on an anteroposterior cephalometric radiograph or a computed tomography scan.
- Excessive procumbency of the maxillary and mandibular incisors is frequently measurable.
- Excessive flattening (i.e., counterclockwise rotation) of the maxillary and mandibular planes is frequently measurable.
- Decreased alveolar bone height associated with the maxillary incisors is measurable (i.e., the distance from the floor of the nose to the incisal edge).
- Decreased alveolar bone height associated with the mandibular incisors is measurable (i.e., the distance from the short menton to the incisal edge).

Treatment Indications and Objectives

The indications for treatment in a patient with a short face growth pattern may be to improve function (e.g., occlusion, chewing, swallowing, speech, breathing, lip posture), but another compelling aspect is typically the enhancement of facial aesthetics. The visually aesthetic advantages of the surgical lengthening and projection of the short and retrusive lower face toward Euclidian proportions are immediately apparent and do not require quantification.[32,33,58]

To restore facial proportions and preferred facial aesthetics, there must be both vertical and horizontal expansion of the maxillomandibular complex.[54] Accomplishing these objectives will eliminate cheek and lip region bunching and distortion and restore a pleasing soft-tissue envelope. When this is done, a youthful oral aperture is restored. The corners of the mouth are no longer downturned, and the lips will be separated when they are at rest. The upper vermillion increases in volume, and the labiomental fold flattens. The maxillary incisors can be seen in repose, and the gingiva is visible with smile. The nose no longer appears excessively prominent, and the nasolabial angle improves. The jowls diminish, and the length-to-width ratio of the face is restored. The shape of the face changes from square to oval with the restoration of the natural contours and attractive ogee curves. The overall facial aesthetic changes from sad and older appearing to younger looking and more attractive (Figs. 23-1 through 23-7).

The Role of Growth Modification in the Preadolescent Patient

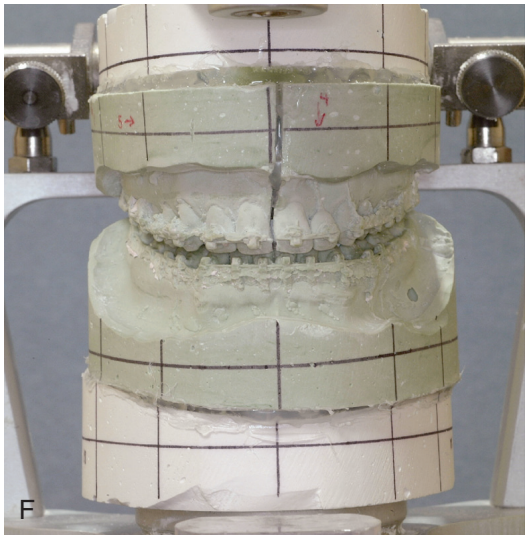
Individuals with lower anterior facial height deficiency can frequently be identified at an early age. They tend to have an acute (flat) mandibular plane angle with a deep bite.[25] This is accompanied by an upward (counterclockwise) rotated anterior mandible that results in a prominent-appearing pogonion.

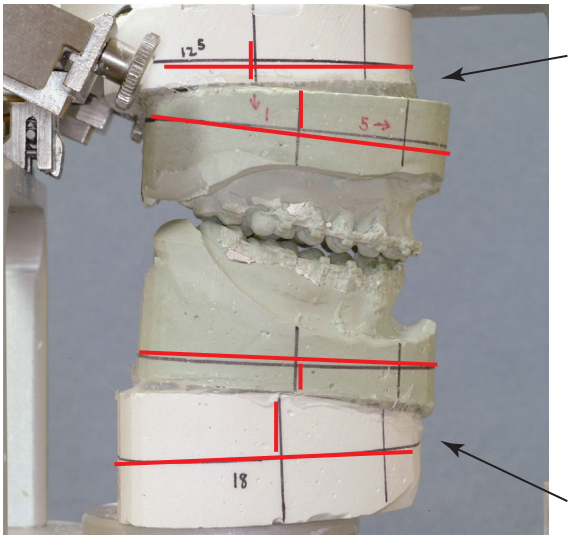
Text continued on p. 892

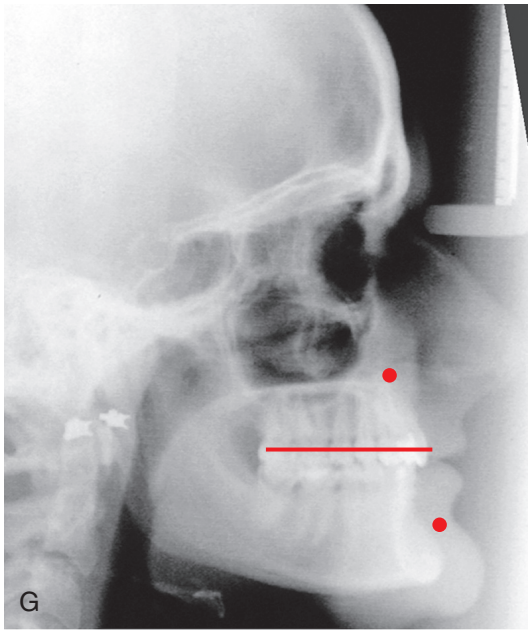
• **Figure 23-1** A 28-year-old woman arrived for a third surgical opinion regarding a reduction rhinoplasty and reduction genioplasty. The diagnosis of a short face growth pattern was made. Maxillomandibular advancement and vertical lengthening were recommended. The patient agreed to a surgical approach with perioperative orthodontics. Her surgery included maxillary Le Fort I osteotomy in segments (vertical lengthening, horizontal advancement, arch expansion and clockwise rotation) with interpositional grafting; sagittal split ramus osteotomies (horizontal advancement and clockwise rotation); and osseous genioplasty (vertical lengthening) with interpositional grafting. **A,** Frontal views in repose before and after reconstruction. **B,** Frontal views with smile before and after reconstruction.

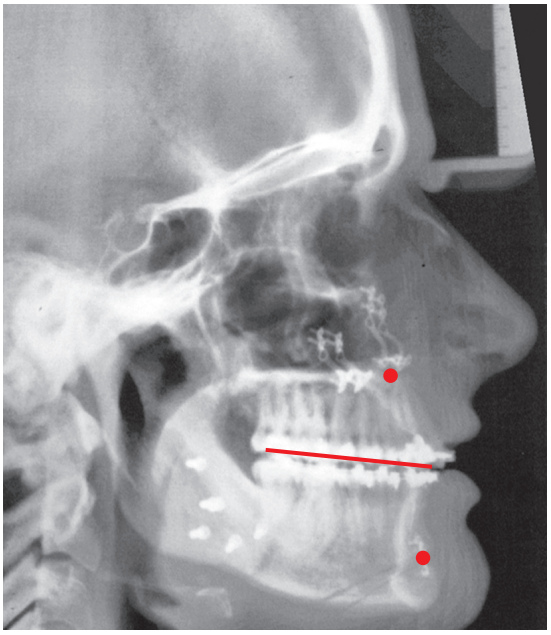
• **Figure 23-1, cont'd** **C,** Oblique facial views before and after reconstruction. **D,** Profile views before and after reconstruction.

Continued







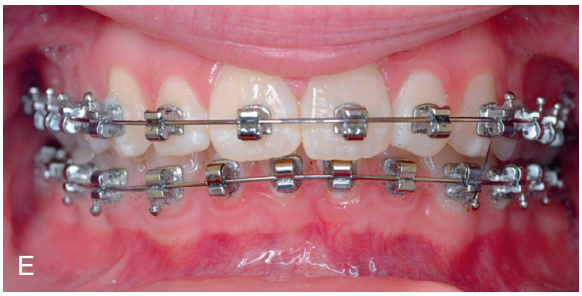


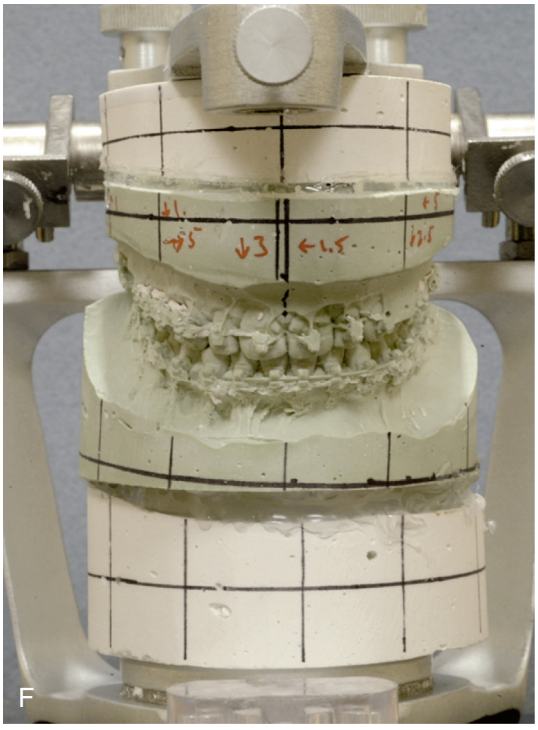
• **Figure 23-1, cont'd** **E,** Occlusal views before and after treatment. Only minimal change in the occlusion was required. **F,** Articulated dental casts that indicate analytic model planning. **G,** Lateral cephalometric radiographs before and after treatment.

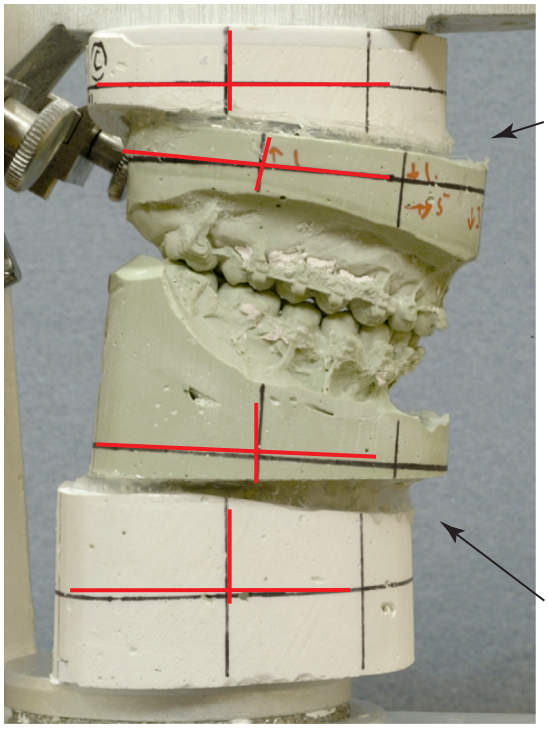
• **Figure 23-2** A 16-year-old girl arrived with her parents for surgical evaluation. She requested a reduction rhinoplasty and a chin implant. A diagnosis of maxillomandibular deficiency with a Class II excess overjet malocclusion was made. She agreed to a surgical and (redo) orthodontic approach. No extractions were carried out, and a degree of incisor proclination was tolerated. The patient's surgery included maxillary Le Fort I osteotomy (horizontal advancement, vertical lengthening, and clockwise rotation) with interpositional grafting; sagittal split ramus osteotomies (horizontal advancement and clockwise rotation); and osseous genioplasty (vertical lengthening) with interpositional grafting. **A,** Frontal views in repose before and after treatment. **B,** Frontal views with smile before and after treatment.

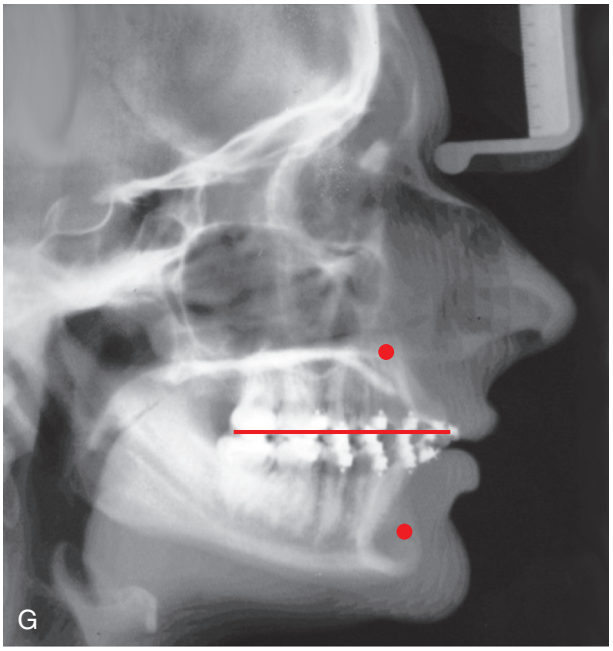
Continued

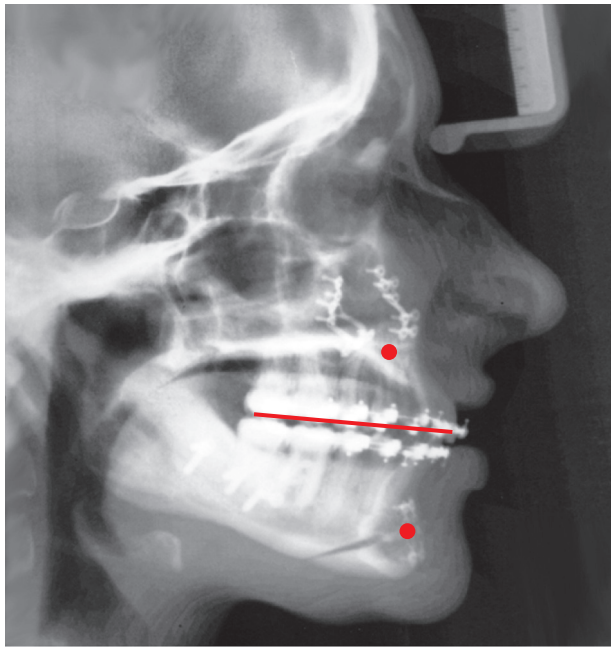
• **Figure 23-2, cont'd C,** Oblique facial views before and after treatment. **D,** Profile views before and after treatment.











• **Figure 23-2, cont'd** **E,** Occlusal views before and after treatment. Only minimal change in the occlusion was required. **F,** Articulated dental casts that indicate analytic model planning. **G,** Lateral cephalometric radiographs before and after treatment.

©Elsevier Inc.
Orthognathic Surgery; J.C. Posnick

• **Figure 23-3** A 24-year-old woman arrived for evaluation. She had previously undergone four bicuspid extractions and orthodontics to neutralize her occlusion during her high school years. Analysis confirmed a short face growth pattern that accounted for the apparent large nose, weak profile, obtuse neck–chin angle, and edentulous look. She agreed to a combined (redo) orthodontic and surgical approach. The patient's surgery included maxillary Le Fort I osteotomy (horizontal advancement, vertical lengthening, and clockwise rotation) with interpositional grafting; sagittal split ramus osteotomies (horizontal advancement and clockwise rotation); and osseous genioplasty (vertical lengthening and minimal horizontal advancement) with interpositional grafting. Minimal change in the occlusion was required. **A,** Frontal views in repose before and after treatment. **B,** Frontal views with smile before and after treatment.

• **Figure 23-3, cont'd C,** Oblique facial views before and after treatment. **D,** Profile views before and after treatment.

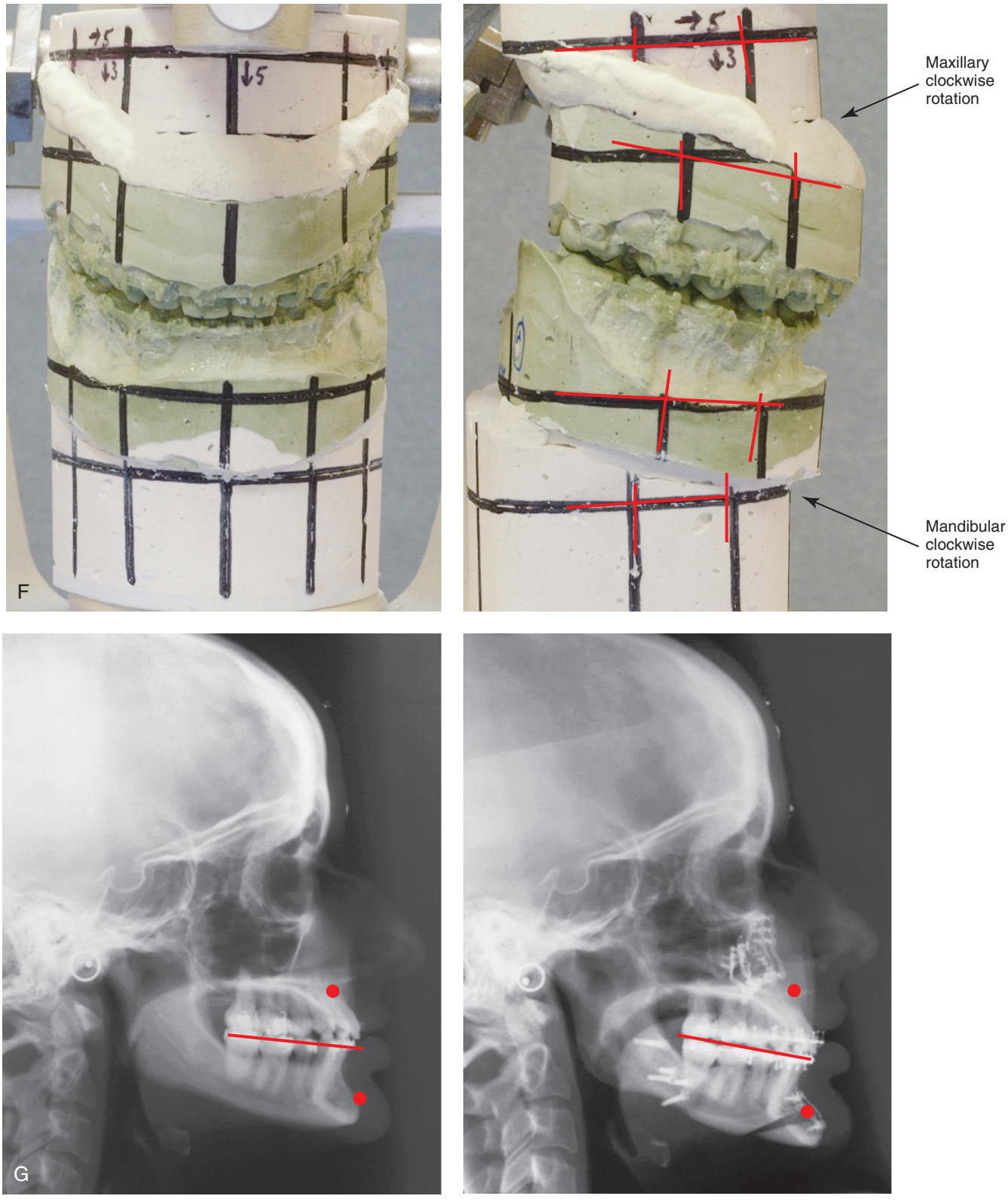
Continued

Prior to retreatment after four bicusbid extractions

Pre surgery

After treatment

• **Figure 23-3, cont'd E,** Occlusal views before retreatment, with orthodontics in progress, and after reconstruction.



• **Figure 23-3, cont'd** **F,** Articulated dental casts that indicate analytic model planning. **G,** Lateral cephalometric radiographs before and after treatment.

• **Figure 23-4** A woman in her late 20s arrived for the evaluation of her edentulous look. She had previously undergone orthodontic neutralization of her occlusion during her high school years. Analysis suggested a short face growth pattern that accounted for her apparent large nose, protrusive chin, and edentulous look. The patient also complained of nasal obstruction, restless sleeping, and daytime fatigue. Examination confirmed septal deviation and enlarged inferior turbinates, and an attended polysomnograph documented mild obstructive sleep apnea. She underwent a combined (redo) orthodontic and surgical approach. The patient's surgery included maxillary Le Fort I osteotomy in segments (horizontal advancement, vertical lengthening, arch expansion and clockwise rotation) with interpositional grafting sagittal split ramus osteotomies (horizontal advancement and clockwise rotation); osseous genioplasty (vertical lengthening and minimal horizontal advancement) with interpositional grafting; and septoplasty and inferior turbinate reduction. **A,** Frontal views in repose before and after treatment. **B,** Frontal views with smile before and after treatment.

• **Figure 23-4, cont'd** **C,** Oblique facial views before and after treatment. **D,** Profile views before and after treatment.

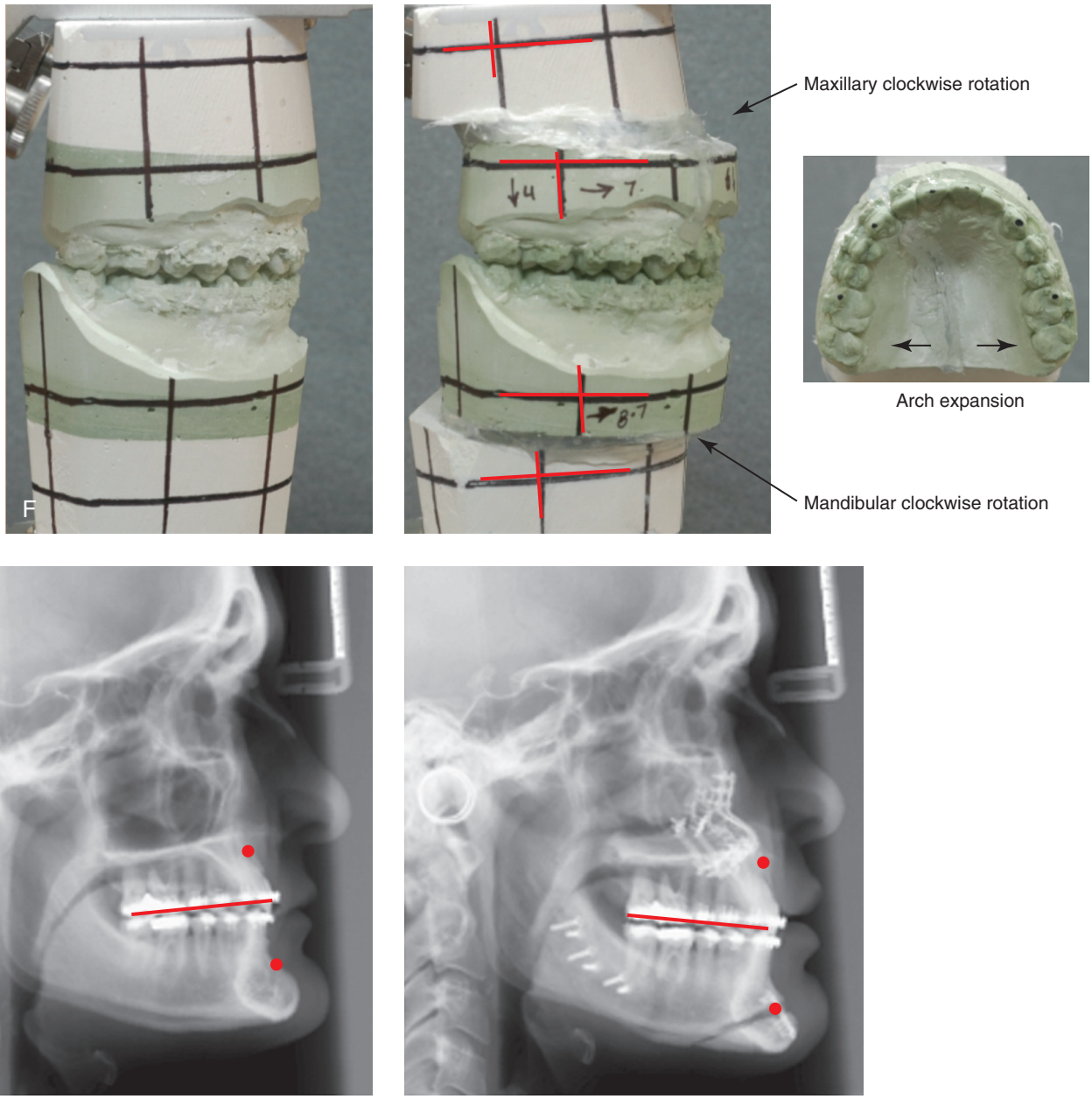
Continued

Prior to retreatment

Pre surgery

After treatment

• **Figure 23-4, cont'd E,** Occlusal views before retreatment, with orthodontics in progress, and 1 year after reconstruction. Minimal change in the occlusion was required.



• **Figure 23-4, cont'd** **F,** Articulated dental casts that indicate analytic model planning. **G,** Lateral cephalometric radiographs before and after treatment.

©Elsevier Inc.
Orthognathic Surgery: J.C. Posnick

• **Figure 23-5** A 16-year-old girl was referred by her orthodontist, primarily for the evaluation of malocclusion. She underwent attempts at growth modification and then orthodontic treatment when she was between 10 and 12 years old. The examination confirmed maxillomandibular deficiency with a Class II excess overjet malocclusion. She had chronic nasal obstruction, and she was also found to have a deviated septum and hypertrophic inferior turbinates. A surgical and (redo) orthodontic approach was recommended. The patient's surgery included maxillary Le Fort I osteotomy in segments (horizontal advancement, vertical lengthening, and arch expansion) with interpositional grafting; sagittal split ramus osteotomies (horizontal advancement) osseous genioplasty (vertical lengthening) with interpositional grafting; and septoplasty and inferior turbinate reduction. **A,** Frontal views in repose before and after treatment. **B,** Frontal views with smile before and after treatment.

• **Figure 23-5, cont'd C,** Oblique facial views before and after treatment. **D,** Profile views before and after treatment.

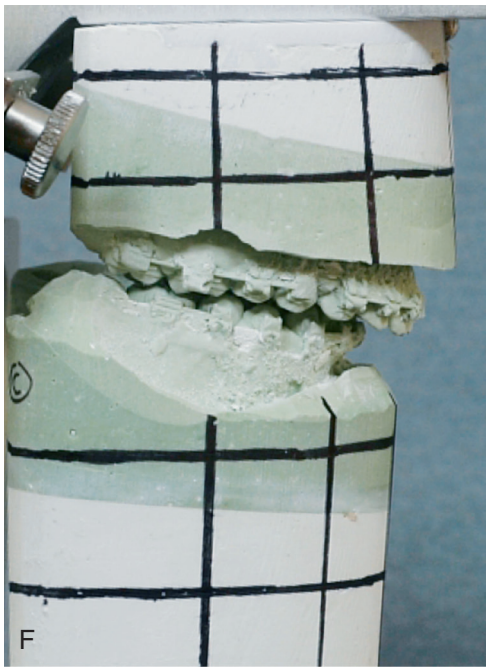
Continued

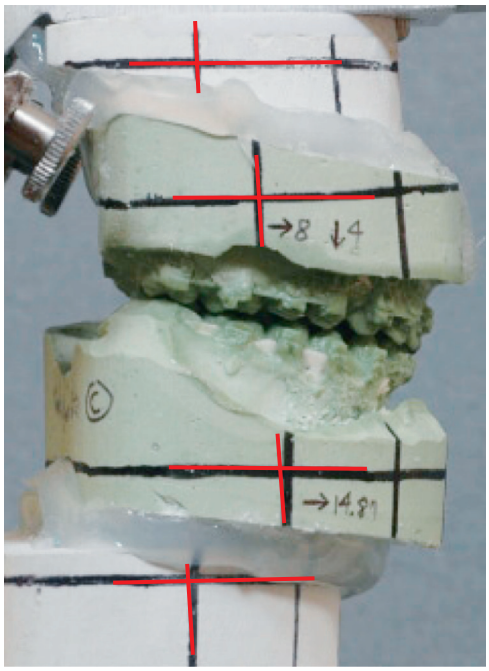
Prior to retreatment

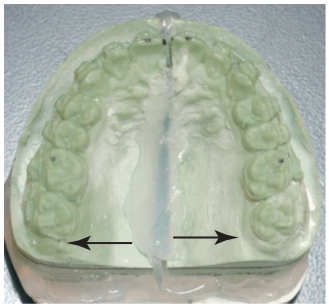
Pre surgery

After treatment

• **Figure 23-5, cont'd E,** Occlusal views before retreatment, with orthodontics in progress, and after treatment.







Arch expansion

• **Figure 23-5, cont'd F,** Articulated dental casts that indicate model planning. **G,** Lateral cephalometric radiographs before and after treatment.

©Elsevier Inc.
Orthognathic Surgery; J.C. Posnick

• **Figure 23-6** A man in his mid 20s arrived for the surgical evaluation of an edentulous look and difficulty breathing. He was found to have a short face growth pattern, nasal obstruction, and mild obstructive sleep apnea. A surgical approach with (redo) orthodontics was recommended. The patient's surgery included maxillary Le Fort I osteotomy (horizontal advancement, vertical lengthening, and clockwise rotation) with interpositional grafting; sagittal split ramus osteotomies (horizontal advancement and clockwise rotation); osseous genioplasty (vertical lengthening) with interpositional grafting; and septoplasty and inferior turbinate reduction. **A,** Frontal views in repose before and after treatment. **B,** Frontal views with smile before and after treatment.

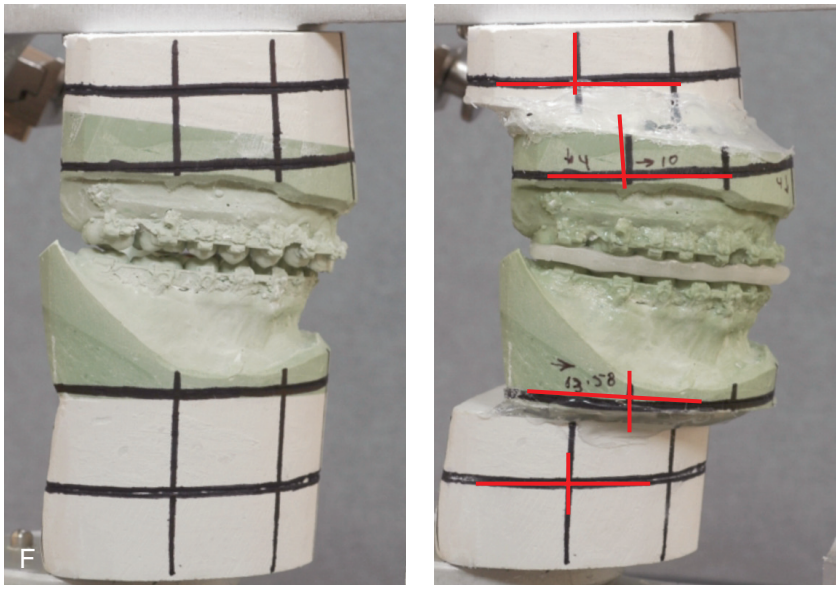
©Elsevier Inc.
Orthognathic Surgery; J.C. Posnick

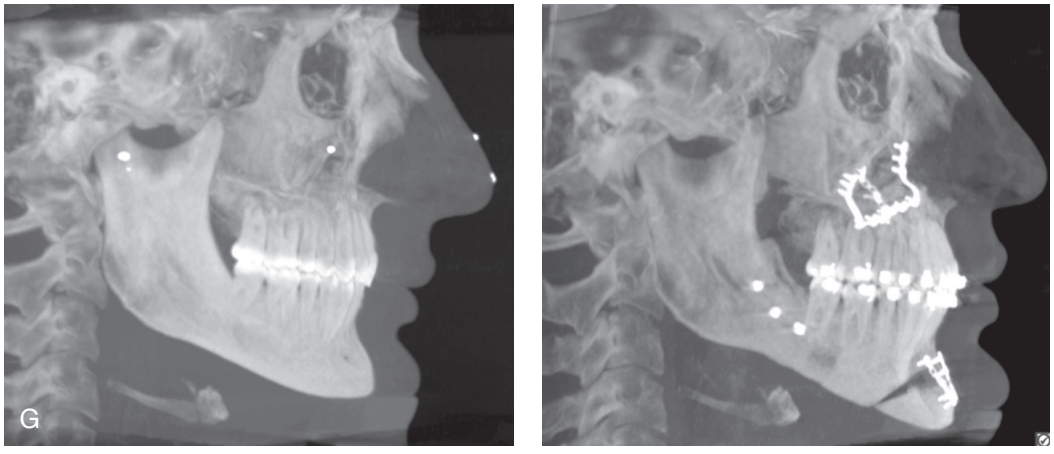
• **Figure 23-6, cont'd** **C,** Oblique facial views before and after treatment. **D,** Profile views before and after treatment.

Continued

Prior to surgery

After treatment





• **Figure 23-6, cont'd** **E,** Occlusal views before and after treatment. **F,** Articulated dental casts that indicate analytic model planning. **G,** Lateral cephalometric radiographs before and after treatment.

• **Figure 23-7** A 26-year-old woman with a short face growth pattern arrived to discuss her maxillofacial issues. During her early teenage years, she underwent a full course of orthodontics. Her maxillary lateral incisors were congenitally absent, and the spaces were orthodontically closed, partially by tipping the canines forward. The maxillary incisor root resorption was significant. There was crowding in the mandibular arch with a degree of procumbency. The patient had an interest in improving her weak profile and her edentulous look. The patient's history confirmed heavy snoring, restless sleeping, and daytime fatigue. Examination confirmed a deviated septum and hypertrophic inferior turbinates. Maxillary retrusion resulted in the close proximity of the soft palate to the pharyngeal wall. A retrusive mandible resulted in the close proximity of the tongue to the posterior pharyngeal wall. An attended polysomnogram confirmed mild obstructive sleep apnea. Evaluation by a periodontist and an orthodontist confirmed the advantage of a comprehensive approach to improve the airway, to achieve a more balanced facial appearance, and to improve long-term periodontal health. A trial of continuous positive airway pressure improved daytime alertness, but the patient's feelings of claustrophobia prevented its use. Periodontal evaluation with gingival grafting and maintenance was carried out. Orthodontic appliances were placed with limited dental repositioning of the maxillary incisors as a result of baseline root resorption. The patient's surgery included maxillary Le Fort I osteotomy in segments (horizontal advancement, vertical lengthening, arch form correction, and clockwise rotation) with interpositional graft; bilateral sagittal split ramus osteotomies (horizontal advancement); and osseous genioplasty (vertical lengthening) and limited horizontal advancement) with interpositional bonegraft. As a result of treatment, the patient has had a relief of her snoring, more restful sleeping, and improved alertness during the day. **A,** Frontal views in repose before and after treatment. **B,** Frontal views with smile before and after treatment.

Continued

• **Figure 23-7, cont'd C,** Oblique facial views before and after treatment. **D,** Profile views before and after treatment.

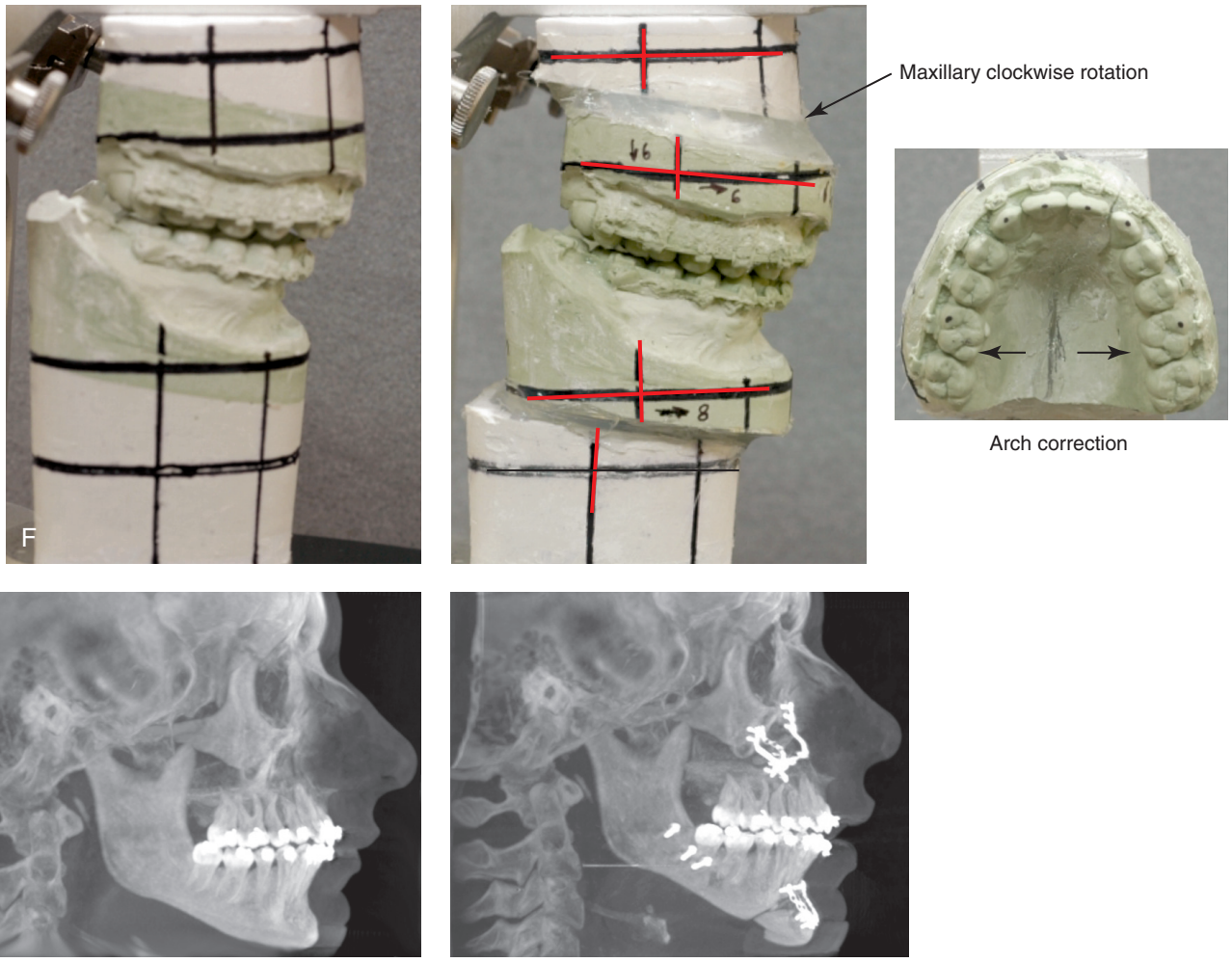
Prior to retreatment with congenital missing lateral incisors and dental gap closure

Pre surgery

After treatment

• **Figure 23-7, cont'd** **E,** Occlusal views before retreatment, with orthodontics in progress, and 1 year after reconstruction. Minimal change in occlusion was required.

Continued



• **Figure 23-7, cont'd F,** Articulated dental casts that indicate analytic model planning. **G,** Lateral cephalometric radiographs before and after treatment.

Orthodontic growth modification treatment during preadolescence attempts to increase the eruption of the posterior teeth.[19,31,44] If this is successful, while lengthening the anterior facial height, this will cause the mandible to rotate clockwise, thereby resulting in a downward and backward shift at the pogonion. Unfortunately, this backward rotation of the mandible is not favorable to the profile unless it is also accompanied by horizontal advancement. For the preadolescent with a Class II excess overjet and a short face growth pattern, cervical headgear has been used to apply extrusion forces to the posterior maxillary dentition; however, this tends to simultaneously "set back" the already retrusive maxilla. As an alternative, functional intraoral appliances have also been tried in an attempt to erupt the posterior dentition. These orthopedic maneuvers, when followed by orthodontic mechanics, may achieve a neutralized occlusion in the child with maxillomandibular deficiency. However, this treatment cannot achieve the vertical height and the horizontal projection required to normalize the facial proportions and improve the airway.[4,7,22,52,59]

Since the late 1970s, Delaire and colleagues have attempted to achieve horizontal advancement and vertical lengthening of the maxilla with a face mask apparatus.[6] The best data indicate that use of the Delaire mask to increase maxillary projection is only effective for young children who are less than 8 to 10 years old, when the sutures remain open.[48,49] Unfortunately, with the short face growth pattern, Delaire face mask therapy is simultaneously counterproductive on the baseline deficient mandible, even if it effectively projects the maxilla.

Definitive Reconstruction

Timing of Orthognathic Surgery

The best evidence available indicates that, when orthognathic surgery is performed on a patient with a short face growth pattern (i.e., maxillomandibular deficiency) as early as the completion of the adolescent growth spurt, the result will be stable from a growth perspective. Further sagittal growth of either the maxilla or the mandible almost never occurs.[45] Vertical growth in both the mandible and the maxilla is also not likely to continue, but, if it does, it should be proportionate. Therefore, proceeding with orthognathic

surgery in the individual with maxillomandibular deficiency as early as the age of 13 or 14 years—but only after the eruption and orthodontic alignment of the teeth—is generally safe from a growth perspective.

Orthodontic Preparation for Orthognathic Surgery

Class II, Division I

The basic presurgical orthodontic goals for the individual with Class II, Division I malocclusion include the following: 1) aligning each dental arch independently; 2) placing the incisors in their preferred anteroposterior and vertical plane position; 3) anticipating a stable arch compatibility after surgery; and 4) considering segmental maxillary osteotomies to accomplish these goals.

Extractions from the maxillary arch are generally not indicated. There is often an excessive curve of Spee in the mandible, and the full leveling of the curve after surgery is generally preferred. This is rapidly accomplished via the extrusion of the premolars after surgery with the use of vertical elastics. The alternative to preoperative leveling involves incisor intrusion. Less clockwise rotation of the mandible at operation should be expected when incisor intrusion has been carried out to flatten the curve of Spee.

The establishment of eventual arch compatibility through orthodontics and surgery requires the following:

- Compatible intercanine widths either through orthodontics alone or with the help of maxillary segmental osteotomies
- Bonding or banding and leveling of the first and second molars
- Anticipating similar maxillary and mandibular arch forms either through orthodontics alone or with the help of maxillary segmental osteotomies

Postsurgical orthodontics will maintain the arch form and bring the teeth into final occlusion. The full leveling of the curve of Spee and closure of the extraction spaces in the mandible is usually accomplished during the detailing phase after surgery.

Class II, Division II

The basic presurgical orthodontic goals for the Class II, Division II malocclusion include the following: 1) the need for the proper vertical and anteroposterior positioning of the maxillary and mandibular incisors; 2) the need to level the curve of Spee; and 3) the need to anticipate coordinated dental arches after surgery, 4) which may include or require segmental maxillary osteotomies.

In general, the maxillary incisors will require labial tipping to correct the inclination. For severe deep-bite cases, it is advisable to fully level the curve of Spee in the mandibular arch after surgery. Preoperative glass ionomer bite planes cemented to the mandibular molars may be helpful, but unwanted molar intrusion can occur. Another option is to bond metal or ceramic "bite turbos" to the lingual surfaces of the maxillary incisors. This provides an anterior bite plane to open the bite without molar extrusion.

Anticipating the advantages of the surgical segmenting of the maxilla to level the curve of Spee and to correct arch-width discrepancies is often helpful. The postsurgical final leveling of the curve in the mandibular arch is generally both efficient and effective.

Basic Surgical Approach

By definition, for the patient with a moderate to severe short face growth pattern, there will be a need to simultaneously surgically reposition the maxilla, the mandible, and the chin region if the normalization of both the facial height and the facial projection is desired.[1,2,5,8,9,27-30,36-41,53,58] For the patient with a Class I short face growth pattern, simply lengthening the maxilla to improve the upper lip to tooth relationship through Le Fort I osteotomy with maintenance of the occlusion will cause an undesirable backward rotation of the mandible. This "single jaw" approach prevents the necessary horizontal advancement of the maxillomandibular complex. Unfortunately, the inability to achieve adequate sagittal (horizontal) projection of the maxillomandibular complex is likely to result in aesthetic disappointment. For this reason, bimaxillary surgery (i.e., Le Fort I and bilateral sagittal split ramus osteotomies) is required for the correction of a short face growth pattern. In borderline cases, a limited camouflage approach involving an osseous genioplasty with significant vertical lengthening and a degree of horizontal advancement may at least provide some aesthetic enhancement without introducing surgical deformity (see Chapters 37 and 40).

Orthognathic planning begins with the making of decisions about the magnitude of vertical lengthening, horizontal advancement, and clockwise rotation required in the maxilla. These decisions are primarily based on aesthetic judgments that are made when examining the patient in the natural head position (see Chapter 12). A common mistake is to err on the side of insufficient lengthening of the midface out of a belief that the short upper lip will not accommodate the skeletal lengthening. Experience has shown that the hypotonic (short) upper lip will stretch and gain function when the maxilla is surgically expanded (i.e., horizontal advancement and vertical lengthening). In addition, as the maxilla is surgically lengthened and simultaneously advanced and clockwise rotated, the pyriform rims and the anterior nasal spine are thrust forward and inferiorly, thus further stretching and filling out the soft tissues of the lower aspect of the nose and the upper lip. When the anterior floor of the nose (i.e., the alveolar bone height above the incisors) is short, consideration should also be given to graft augmentation of the anterior floor at the rim just above the incisors to further improve nasal base aesthetics. The need for this should be definitively reassessed during the operation, after the maxilla is stabilized with plate and screw fixation in its new location. In addition to maxillary

reconstruction, the mandible requires horizontal advancement and often clockwise rotation. This is accomplished through sagittal split ramus osteotomies. In most cases, an osseous genioplasty provides access to vertically lengthen the deficient anterior mandibular alveolar height and interpositional grafting is required. Horizontal advancement of the chin is not generally beneficial.

The previous line of thinking was that vertical lengthening, especially in combination with horizontal advancement of the maxilla, is unstable and inadvisable.[5,13,14,17,18,24,46,57] We now know that, with full mobilization of the down-fractured maxilla, lengthening and advancement can be carried out with occlusal stability and the routine achievement of improved facial aesthetics. This requires the following: 1) well-designed and executed osteotomies; 2) the full mobilization of the down-fractured maxilla; 3) rigid plate and screw fixation; 4) the placement of an interpositional graft (e.g., autogenous or allogenic corticocancellous iliac bloc graft) into the gaps between the zygomatic buttress and the pyriform rim on each side; 5) additional rigid plate and screw fixation to secure each graft; and 6) appropriate postoperative management to control bite forces during the early healing phase at the Le Fort I osteotomy sites (see Chapter 15). Kretschmer and colleagues studied the incidence of relapse after Le Fort I osteotomy with vertical lengthening with both single piece and segmental Le Fort I osteotomies (see Chapter 17).[16] The authors' data confirms that, when significant advancement and lengthening at the Le Fort I osteotomy site are carried out, key factors to achieve postoperative stability include the interpositional bone grafting of defects and rigid plate and screw fixation. Interestingly, the researchers did not find segmentation of the maxilla during bimaxillary procedures to be a significant cause of either skeletal or dental relapse.

Immediate Presurgical Planning

The immediate presurgical planning takes into account collaborative clinical efforts among the orthodontist, the surgeon, the patient, and the patient's family that will have been ongoing since the initial consultation visits. Depending on the condition of the dentition and its supporting structures, the restorative dentist and the periodontist may also have been intricately involved.

The patient's specific medical and dental records are reviewed, including radiographs (e.g., computed tomography scans; panoramic, lateral, and posteroanterior cephalometric radiographs; periapical images); photographs (facial and occlusal); and dental models. Immediate presurgical records (i.e., within 2 to 6 weeks of surgery) will most importantly include facial measurements obtained from direct visual examination; alginate impressions of the maxilla and mandible; an accurate centric relation bite registration; and face-bow registration. First, broad-stroke decisions are made regarding the preferred vector changes for each jaw; only then can the precise millimeter distances and angular changes required for each jaw to achieve the desired result be determined (see Chapter 12). Analytic model planning is carried out on the articulated dental casts with use of the Erickson Model Table. Splints are constructed to assist with the achievement of the precise occlusion and preferred facial aesthetics in the operating room (see Chapter 13).

Postsurgical Orthodontic Maintenance and Detailing

If the Class II excess overjet patient has a presurgical habit of posturing the mandible forward (i.e., a dual bite), there may also be a tendency to do so during the immediate postoperative healing phase, thereby producing a pseudo-anterior crossbite. Intermittent intermaxillary elastics are used during this time to retrain the masticatory muscles, to provide motion in the temporomandibular joints, to increase patient comfort, and to confirm the stability of the occlusion. If a splint is used, when it is removed (e.g., 5 weeks after surgery), the patient should immediately (ideally within 24 hours) return to the orthodontist for the placement of a continuous maxillary arch wire. In general, the maintenance of the new arch form after segmental surgery is the most challenging aspect of the postsurgical orthodontic phase of treatment.

The postsurgical orthodontic objectives should be to maintain the surgical result and to bring the teeth into ideal occlusion. In the patient in whom the curve of Spee is to be fully leveled postoperatively, the use of vertical elastics to close the mid arch open bites will also be a priority.

Complications, Informed Consent, and Patient Education

The orthodontist and the surgeon are asked to evaluate the individual with maxillomandibular deficiency and to then attempt to alter nature's events. By understanding the expected effects of the uncorrected short face growth pattern (i.e., airway, aesthetics, long-term dental health) and with knowledge of how specific orthodontic and orthognathic interventions will alter head and neck function and enhance facial aesthetics, a successful outcome is expected in most cases. An understanding of the potential risks in comparison with the anticipated benefits of each intervention is taken into account, because complications and dissatisfaction will always occur in a small percentage of individuals. The clinician's ability to communicate the potential risks, complications, alternative approaches, and benefits of the recommended treatment to the patient and their family is essential. The patient's informed decision to proceed provides the basis for shared responsibility of the expected convalescence and long-term outcome (see Chapter 16).

Malocclusion after Orthodontics and Orthognathic Surgery

There are inherent biologic challenges to the orthodontic movement of teeth within the alveolar process and the

surgical repositioning of the jaws or jaw segments after osteotomies in a patient with a short face growth pattern. This includes the technical aspects of the repositioning; the initial healing requirements; and the difficulties associated with the long-term maintenance of the relocated teeth, jaws, and jaw segments. When following sound biomechanical and aesthetic principles, clinicians carry out orthodontic treatment and surgical procedures that involve correction and then successful maintenance of the result in most cases. Potential intraoperative technical difficulties associated with sagittal split ramus osteotomies of the mandible and with maxillary Le Fort I osteotomy can result in immediate (early) malocclusion, but this is preventable in most cases. Ongoing growth after surgery is rarely an issue with a short face growth pattern. Although condylar resorption after orthognathic surgery is not entirely predictable, it is uncommon (see Chapter 36). The orthodontic removal of dental compensations and the placement of the teeth solidly in the alveolar housing will best ensure a long-term stable occlusion and periodontal health. All of these factors are taken into account to achieve and then maintain a favorable result (see Chapter 17).

Temporomandibular Disorders: The Effects of Orthodontics and Orthognathic Surgery

It is estimated that an average of 32% of the population reports at least one symptom of temporomandibular disorder, whereas an average of 55% demonstrate at least one clinical sign. Temporomandibular disorder includes various symptoms and signs of the temporomandibular joints, the masticatory muscles, and the related structures. The symptoms and signs may include a spectrum of referred head and neck pain; joint noise (e.g., popping, clicking, crepitus); reduced or altered mandibular movements (i.e., muscle spasm or disc displacement with or without reduction); condylar head erosion; and pain on direct palpation of either temporomandibular joint or of the masticatory muscles. Occlusal factors (i.e., degrees of malocclusion) are often claimed to be associated with temporomandibular disorders. Interestingly, patients with maxillomandibular deficiency (i.e., the short face growth pattern) tend to have higher maximum biting forces than those with normal or long vertical facial dimensions.[12,26,47,55] This has been documented to be statistically significant during swallowing, simulated chewing, and maximum biting.[31] Affected patients often claim to have difficulty chewing through foods and also experience masseter muscle discomfort. The individual with a short face growth pattern and a Class II significant overjet malocclusion is likely to have an uncomfortable dual bite, whereas the patient with a Class I deep bite is more likely to have temporomandibular joint clicking; in the latter case, there is no reason to believe that progressive temporomandibular joint pathology will occur (see Chapter 9).

Conclusions

The dentofacial disharmony referred to as *short face growth pattern* has the common denominator of a significant degree of vertical and horizontal deficiency in the maxilla and the mandible. The aesthetic presentation generally includes an apparent fullness of the cheeks and a deficiency of the lower face in the adolescent, with a prematurely aged look present by early adulthood. To achieve Euclidian facial proportions with enhanced facial aesthetics, there must be both vertical and horizontal expansion of the maxillomandibular complex. Accomplishing these objectives should successfully eliminate soft-tissue bunching, restore a more pleasing facial appearance, improve the upper airway, and correct any presenting malocclusion.

References

1. Bell WH: Correction of the short-face syndrome-vertical maxillary deficiency: A preliminary report. *J Oral Surg* 35:110, 1977.
2. Bell WH, Jacobs JD, Legan HL: Treatment of Class II deep bite by orthodontic and surgical means. *Am J Orthod* 85:1–20, 1984.
3. Blanchette M, Nanda R, Currier G, et al: A longitudinal cephalometric study of the soft tissue profile of short- and long-face syndromes from 7 to 17 years. *Am J Orthod Dentofac Orthop* 109:116–131, 1996.
4. Burstone CR: Deep overbite correction by intrusion. *Am J Orthod* 72:1–22, 1977.
5. de Mol van Otterloo JJ, Tuinzing DB, Kostense P: Inferior positioning of the maxilla by a Le Fort I osteotomy: A review of 25 patients with vertical maxillary deficiency. *J Craniomaxillofac Surg* 24:69, 1996.
6. Delaire J, Verdon P, Lumineau J, et al: Quelques resultats des tractions extra-orales B appui fronto-mentonnier dans le traitement orthopedique des malformations maxillomandibulaires de classe III et des sequelles osseuses des fentes labio-maxillaires. *Rev Stomatol (Paris)* 73:633, 1972.
7. Engel G, Cornforth G, Damerell JM, et al: Treatment of deep-bite cases. *Am J Orthod* 77:1–13, 1980.
8. Epker BN, Fish LC, Paulus PJ: The surgical–orthodontic correction of maxillary deficiency. *Oral Surg Oral Med Oral Pathol* 46:171, 1978.
9. Epker BN, Turvey TA, Fish LC: Indications for simultaneous mobilization of the maxilla and mandible for the correction of dentofacial deformities. *Oral Surg Oral Med Oral Pathol* 54:369, 1982.
10. Farkas LG, Sohm P, Kolar JC, et al: Inclinations of the facial profile: Art versus reality. *Plast Reconstr Surg* 75:509, 1985.
11. Freudenthaler JW, Celar AG, Schneider B: Overbite depth and anteroposterior dysplasia indicators: The relationship between occlusal and skeletal patterns using the receiver operating characteristic (ROC) analysis. *Eur J Orthod* 22:75–83, 2000.
12. Hart DL, Lundquist DO, Davis HC: The effect of vertical dimension on muscular strength. *J Orthop Sports Phys Ther* 3:57–61, 1981.
13. Hedemark A, Freihofer HP, Jr: The behaviour of the maxilla in vertical movements after Le Fort I osteotomy. *J Maxillofac Surg* 6:244, 1978.
14. Hirdnaka D, Kelly J: Stability of simultaneous orthognathic surgery on the maxilla and mandible: A computer-assisted cephalometric study. *Int J Adult Orthod Orthognath Surg* 2:193, 1987.
15. Isaacson J: Extreme variation in vertical facial growth and associated variation in skeletal and dental relations. *Angle Orthod* 71(40–41):219–229, 1970.

16. Kretschmer WB, Baciut G, Baciut M, et al: Stability of Le Fort I osteotomy in bimaxillary osteotomies: Single-piece versus 3-piece maxilla. *J Oral Maxillofac Surg* 68:372–380, 2010.
17. LaBlanc JP, Turvey TA, Epker BN: Results following simultaneous mobilization of the maxilla and mandible for the correction of dentofacial deformities: Analysis of 100 consecutive patients. *J Oral Surg* 54:607, 1982.
18. Moser K, Freihofer HP: Long-term experience with simultaneous movement of the upper and lower jaw. *J Oral Maxillofac Surg* 8:271, 1980.
19. Moss M, Salentijn L: Differences between the functional matrices in anterior open-bite and in deep-bite. *Am J Orthod* 60:264–280, 1971.
20. Nanda SK: Patterns of vertical growth in the face. *Am J Orthod Dentofac Orthop* 93:103–116, 1988.
21. Nanda RS: Growth patterns in subjects with long and short faces. *Am J Orthod Dentofac Orthop* 98:247–258, 1990.
22. Nemeth RB, Isaacson RJ: Vertical anterior relapse. *Am J Orthod* 65:565–585, 1974.
23. Nielsen, IL: Vertical malocclusions: Etiology, development, diagnosis and some aspects of treatment. *Angle Orthod* 61:247–260, 1991.
24. Norholt SE, Sindet-Pedersen S, Jensen J: An extended Le Fort I osteotomy for correction of midface hypoplasia: A modified technique and results in 35 patients. *J Oral Maxillofac Surg* 54:1297, 1996.
25. Opdebeeck H, Bell W: The short face syndrome. *Am J Orthod* 73:499–511, 1978.
26. Piecuch J, Tideman H, DeKoomen H: Short-face syndrome: Treatment of myofascial pain dysfunction by maxillary disimpaction. *Oral Surg Oral Med Oral Pathol* 49:112–116, 1980.
27. Posnick JC, Fantuzzo J, Orchin J: Deliberate operative rotation of the maxillomandibular complex to alter the A-point to B-point relationship for enhanced facial esthetics. *J Oral Maxillofac Surg* 64:1687–1695, 2006.
28. Posnick JC, Fantuzzo JJ, Troost T: Simultaneous intranasal procedures to improve chronic obstructive nasal breathing in patients undergoing maxillary (Le Fort I) osteotomy. *J Oral Maxillofac Surg* 65:2273–2281, 2007.
29. Posnick JC, Agnihotri N: Managing chronic nasal airway obstruction at the time of orthognathic surgery: A twofer. *J Oral Maxillofac Surg* 69:695–701, 2011
30. Poulton DR: Correction of extreme deep overbite with orthodontics and orthognathic surgery. *Am J Orthod Dentofac Orthop* 96:275–280, 1989.
31. Proffit WR, Sellers KT: The effect of intermittent forces on eruption of the rabbit incisor. *J Dent Res* 65:118–122, 1986.
32. Proffit WR, Phillips C, Dann C IV: Who seeks surgical orthodontic treatment? *Int J Adult Orthodon Orthognath Surg* 3:153, 1990.
33. Proffit WR, Phillips C, Douvartzidis N: A comparison of outcomes of orthodontic and surgical-orthodontic treatment of Class II malocclusion in adults. *Am J Orthod Dentofacial Orthop* 101:556–565, 1992.
34. Proffit WR, Fields HW, Moray LJ: Prevalence of malocclusion and orthodontic treatment need in the United States: estimates from the N-HANES III survey. *Int J Adult Orthodon Orthognath Surg* 13:97–106, 1998.
35. Proffit WR, Fields HW, Sarver DM: *Contemporary orthodontics*, ed 4, St. Louis, 2007, Mosby.
36. Reyneke JP, Evans WG: Surgical manipulation of the occlusal plane. *Int J Adult Orthodon Orthognath Surg* 5:99–110, 1990.
37. Rosen HM: Definitive surgical correction of vertical maxillary deficiency. *Plast Reconstr Surg* 85:215, 1990.
38. Rosen HM, Ackerman JL: Porous block hydroxyapatite in orthognathic surgery. *Angle Orthod* 61:185, 1991.
39. Rosen HM: Facial skeletal expansion: Treatment strategies and rationale. *Plast Reconstr Surg* 89:798, 1992.
40. Rosen H: Aesthetics in facial skeletal surgery. *Perspectives in Plastic Surgery* 6:1, 1993.
41. Rosen HM: Occlusal plane rotation: Aesthetic enhancement in mandibular micrognathia. *Plast Reconstr Surg* 91:1231, 1993.
42. Sassouni V, Nanda S: Analysis of dentofacial vertical proportions. *Am J Orthod* 50:801–823, 1964.
43. Sassouni V: A classification of skeletal facial types. *Am J Orthod* 55:109–122, 1969.
44. Schudy FF: Vertical growth versus anteroposterior growth as related to function and treatment. *Angle Orthod* 34:75–93, 1964.
45. Stahl F, Baccetti T, Franchi L, McNamara JA: Longitudinal growth changes in untreated subjects with class II division malocclusion. *Am J Orthod Dentofacial Orthop* 134:125–137, 2008.
46. Stella JP, Streater MR, Epker BN, Sinn DP: Predictability of upper lip soft tissue changes with maxillary advancement. *J Oral Maxillofac Surg* 47:697, 1989.
47. Throckmorton G, Finn R, Bell W: Biomechanics of differences in lower face height. *Am J Orthod* 77:410–420, 1980.
48. Tollaro I, Baccetti T, Franchi L: Class III malocclusion in the deciduous dentition: A morphological and correlation study. *Eur J Orthod* 16:401–408, 1994.
49. Tollaro I, Baccetti T, Franchi L, Tanasescu CD: Role of posterior transverse interarch discrepancy in class II, division 1 malocclusion during the mixed dentition phase. *Am J Orthod Dentofacial Orthop* 110:417–422, 1996.
50. Trouten JC, Enlow DH, Rabine M, et al: Morphologic factors in open-bite and deep-bite. *Angle Orthod* 53:192–211, 1983.
51. Tsai H-H: Cephalometric studies of children with long and short faces. *J Clin Pediatr Dent* 25:23–28, 2000.
52. Turley PK: Orthodontic management of the short face patient. *Semin Orthod* 2:138–153, 1996.
53. Turvey TA, Hall DJ, Fish LC, Epker BN: Surgical orthodontic treatment planning for simultaneous mobilization of the maxilla and mandible in the correction of dentofacial deformities. *Oral Surg Oral Med Oral Pathol* 54:491, 1982.
54. Van der Dussen FN, Egyedi P: Premature aging of the face after orthognathic surgery. *J Craniofac Surg* 18:335, 1990.
55. Van Sickels JE, Ivey DW: Myofacial pain dysfunction: A manifestation of the short-face syndrome. *J Prosthet Dent* 42:547–550, 1979.
56. Vanarsdale R, White R: Three-dimensional analysis for skeletal problems. *Int J Adult Orthodon Orthognath Surg* 9:159, 1994.
57. Wardrop RW, Wolford LM: Maxillary stability following downgraft and/or advancement procedures with stabilization using rigid fixation and porous block hydroxyapatite implants. *J Oral Maxillofac Surg* 47:336, 1989.
58. Watted N, Bill JS, Witt E: A therapeutic concept for the combined orthodontic surgical correction of Angle Class II deformities with short-face syndrome: Surgical lengthening of the lower face. *Clin Orthod Res* 3:78–93, 2000.
59. Weinberg H, Kronman JH: Orthodontic influence upon anterior face height. *Angle Orthod* 36:80–88, 1966.

24

Bimaxillary Dental Protrusive Growth Patterns with Dentofacial Disharmony

JEFFREY C. POSNICK, DMD, MD

- Functional Aspects
- Clinical Characteristics
- Dental Findings
- Radiographic Findings
- Treatment Pitfalls
- Orthodontic Approach
- Orthognathic and Dentoalveolar Surgical Approaches
- Postsurgical Orthodontic Maintenance and Detailing
- Complications, Informed Consent, and Patient Education
- Malocclusion after Orthodontics and Orthognathic Surgery
- Temporomandibular Disorders: The Effects of Orthodontics and Orthognathic Surgery
- Conclusions

Bimaxillary dental protrusion can occur in any racial group, but facial divergence (i.e., convexity) and lip prominence are strongly influenced by racial and ethnic characteristics.[37,67,95,107] Greater degrees of lip and incisor prominence tend to occur in Asians, Africans, and Latinos as compared with Caucasians.[68,75] In Caucasian populations, dental protrusion is more common among individuals of Mediterranean background than among those from more northern European regions. The Australian aborigines, who are classified as Caucasoid, have greater degrees of facial and dental protrusion. Etiologic factors that favor bimaxillary dental protrusion with anterior open bite include heredity; paraoral habits (e.g., thumb sucking); and the presence of strong protrusive tongue muscle forces in combination with weak bilabial (lip) forces.[103,104,120] The common physical findings include flaccid lips, procumbent maxillary and mandibular incisors, and an anterior open bite.

Drummond compared the facial morphology of Caucasian Americans to that of African Americans.[35] He found that Africans more frequently have a protrusive and strong tongue with loose, flaccid lips. These altered forces during growth are known to gradually push the anterior teeth into a procumbent position. The resulting proclination of the teeth in combination with the thickness of the lips make the lower face appear to be more full. Altemus compared the cephalofacial relationships of Caucasian, African, Chinese, Japanese, Navajo Indian, and Australian aboriginal individuals.[3] He concluded that the relative straightness of an individual's facial profile is very much a reflection of the underlying skeletal parts. He also found that, although the upper facial profiles (i.e., the forehead, orbits, and cheekbones) of the varied racial groups appeared to be remarkably similar, the lower facial profiles (i.e., the maxilla, mandible, and chin region) did not. The position of the anterior teeth and lips of African children were found to be more protrusive as compared with those of Caucasians. Sushner studied 100 lateral facial photographs of "attractive" Africans.[119] He used the Steiner, Holdaway, and Ricketts cephalometric standards to analyze the "attractive" African facial photographs,[53,88] and he concluded that the soft-tissue profiles of the individuals in those photographs were significantly more protrusive than the accepted Caucasian standards. He went on to establish African cephalometric norms. Similar studies completed by Guinn and Connor confirmed that the African soft-tissue profile is more protrusive and significantly different from Caucasian cephalometric norms.[5,24,25,50]

Peck and Peck set out to determine the public's concept of beauty as related to the Caucasian profile.[94] The authors found that the general public admires a more full face as compared with the accepted cephalometric normative data and clinical orthodontic standards of the day (e.g., four bicuspid extractions with orthodontic retraction). These

authors pointed out that the "ultimate source of our aesthetic values should be the people [i.e., public opinion], not just ourselves [i.e., clinicians]." Foster studied facial aesthetic preferences among various groups by using profile silhouettes.[44] The evaluators included Africans, Caucasians, and Chinese individuals; art students; general dentists; and orthodontists. The results confirmed a common aesthetic preference for the position of the lips in profile to be within 1 mm to 2 mm of each other. Another finding was that all groups consistently associated fuller lips with a more youthful appearance. Farrow and colleagues attempted to discover what African Americans find attractive about their own profiles.[40] The study results found that the preferred aesthetic profile involved slight convexity and more protrusion as compared with standard Caucasian cephalometric norms. A review of the literature confirms that, as a group, African Americans differ from Caucasian Americans with regard to dental, skeletal, and soft-tissue facial parameters.[2,31,43,58,122] For both groups, facial convexity is a preferred feature.[28,30,76,77,100] However, the question remains: at what point does attractive fullness (i.e., convexity) of the profile become dysmorphic and unattractive?

Distorted vertical and horizontal growth of the maxillomandibular skeleton in association with bimaxillary dental protrusion will result in both facial and dental disharmony.[84] This growth pattern may be associated with Class I, Class II, and Class III molar relationships, but it always occurs with mandibular and maxillary incisor procumbency and anterior open-bite malocclusion.[5,47,52,85,113,118] Individuals with bimaxillary dental protrusion will also have varying degrees of lip incompetence and mentalis strain; exposure of the anterior teeth in repose and gingival show with smile; and, typically, a dysmorphic chin in profile. If these aspects become excessive, then the diagnosis of an associated dentofacial disharmony is appropriate.[109]

The generally offered correction of the anterior open-bite malocclusion associated with bimaxillary dental protrusion is the extraction of four premolars followed by the orthodontic retraction of the anterior teeth into the edentulous gaps.[6,121,126] This treatment also tends to retract the lips and to reduce facial convexity.[33,34,48,72] To determine which individuals also have significant dentofacial (jaw) disharmony and thus will benefit from an orthognathic approach (i.e., the surgical repositioning of the upper and lower jaws), the clinician should assess pretreatment skeletal, dental, and soft-tissue parameters. This includes the following:

- An evaluation of the maxillomandibular skeleton (i.e., horizontal and vertical dimensions)
- An assessment of the dental condition (i.e., incisor inclination, spacing and crowding, the curve of Spee, arch width, overjet, and overbite)
- An evaluation of the soft-tissue static and dynamic position of the lips (i.e., the lip-to-tooth relationship in repose and with smile, the interlabial gap, the degree of mentalis strain with closure, and the nasolabial and labiomental angles).

Functional Aspects

- Wide lip separation with excessive effort required to achieve lip seal is present.
- Chronic obstructive nasal breathing as a result of increased intranasal airway resistance is frequent (see Chapter 10).
- Classic articulation errors in speech caused by the anterior open-bite malocclusion are expected (see Chapter 8).
- Chewing and swallowing difficulties as a result of the anterior open-bite malocclusion and lip incompetence are expected (see Chapter 8).
- Detrimental long-term effects on the dentition and the periodontium caused by traumatic occlusal forces, the spacing of the teeth in the alveolar housing, or previous camouflage orthodontic treatment may be seen (see Chapter 5 and 6).
- Temporomandibular disorder (TMD) should be looked for but is not generally problematic in these individuals (see Chapter 9).

Clinical Characteristics

- Separation of the lips at rest of >4 mm
- Excessive dental show with the upper lip at rest and excessive gingival show with a broad smile
- Flaccid lips with more effort required to bring them into closure (i.e., mentalis strain and lip incompetence)
- Excessive prominence of the lips with everted vermillion borders when viewed in profile
- Typically a mouth-breathing habit and an open-mouth posture with a protrusive tongue
- Convex maxillomandibular profile
- Chin deformity frequently seen in profile

Dental Findings

- Anterior open bite
- Maxillary and mandibular incisor proclination
- Exaggerated curve of Spee in the maxilla and a reverse curve in the mandible
- Frequently with narrow maxillary arch width
- Frequently with a Class II molar relationship, although a Class I or Class III malocclusion may be present
- Frequently with interdental spacing in the anterior dentition of the maxilla and mandible
- Despite incisor proclination, presence of adequate periodontal support without gingival recession, labial bone dehiscence, or fenestration is typical

Radiographic Findings

- Procumbency of the maxillary incisors
- Procumbency of the mandibular incisors
- Lack of chin prominence at the pogonion is generally notable

- Anterior open bite measured at the incisal edges
- Excessive lower anterior facial height (i.e., the distance measured from nasion to the pogonion)

Treatment Pitfalls

When considering orthognathic surgery for the individual with bimaxillary dental protrusion, there must be a compelling aesthetic or functional rationale for the shortening of the anterior vertical dimension of the face and the alteration of the horizontal projection (Figs. 24-1 through 24-11).[18,41,46,64,65,80,115] When present, facial aesthetic disharmony in association with bimaxillary dental protrusion is generally characterized by the following:

- *Excessive separation of the lips at rest (i.e., lip incompetence).* As a general rule, lip separation at rest should be less than 4 mm.
- *Excessive incisor show with the upper lip at rest.* As a general rule, more than 50% of the vertical height of the incisor should be seen to be considered excessive.
- *Excessive effort required to bring the lips into closure (i.e., mentalis strain).*
- *Significant vertical and horizontal facial disproportion when viewed in profile.*

All four characteristics are preferred to make the diagnosis of a significant dentofacial disharmony in association with bimaxillary dental protrusion to the extent that the surgical alteration of the facial height and facial projection through orthognathic surgery is recommended.

NOTE: An individual's degree of soft-tissue lip prominence may be independent of jaw and tooth position. The establishment of reasonable skeletal harmony and dental occlusion is not a guarantee of the achievement of preferred soft tissue lip shape and function.

When planning the orthognathic correction, a determination is made of how much incisor procumbency is too much.[96] Anterior teeth that protrude excessively are likely to be associated with lips that are prominent, everted, and separated at rest by more than 3 mm to 4 mm (i.e., lip incompetence). This will cause the individual to strain in an attempt to bring the lips together over the protruding teeth. Retracting (i.e., uprightening) the incisors with the successful closure of the open bite by whatever means tends to decrease facial convexity when the face is viewed in profile.[86,93,111,112,116] If the lips are prominent but are able to comfortably close over the teeth without excessive strain, then the lip posture (i.e., the position at rest) is largely independent of the current jaw and tooth position and more a result of lip muscle tone.[16,17,91,101,106,123] For such an individual, achieving correction of incisor procumbency and the open bite will have only a limited impact on lip function and prominence.[4,42,90,128]

Orthodontic Approach

Historical Perspective

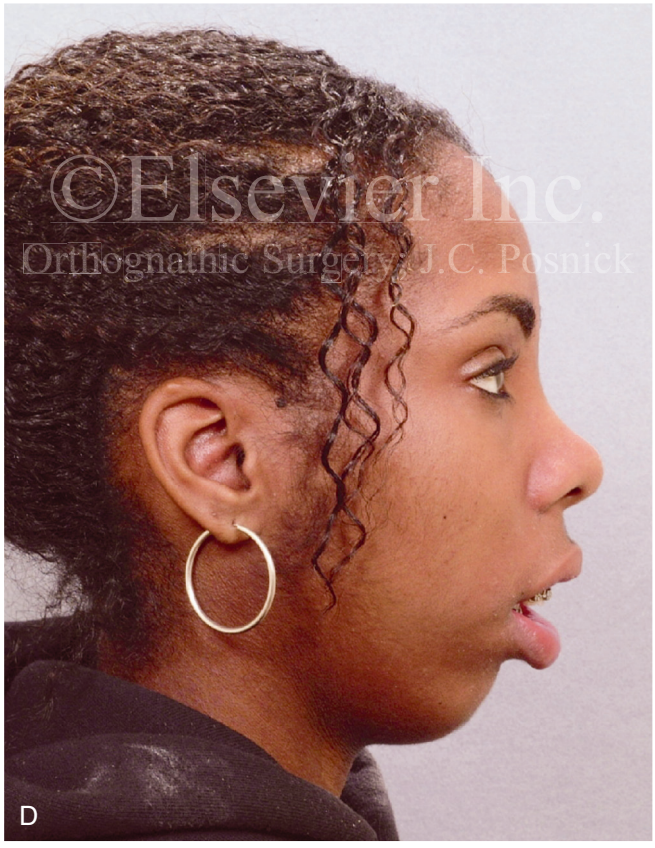
During the early 1900s, the uncompromising non-extraction orthodontic approach set forth by Angle was the treatment of the day (see Chapter 2).[5] Angle used full-banded appliances to expand the arches as a method of bringing the teeth into ideal alignment, no matter how much additional protrusion was created in the process. Calvin Case disagreed with Angle's treatment concepts for the bimaxillary protrusive anterior open-bite problem and stated that *"one of the most dangerous features of the Angle classification is the universally applied teaching that when the dentures [teeth] are placed in normal occlusion, the facial outlines will take care of themselves."*[19] For the individual with bimaxillary dental protrusion, Case proposed bicuspid extraction in all four quadrants, followed by the placement of full-banded orthodontic appliances to retract the procumbent incisors into the extraction sites. It was not until well after Case's death that these techniques for orthodontic management were popularized by two of Angle's own students. Charles Tweed[124] in the United States and Raymond Begg[10] in Australia reintroduced extraction treatment into everyday orthodontic practice. The mainstay of treatment for dentoalveolar protrusion with anterior open bite continues to involve the removal of a premolar in each quadrant followed by orthodontic retraction of the protruded anterior teeth (see Chapter 17).[32,36,60,61,62,73,82]

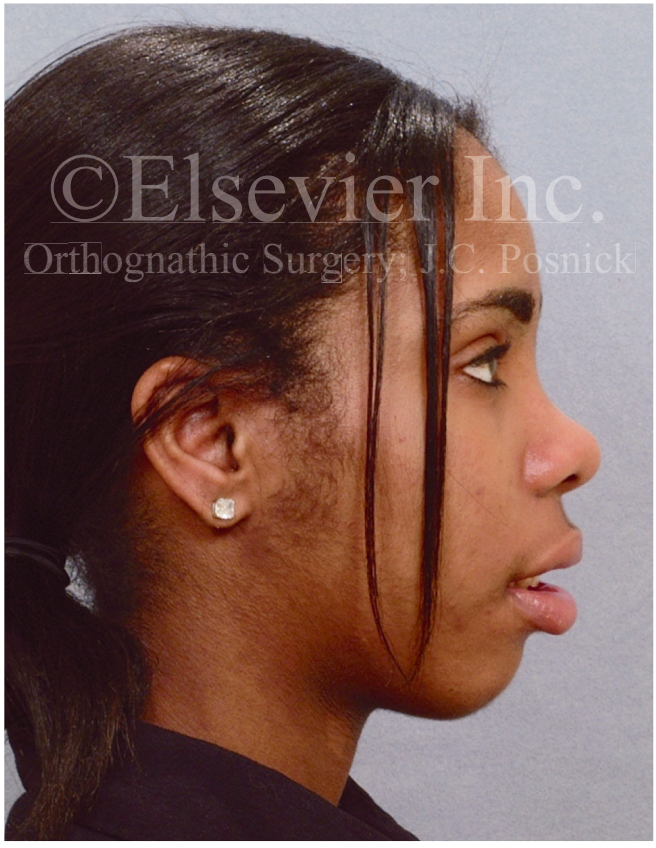
Review of the Literature

There is a definite association between bimaxillary skeletal (jaw) disharmony and dentoalveolar (dental) protrusion with open bite. The standard treatment for the dentoalveolar aspects of this condition is four bicuspid extractions and then the use of maximum anchorage to orthodontically retract the anterior dentition to achieve space closure; this will also tend to decrease the chief complaint of lip fullness. The side effects of this form of treatment may include root resorption; excess lingual tipping of the anterior teeth; remodeling with dehiscence and fenestration of the cortical plate; insufficient retraction as a result of anchorage loss; and even more pronounced incisor exposure (i.e., gummy smile). Orthodontic technical maneuvers may also include the use of mini-screws as temporary anchorage devices (TADs), torque control of the upper and lower incisors, and alveolar corticotomies followed by more rapid orthodontic retraction into the bicuspid extraction sites.[7,21,22,55,56,59,69,71,78,83,87,89,92,125,127] These options also run the risk of the previously mentioned negative sequelae, but they are believed to shorten overall treatment time.

Text continued on p. 939

• **Figure 24-1** A 16-year-old girl was referred by her orthodontist for the evaluation of lip incompetence, anterior open bite, gummy smile, weak chin, and nasal obstruction. Orthodontics had previously been carried out without extraction therapy. The patient presented with a Class II anterior open bite dental protrusive malocclusion. A combined orthodontic and surgical approach was recommended. With non-extraction orthodontic decompensation complete, the patient's surgery included maxillary Le Fort I osteotomy in segments (arch expansion, correction of the curve of Spee, vertical intrusion, minimal horizontal advancement and counterclockwise rotation); sagittal split ramus osteotomies (horizontal advancement and counterclockwise rotation); osseous genioplasty (vertical reduction and horizontal advancement); and septoplasty, inferior turbinate reduction, and nasal floor recontouring. **A,** Frontal views in repose before and after treatment. **B,** Frontal views with smile before and after treatment.





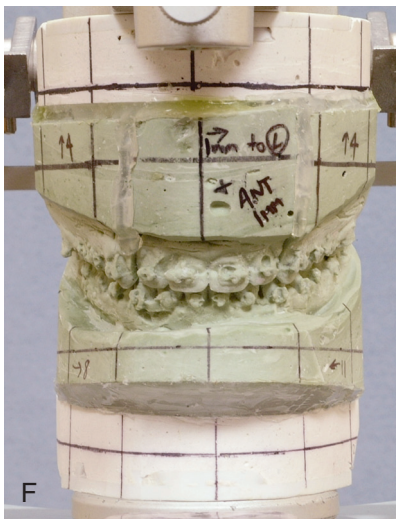
• **Figure 24-1, cont'd C,** Oblique facial views before and after treatment. **D,** Profile views before and after treatment.

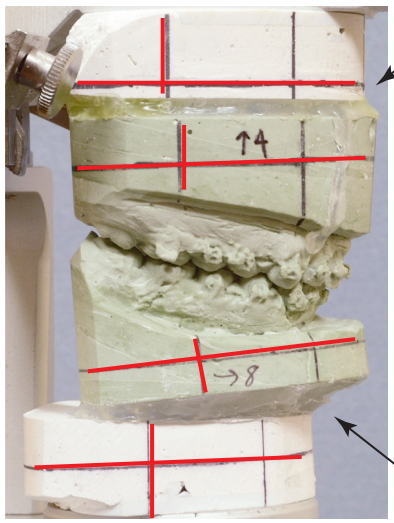
Continued

Prior to retreatment

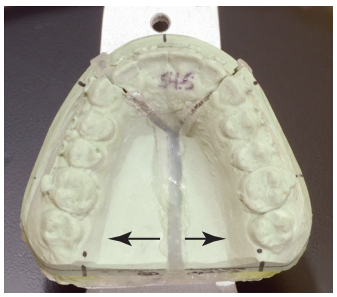
Pre surgery

After treatment





Maxillary counterclockwise rotation



Arch expansion

Mandibular counterclockwise rotation

• **Figure 24-1, cont'd** **E,** Occlusal views before retreatment, with orthodontics in progress, and after treatment. **F,** Articulated dental casts that indicate analytic model planning.

• **Figure 24-2** A 28-year-old woman arrived for the evaluation of malocclusion, gummy smile, lip incompetence, and nasal obstruction. She presented with a Class III anterior open-bite dental protrusive malocclusion. She underwent a combined orthodontic and surgical approach without extractions. The patient's surgery included maxillary Le Fort I osteotomy in segments (transverse expansion, vertical intrusion, minimal advancement, and clockwise rotation); sagittal split ramus osteotomies (minimal advancement and counterclockwise rotation); osseous genioplasty (vertical reduction and horizontal advancement); and septoplasty, inferior turbinate reduction, and nasal floor recontouring. **A,** Frontal views in repose before and after treatment. **B,** Frontal views with smile before and after treatment.

Continued

• **Figure 24-2, cont'd** **C,** Oblique facial views before and after treatment. **D,** Profile views before and after treatment.

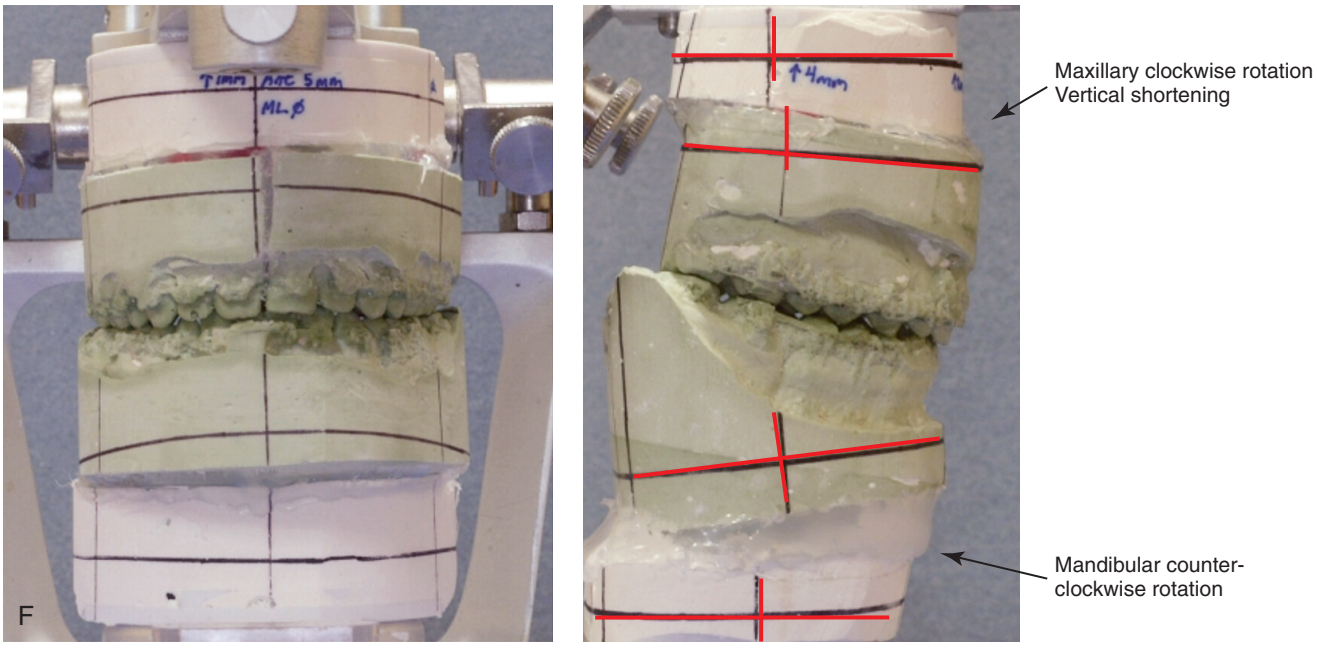
Prior to treatment

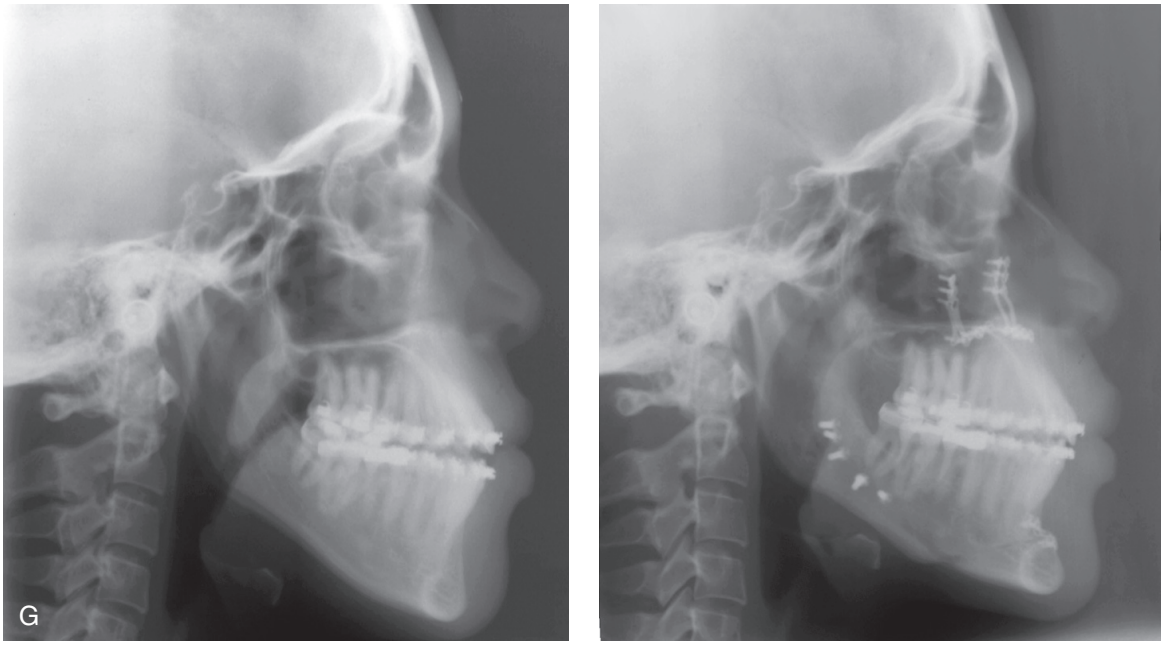
Pre surgery

After treatment

• **Figure 24-2, cont'd E,** Occlusal views before treatment, with orthodontics in progress, and 3 years after treatment.

Continued





• **Figure 24-2, cont'd** **F,** Articulated dental casts that indicate analytic model planning. **G,** Lateral cephalometric radiographs before and after treatment.

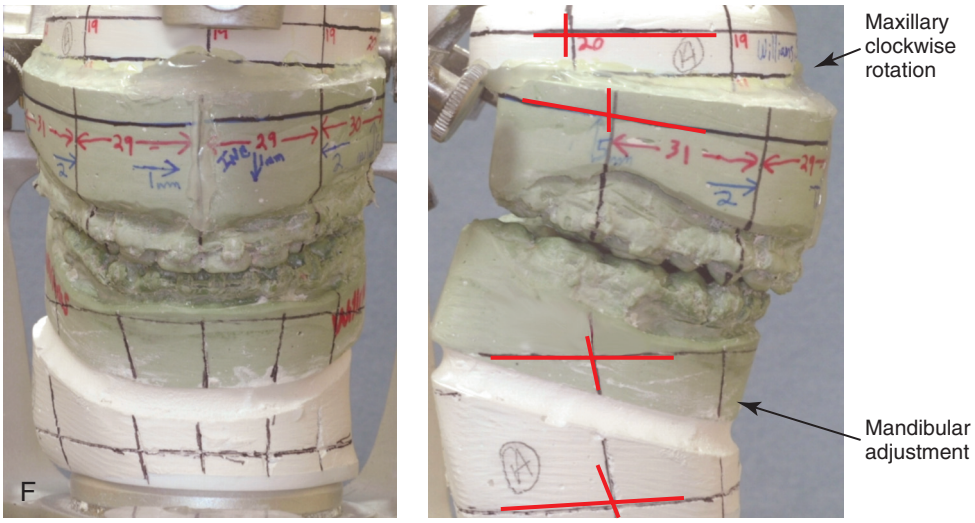
• **Figure 24-3** A 24-year-old woman was referred by her orthodontist for surgical evaluation. She presented with a Class III anterior open-bite dental protrusive malocclusion. She underwent four bicuspid extractions with orthodontic decompensation followed by orthognathic surgery. Her procedures included maxillary Le Fort I osteotomy in segments (clockwise rotation, horizontal advancement, arch expansion, and vertical shortening); sagittal split ramus osteotomies (minimal clockwise rotation); osseous genioplasty (horizontal advancement); and septoplasty, inferior turbinate reduction, and nasal floor recontouring. **A,** Frontal views in repose before and after treatment. **B,** Frontal views with smile before and after treatment.

Continued

• **Figure 24-3, cont'd C,** Oblique facial views before and after treatment. **D,** Profile views before and after treatment.

Pre surgery after four bicuspid extractions

After treatment



• **Figure 24-3, cont'd** **E,** Occlusal views with orthodontics in progress and after treatment. **F,** Articulated dental casts that indicate analytic model planning.

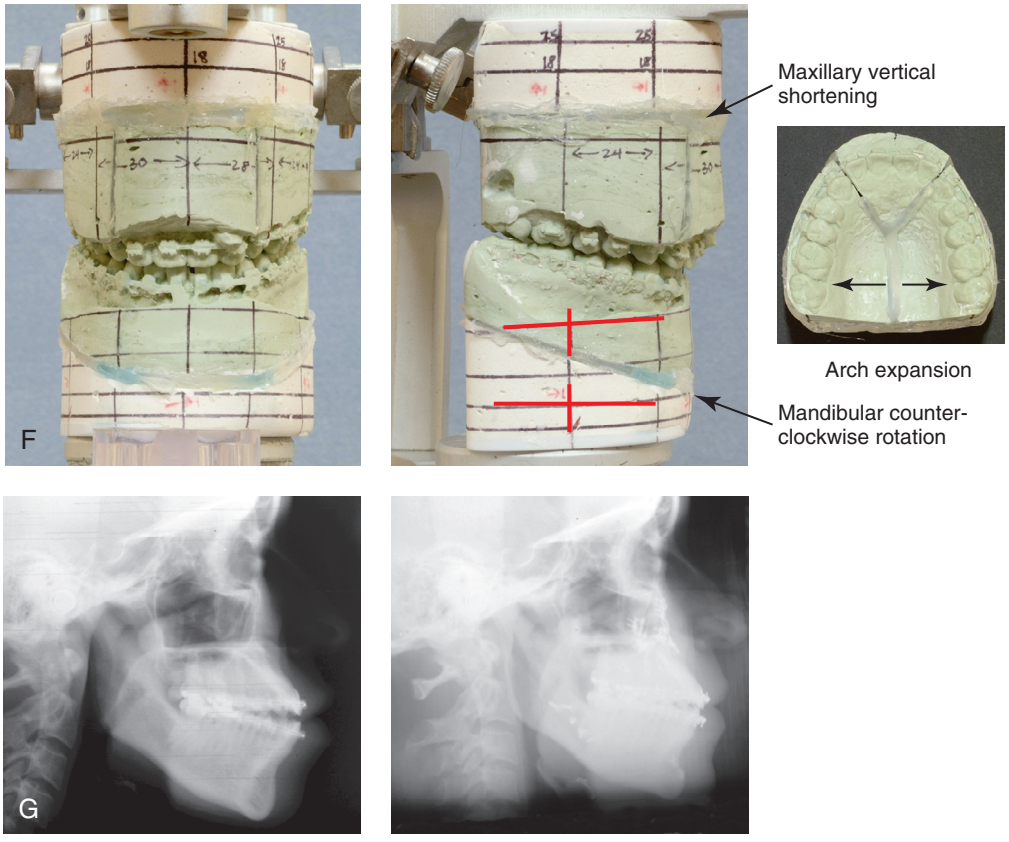
• **Figure 24-4** A 28-year-old Hispanic woman was referred by her orthodontist for the evaluation of malocclusion and lip incompetence. She was found to have a Class III anterior open-bite malocclusion and lifelong nasal obstruction. She underwent non-extraction orthodontic decompensation followed by surgery. Her procedures included maxillary Le Fort I osteotomy in segments (correction of the arch form); sagittal split ramus osteotomies (minimal counterclockwise rotation); osseous genioplasty (horizontal advancement); and septoplasty, inferior turbinate reduction, and nasal floor recontouring. **A,** Frontal views in repose before and after treatment. **B,** Frontal views with smile before and after treatment.

• **Figure 24-4, cont'd C,** Oblique facial views before and after treatment. **D,** Profile views before and after treatment.

Continued

Prior to treatment

After treatment



• **Figure 24-4, cont'd** **E,** Occlusal views before orthodontics and after treatment. Note the limited overjet as a result of mild dental relapse. **F,** Articulated dental casts that indicate analytic model planning. **G,** Lateral cephalometric radiographs before and after treatment.

• **Figure 24-5** An 18-year-old Asian man was referred for surgical evaluation. He presented with a Class III anterior open-bite dental protrusive malocclusion. Concerns included nasal obstruction, gingivitis, gummy smile, anterior open-bite malocclusion, lip incompetence, and retrusive profile. Orthodontic decompensation was carried out and included four bicuspid extractions. The patient's surgery included maxillary Le Fort I osteotomy in segments (vertical shortening, minimal horizontal advancement, counterclockwise rotation, and transverse widening); sagittal split ramus osteotomies (minimal horizontal advancement and counterclockwise rotation); osseous genioplasty (vertical shortening and horizontal advancement); and septoplasty, inferior turbinate reduction, and nasal floor recontouring. **A,** Frontal views in repose before and after treatment. **B,** Frontal views with smile before and after treatment.

Continued

• **Figure 24-5, cont'd** **C,** Oblique facial views before and after treatment. **D,** Profile views before and after treatment.

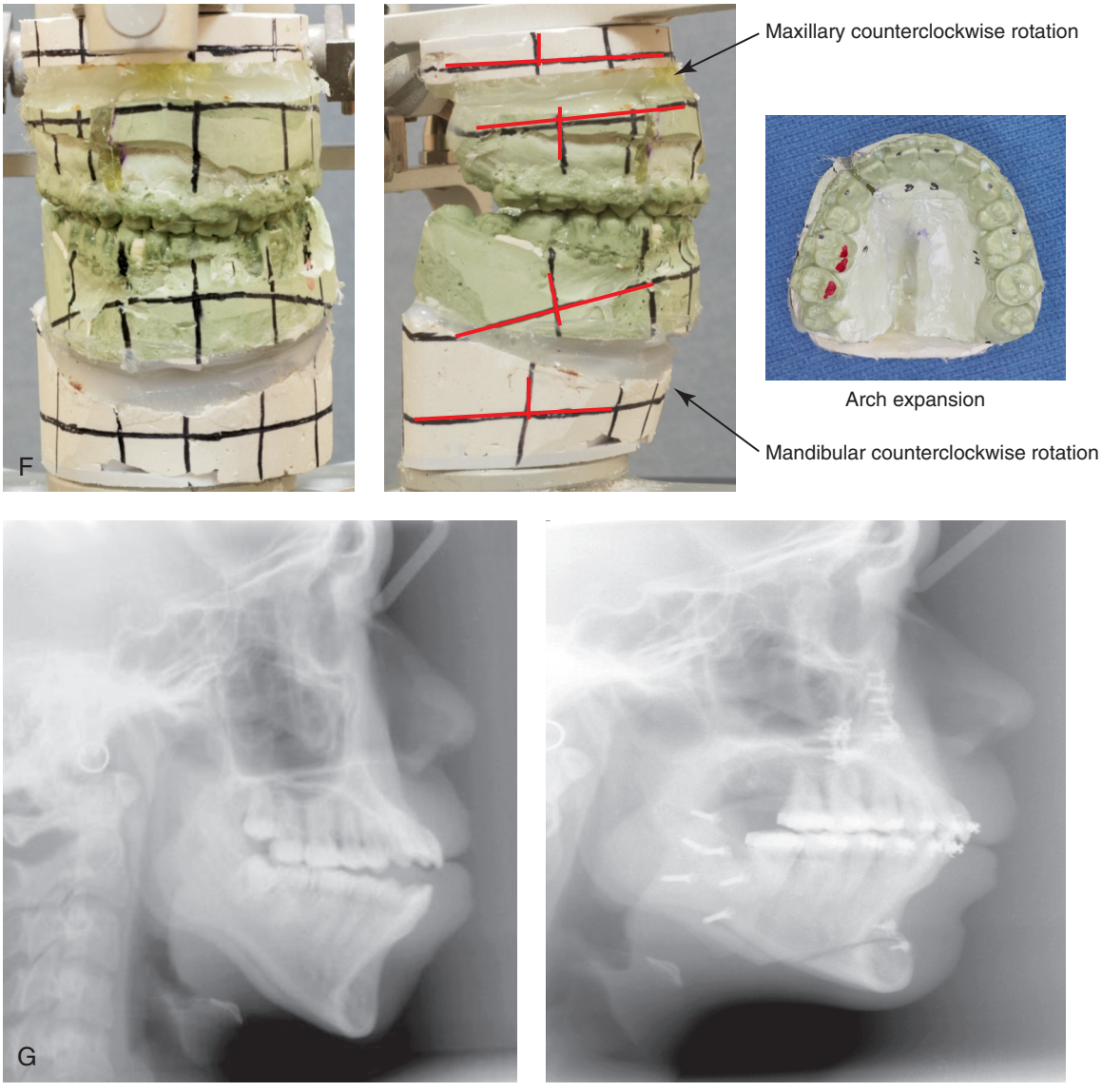
Prior to treatment

Pre surgery after four bicusbid extractions

After treatment

• **Figure 24-5, cont'd E,** Occlusal views before treatment, before surgery with four bicuspid extractions, and after treatment.

Continued



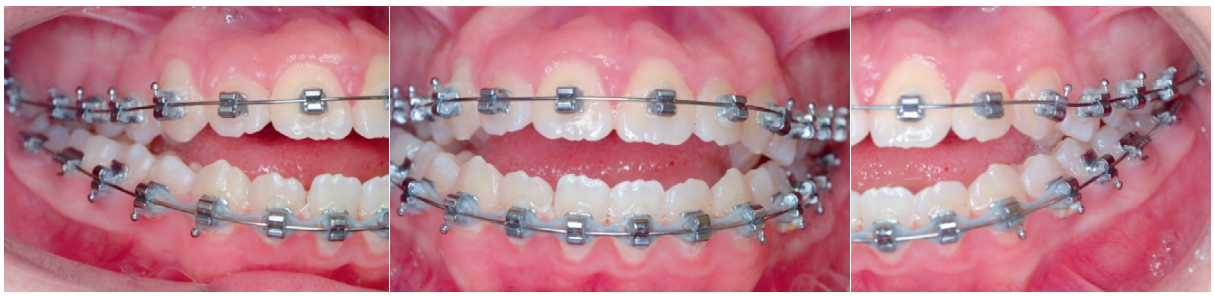
• **Figure 24-5, cont'd** **F,** Articulated dental casts that indicate analytic model planning. **G,** Lateral cephalometric radiographs before and after treatment. Despite the bicuspid extractions, procumbency remains.

• **Figure 24-6** One of two 15-year-old twin girls who was referred for surgical evaluation. She presented with a Class III anterior open-bite dental protrusive malocclusion. Orthodontic treatment was carried out without extractions, and maxillary incisor procumbency remained. The patient's surgery included maxillary Le Fort I osteotomy in segments (horizontal advancement, vertical shortening, clockwise rotation, and arch expansion); sagittal split ramus osteotomies (minimal horizontal advancement and counterclockwise rotation); osseous genioplasty (vertical shortening and horizontal advancement); and septoplasty, inferior turbinate reduction, and nasal floor recontouring. **A,** Frontal views in repose before and after treatment. **B,** Frontal views with smile before and after treatment.

Continued

• **Figure 24-6, cont'd C,** Oblique facial views before and after treatment. **D,** Profile views before and after treatment.

Pre treatment

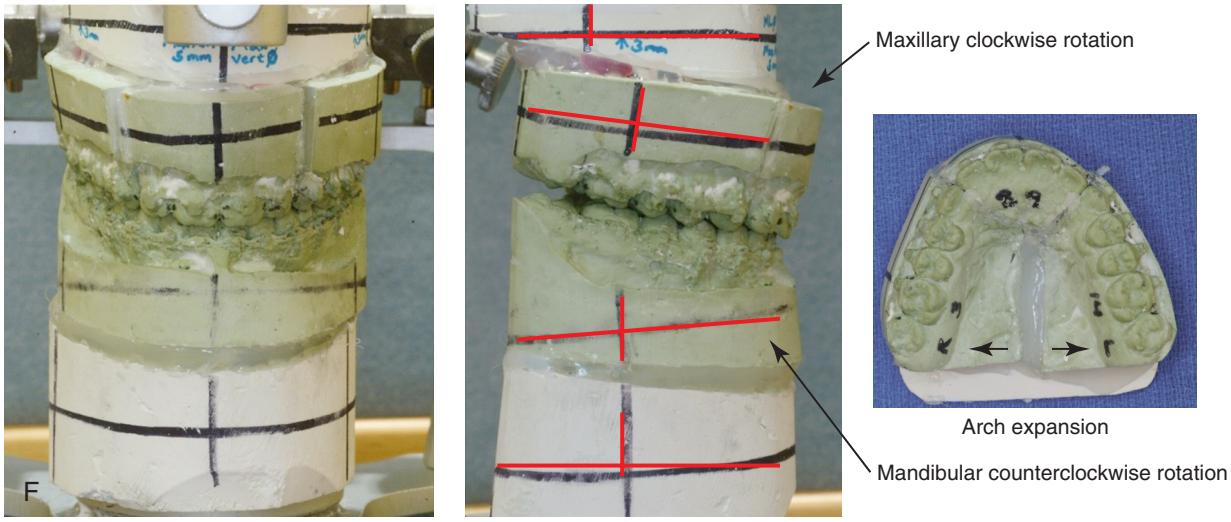


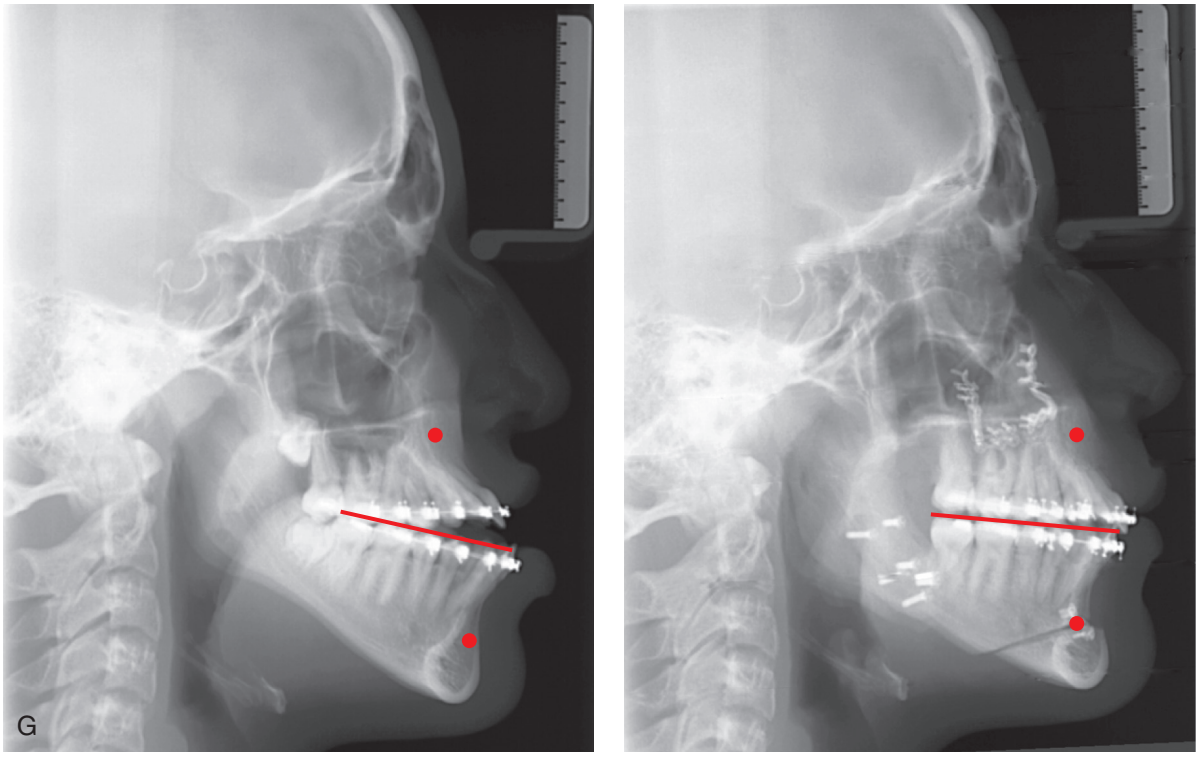
Pre surgery

After treatment

• **Figure 24-6, cont'd E,** Occlusal views before orthodontics, before surgery, and after treatment.

Continued





• **Figure 24-6, cont'd** **F,** Articulated dental casts that demonstrate analytic model planning. **G,** Lateral cephalometric radiographs before and after treatment.

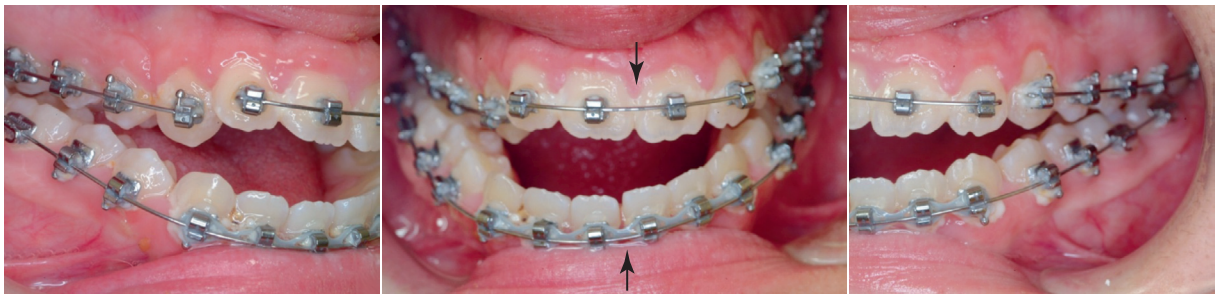
• **Figure 24-7** The 15-year-old twin sister of the patient shown in Figure 24-6 was also referred for surgical evaluation. She underwent a combined orthodontic and surgical approach without extractions. She presented with a Class III anterior open-bite dental protrusive malocclusion. The patient's surgery included maxillary Le Fort I osteotomy in segments (horizontal advancement, vertical shortening, clockwise rotation, and arch expansion); sagittal split ramus osteotomies (horizontal advancement and counterclockwise rotation); osseous genioplasty (vertical shortening and horizontal advancement); and septoplasty, inferior turbinate reduction, and nasal floor recontouring. **A,** Frontal views in repose before and after treatment. **B,** Frontal views with smile before and after treatment.

Continued

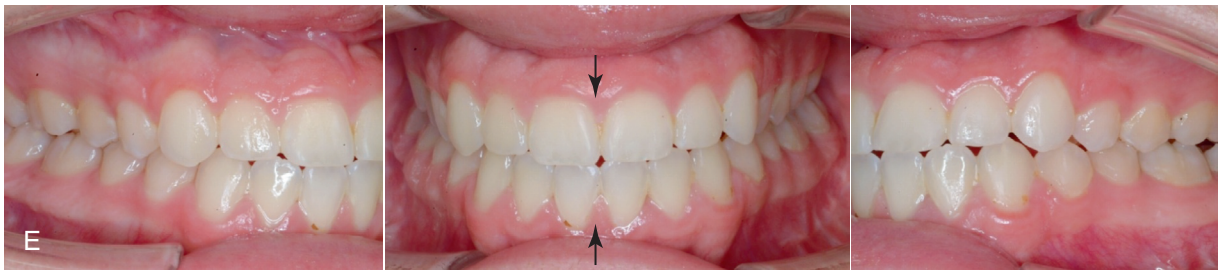
©Elsevier Inc.
Orthognathic Surgery; J.C. Posnick

• **Figure 24-7, cont'd C,** Oblique facial views before and after treatment. **D,** Profile views before and after treatment.

Prior to treatment



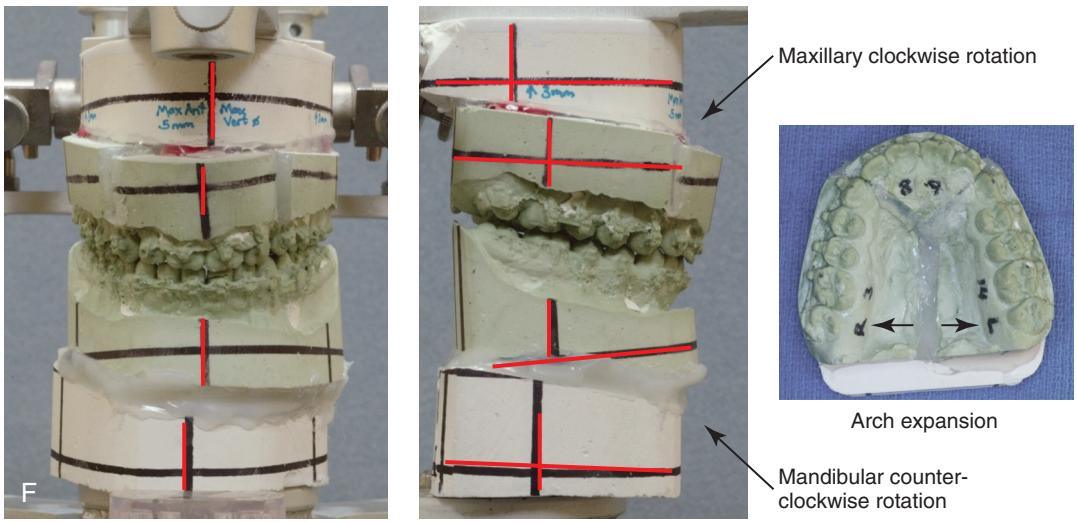
Pre surgery

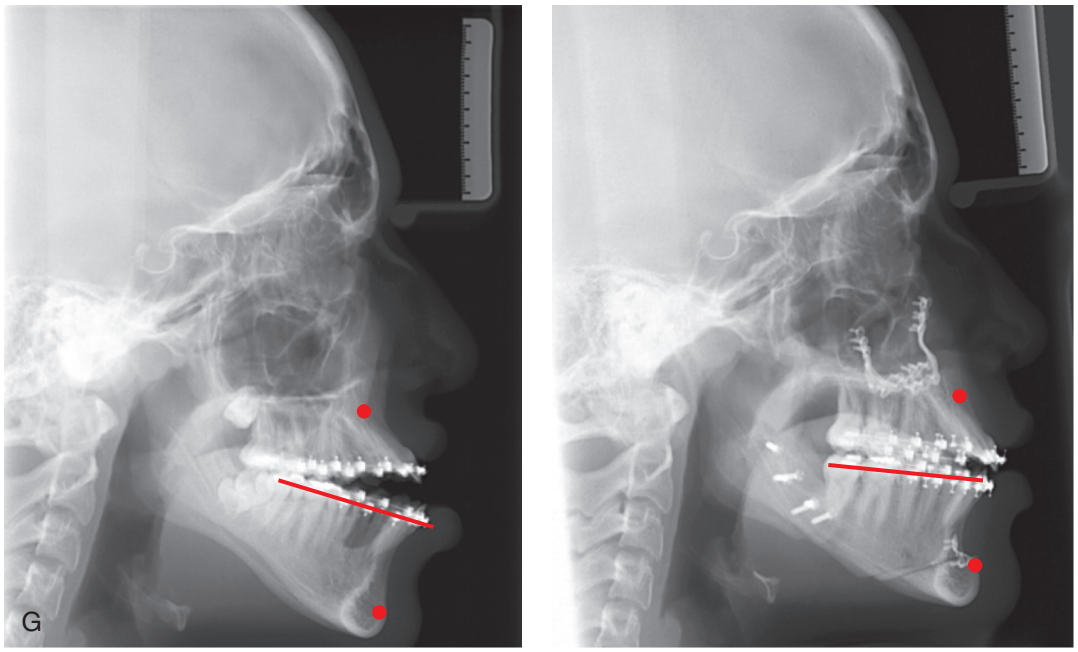


After treatment

• **Figure 24-7, cont'd E,** Occlusal views before treatment, before surgery, and after treatment.

Continued





• **Figure 24-7, cont'd** **F,** Articulated dental casts that indicate analytic model planning. **G,** Lateral cephalometric radiographs before and after treatment.

• **Figure 24-8** A 28-year-old woman arrived for the evaluation of malocclusion,, lip incompetence, nasal obstruction, and anterior open bite malocclusion. She had previously undergone Phase I orthodontics that included rapid palatal expansion and an attempt at growth modification before graduating from high school. She now presented with a Class II anterior open-bite dental protrusive malocclusion. She underwent a combined orthodontic and surgical approach without extractions. The patient's surgery included maxillary Le Fort I osteotomy in segments (vertical intrusion, horizontal advancement, correction of the curve of Spee, and arch width expansion); sagittal split ramus osteotomies (advancement and counterclockwise rotation); and septoplasty and inferior turbinate reduction. **A,** Frontal views in repose before and after treatment. **B,** Frontal views with smile before and after treatment.

Continued

• **Figure 24-8, cont'd C,** Oblique facial views before and after treatment. **D,** Profile views before and after treatment.

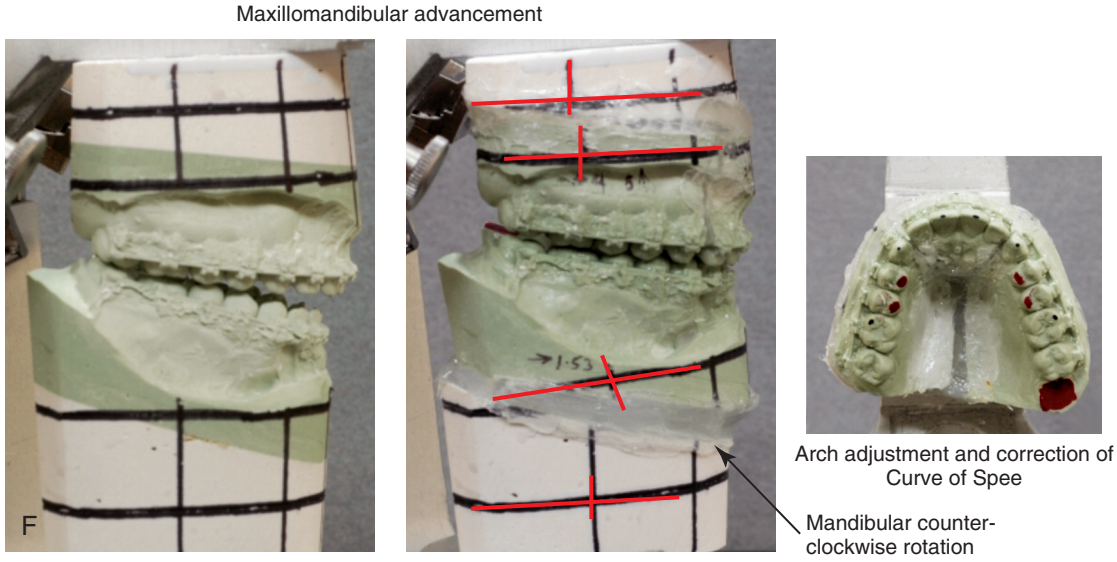
Prior to re-treatment

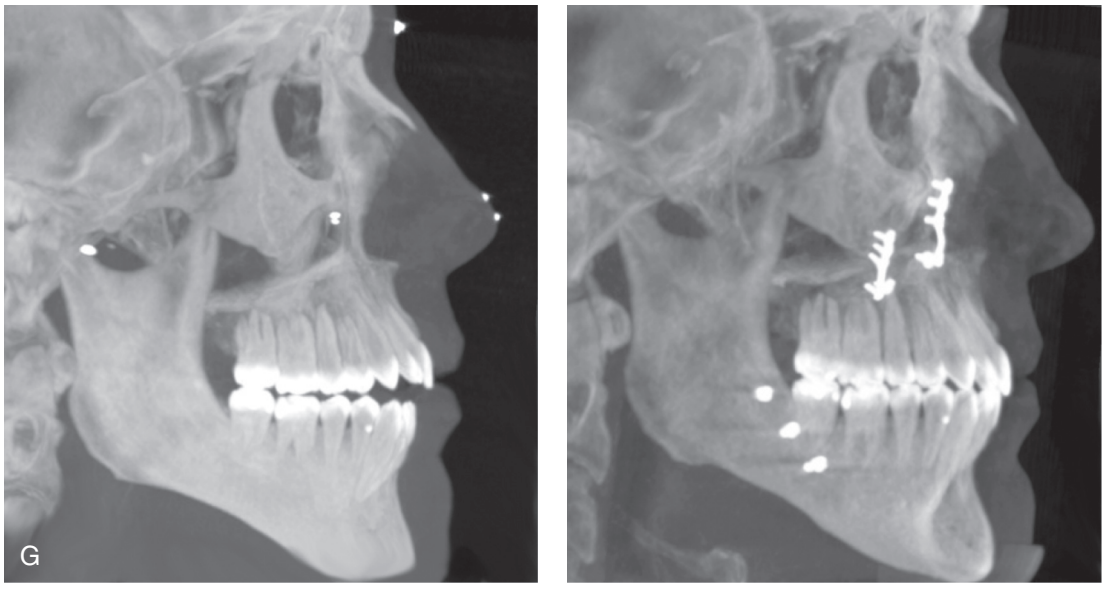
Prior to surgery

After surgery

• **Figure 24-8, cont'd E,** Occlusal views before treatment, with orthodontics in progress, and after treatment.

Continued





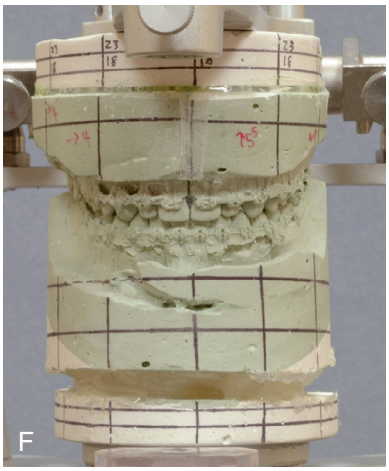
• **Figure 24-8, cont'd F,** Articulated dental casts that indicate analytic model planning. **G,** Lateral cephalometric radiographs before and after treatment.

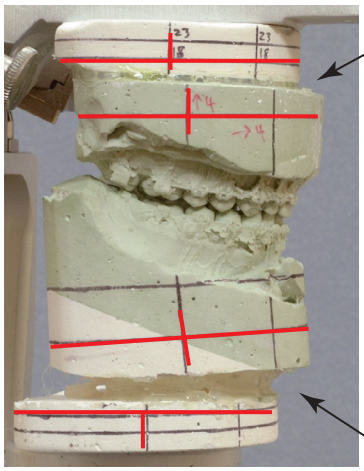
• **Figure 24-9** A 17-year-old Caucasian boy was referred for the surgical evaluation of gummy smile, lip incompetence, and Class II anterior open-bite protrusive malocclusion. He presented with orthodontics in progress. The patient's surgery included maxillary Le Fort I osteotomy in segments (horizontal advancement, vertical shortening, and arch expansion); sagittal split ramus osteotomies (horizontal advancement and counterclockwise rotation); osseous genioplasty (vertical shortening and horizontal advancement); and septoplasty, inferior turbinate reduction, and nasal floor recontouring. **A,** Frontal views in repose before and after treatment. **B,** Frontal views with smile before and after treatment. *Continued*

• **Figure 24-9, cont'd C,** Oblique facial views before and after treatment. **D,** Profile views before and after treatment.

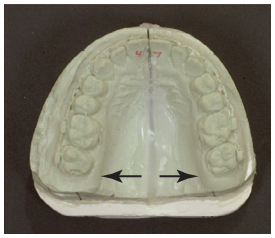
Pre surgery

After treatment



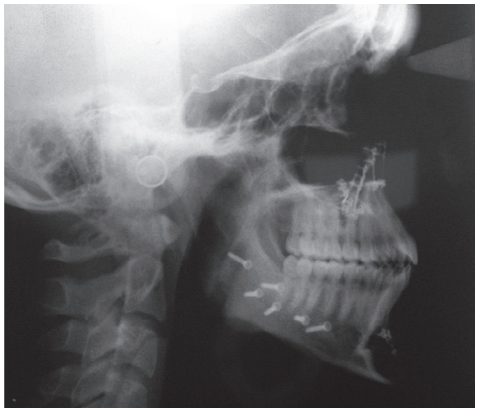


Maxillary vertical intrusion



Arch expansion

Mandibular counter-clockwise rotation



• **Figure 24-9, cont'd** **E,** Occlusal views before surgery and after treatment. **F,** Articulated dental casts that indicate analytic model planning. **G,** Lateral cephalometric radiographs before and after treatment.

• **Figure 24-10** A 22-year-old Caucasian man was referred for the surgical evaluation of an anterior open-bite bimaxillary protrusive malocclusion. He has lip incompetence with mentalis strain and limited horizontal jaw projection, and his nasal airway was open without obstruction. He agreed to a combined orthodontic and surgical approach. The patient's procedures included maxillary Le Fort I osteotomy in segments (horizontal advancement, clockwise rotation, and arch form correction); sagittal split ramus osteotomies (horizontal advancement and counterclockwise rotation); and osseous genioplasty (horizontal advancement). **A,** Frontal views in repose before and after treatment. **B,** Frontal views with smile before and after treatment.

©Elsevier Inc.
Orthognathic Surgery; J.C. Posnick

• **Figure 24-10, cont'd C,** Oblique facial views before and after treatment. **D,** Profile views before and after treatment.

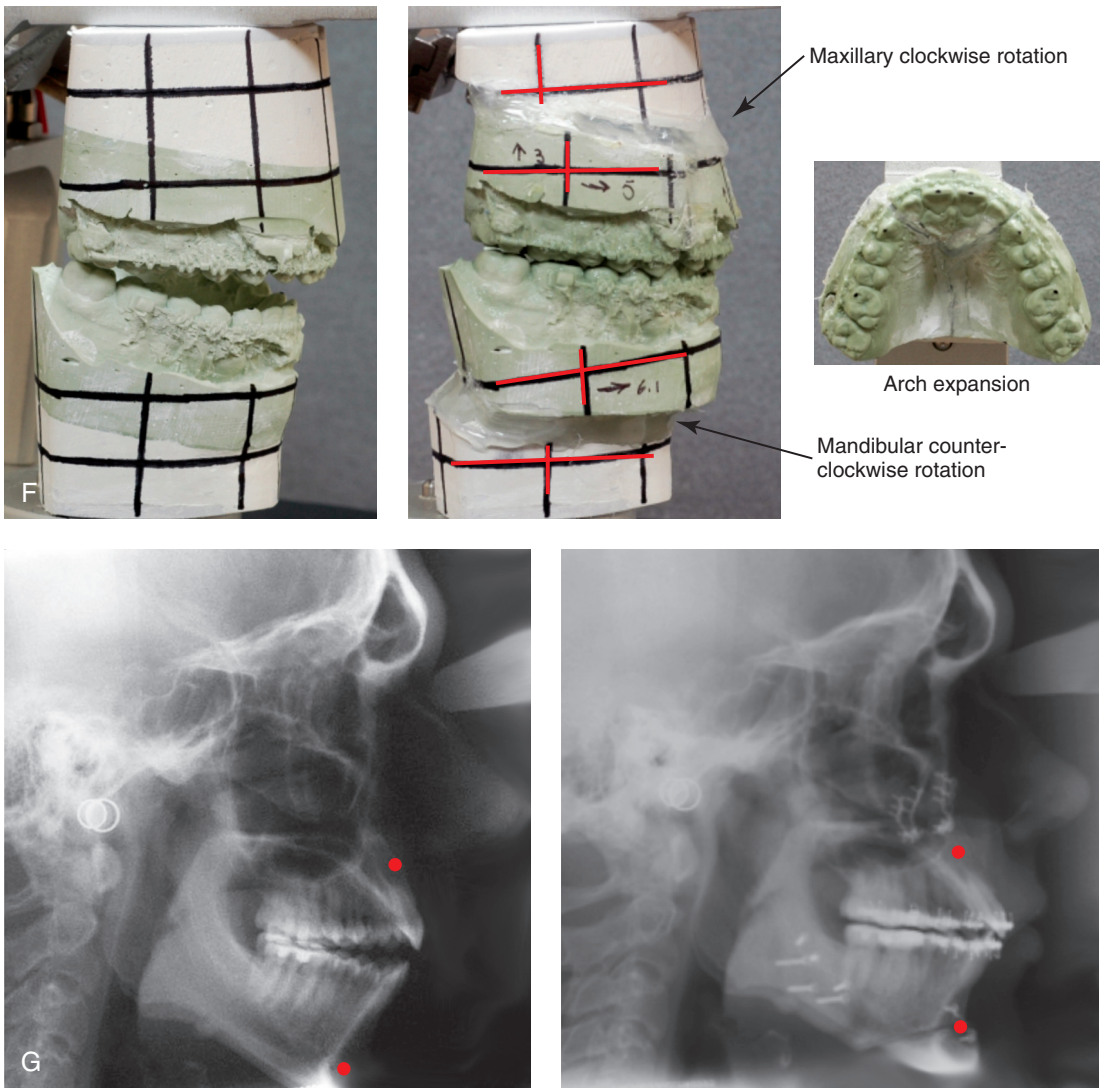
Continued

Pre treatment

Pre surgery

After treatment

• **Figure 24-10, cont'd E,** Occlusal views before orthodontics, before surgery and after treatment.



• **Figure 24-10, cont'd F,** Articulated dental casts that indicate analytic model planning. **G,** Lateral cephalometric radiographs before and after treatment.

• **Figure 24-11** A 20-year-old Caucasian man was referred for the surgical evaluation of horizontal deficiency of both jaws and anterior open-bite malocclusion. Orthodontics had previously been tried without success. The patient had a lifelong history of nasal obstruction. He agreed to a combined orthodontic and surgical approach. The patient's procedures included maxillary Le Fort I osteotomy in segments (horizontal advancement and arch form correction); sagittal split ramus osteotomies (horizontal advancement and counterclockwise rotation); osseous genioplasty (vertical shortening and horizontal advancement); and septoplasty, inferior turbinate reduction, and nasal floor recontouring. **A,** Frontal views in repose before and after treatment. **B,** Frontal views with smile before and after treatment.

• **Figure 24-11, cont'd C,** Oblique facial views before and after treatment. **D,** Profile views before and after treatment.

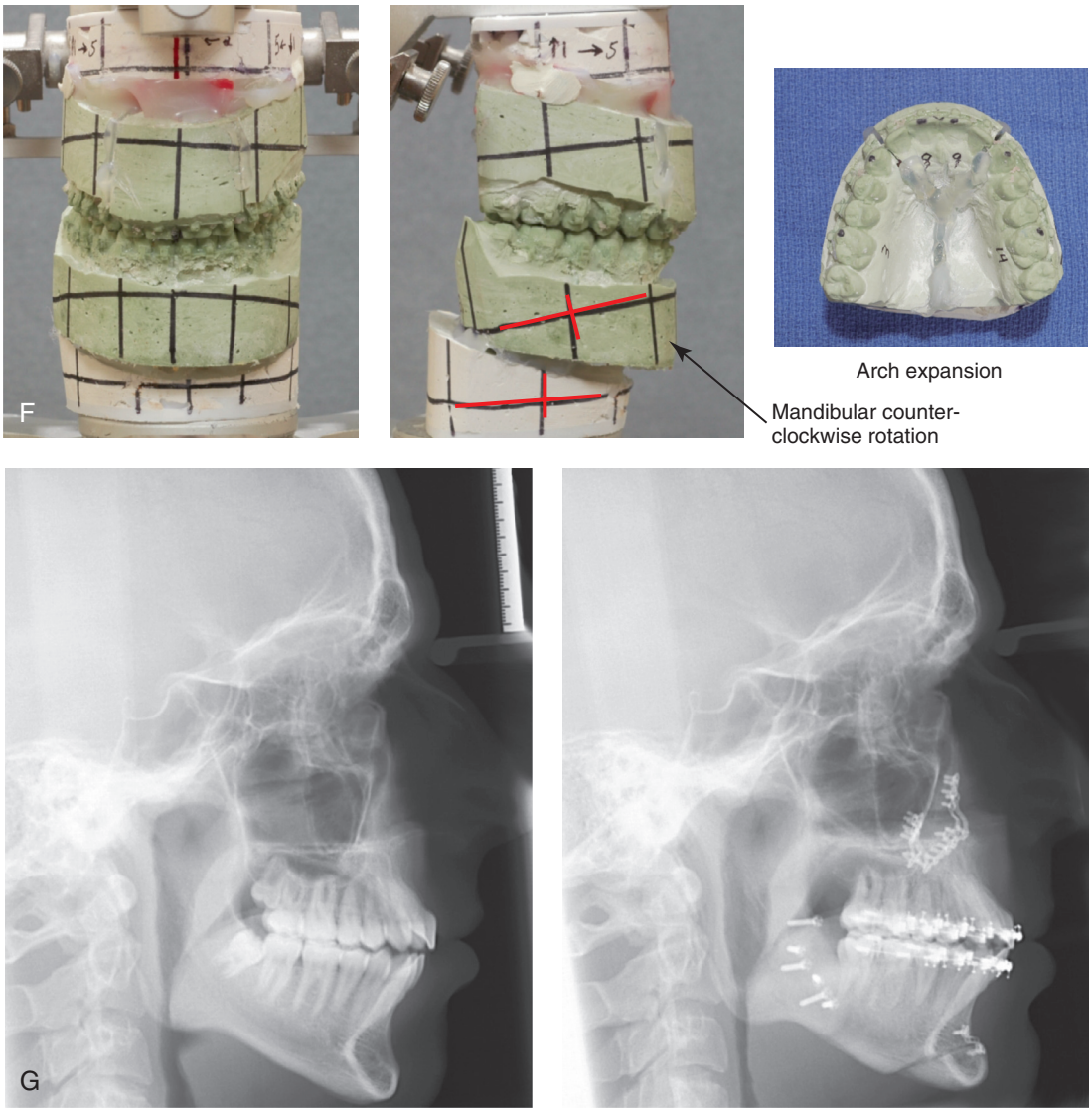
Continued

Prior to retreatment

Pre surgery

After treatment

• **Figure 24-11, cont'd E,** Occlusal views before retreatment orthodontics, before surgery and after treatment.



• **Figure 24-11, cont'd F,** Articulated dental casts that indicate analytic model planning. **G,** Lateral cephalometric radiographs before and after treatment.

For a subgroup of individuals with bimaxillary dental protrusion, some clinicians find that there are advantages to an alternative surgical approach, including intraoperative first bicuspid extractions; the removal of associated alveolar bone; and anterior maxillary and mandibular segmental osteotomies with incisor uprightening and posterior repositioning. This approach should be compared with the classic mechanics of orthodontic retraction of the anterior teeth after extractions. Theoretic advantages of the anterior segmental dentoalveolar surgical approach include the ability to simultaneously reshape the pyriform rims and the anterior nasal spine region; alter the labiomental fold; and shorten the treatment time. Interestingly, in North America, the anterior segmental approach for the correction of bimaxillary dental protrusion has never gained traction. Unfortunately, neither approach (i.e., dentoalveolar segmental surgery or orthodontics only) will decrease an elongated lower facial height or improve a retrusive pogonion (i.e., neither approach will correct an associated jaw disharmony); doing so would require an orthognathic approach.

Iino and colleagues described a single case of bimaxillary dental protrusion (not associated with jaw disharmony) that was treated with corticotomy-facilitated orthodontics and titanium mini-plate anchorage devices (TADs).[54] The authors performed orthodontic treatment after multiple corticotomies and the placement of TADs for skeletal anchorage in an adult patient who desired a shortened treatment period. The patient had an Angle Class I molar occlusion with flaring of the maxillary and mandible incisors. Other findings included moderate dental crowding in both arches and a limited overbite relationship (i.e., a 5.5-mm

overjet and a 0.5-mm overbite). The upper and lower lips were protrusive with hypermentalis activity but without lip incompetence. The treatment rendered included the placement of TADs into the buccal alveolar bone of the maxilla for skeletal anchorage; the placement of edgewise appliances on the maxillary and mandibular teeth; maxillary first premolar and mandibular second premolar extractions; corticotomies on the lingual and buccal sides in the maxillary anterior and the mandibular anterior and posterior regions; and orthodontic leveling was initiated immediately after the corticotomies. The edgewise appliances were adjusted once every 2 weeks, and the total treatment time was 1 year. Cephalometric superimposition showed no anchorage loss in the maxilla and 2 mm of loss in the mandible. Panorex radiographs showed no reduction in crest bone height and no significant apical root resorption.

Baek and colleagues completed a study to investigate the differences in skeletal, dental, and soft-tissue results found among patients with bimaxillary dental protrusion who were undergoing either four bicuspid extractions with standard orthodontic retraction or four bicuspid extractions with anterior segmental osteotomies and repositioning.[8,9]

- The authors concluded that bimaxillary dental protrusion individuals with a significant associated dentofacial disharmony will not respond favorably to either of these approaches. Preoperative findings in the jaw disharmony subgroup included significant vertical and horizontal skeletal disproportion with specific pretreatment characteristics of skeletal Class II malocclusion; a poorly developed chin; and a measurably long vertical facial height. These individuals were found to require bimaxillary orthognathic surgery and were therefore excluded from the study.
- The authors concluded that individuals with isolated bimaxillary dental protrusion and anterior open bite (i.e., without associated dentofacial disharmony) who were treated with extractions and anterior segmental set-back osteotomies achieved less favorable facial aesthetics as compared with those patients who were treated with 4 bicuspid extractions and classic orthodontic mechanics.

Lee and colleagues analyzed the outcomes of the treatment of bimaxillary dental protrusion involving three different approaches.[70] The sample consisted of Korean adult female patients who presented with this condition (N = 65). None had yet received any orthodontic treatment or surgical procedures. These individuals were not felt to have significant vertical or horizontal skeletal disproportion of the jaws. Strict criteria for study inclusion were as follows: 1) Angle Class I molar relationship; 2) no significant anterior vertical facial discrepancy; 3) no significant anteroposterior skeletal discrepancy; 4) crowding of less than 3 mm; 5) Rickets aesthetic line to lower lip distance of more than 2 mm; (6) anterior incisal angle of less than 125 degrees; 7) chin point deviation of less than 3 mm; and 8) no symptoms of TMD.

The study patients were then divided into three treatment groups:

Group I (n = 29) was treated with four bicuspid extractions and conventional orthodontics to achieve maximum anchorage with a transpalatal arch, head gear, or both.
Group II (n = 20) was treated with four bicuspid extractions, corticotomy-assisted orthodontics, TADs in the maxilla, and anterior segmental (set-back) osteotomy in the mandible.
Group III (n = 16) was treated with four bicuspid extractions and anterior segmental (set-back) osteotomies in the maxilla and the mandible.

All lateral cephalometric radiographs were taken in centric relation and in the natural head position. Pretreatment and posttreatment lateral cephalograms were traced and digitized for analysis. The authors attempted to understand which specific individuals with bimaxillary dental protrusion did better with each of the three dentoalveolar treatment approaches. The authors concluded the following:

1. If a patient with bimaxillary dental protrusion does not have significant vertical or horizontal skeletal dysplasia and has a Class I molar relationship with proclined upper and lower incisors, then four bicuspid extractions and conventional orthodontic treatment can produce a good result.
2. If a patient with bimaxillary dental protrusion has relatively normal upper incisor inclination and a Class I molar skeletal pattern but markedly proclined mandibular incisors and poor chin development, then treatment with bicuspid extractions, anterior segmental osteotomy in the mandible, and conventional orthodontics in the maxilla will be a good option. This approach will reduce the lower lip protrusion without unwanted change of the upper incisor inclination.
3. If a patient with bimaxillary dental protrusion has severe proclination of the upper incisors, then four bicuspid extractions and corticotomy-assisted orthodontic treatment with the use of TADs will achieve maximum retraction and uprighting in the shortest treatment period and will generally produce good results.

Orthognathic and Dentoalveolar Surgical Approaches

If orthognathic surgery (i.e., Le Fort I down-fracture with segmentation and sagittal split ramus osteotomies of the mandible in combination with an osteotomy of the chin)

is undertaken to treat a significant associated dentofacial disharmony or if dentoalveolar surgery (i.e., anterior segmental osteotomies) is undertaken to manage bimaxillary dentoalveolar protrusion with anterior open bite (i.e., occlusal disharmony), then the surgical option selected should be in accordance with patient-specific facial characteristics and dental findings.[11-15,26,27,29,38,39,45,49,57,63,66,74,79,81,129] In this author's opinion, the dentoalveolar surgical approach has aesthetic advantages over the standard orthodontic approach for only a very specific subgroup of patients. A key point is that neither of these dentoalveolar approaches (i.e., segmental osteotomies versus orthodontics only) will reduce the anterior lower facial height; correct significant horizontal discrepancies; or alter chin height or projection. To accomplish these objectives, orthognathic surgery is required. Details of the individual's presenting dysmorphology are important when choosing either of the limited dentoalveolar forms of treatment (i.e., segmental osteotomies versus orthodontics only) or when selecting an orthognathic (jaw) approach.[20,23,97,105,108,130]

The orthognathic surgical correction of bimaxillary dental protrusion in association with excess vertical height and horizontal disproportion of the jaws should include maxillary Le Fort I osteotomy (often with segmentation) in combination with sagittal split ramus osteotomies of the mandible (see Chapter 15). This approach provides the flexibility needed to alter the maxilla and the mandible in all three dimensions (i.e., horizontal, vertical, and transverse). The maxilla will generally require anterior vertical shortening, transverse widening, correction of the curve of Spee, minimal horizontal advancement, and a degree of clockwise rotation. The mandible will generally require horizontal advancement and counterclockwise rotation. An osteotomy of the chin with minimal horizontal advancement is generally useful to further improve profile aesthetics and ease of lip closure. The simultaneous management of lifelong nasal obstruction may include septoplasty, inferior turbinate reduction, and recontouring of the nasal floor. This orthognathic and intranasal approach is carried out to achieve improved head and neck function (i.e., breathing, swallowing, chewing, speech, and lip competence) and to enhance facial aesthetics.[98,99]

Postsurgical Orthodontic Maintenance and Detailing

Managing the details of the in-hospital and at-home convalescence during initial healing after orthognathic surgery is essential for a successful outcome (see Chapter 11). Cephalometric and Panorex radiographs as well as facial and occlusal photographs are obtained at standard postoperative intervals to document healing. If segmental maxillary osteotomies were carried out, a prefabricated splint will have been secured to the occlusal surfaces during surgery. The orthodontist sees the patient within 24 hours of splint removal and replaces the maxillary sectional arch wires with a rigid continuous one (i.e., ≈5 weeks after surgery). The teeth are ligated together to maintain the arch form. The use of a transpalatal wire or a palatal acrylic plate may also be used to stabilize the arch form for several additional months if significant transverse expansion was carried out. Close orthodontic monitoring for skeletal and dental shifts and detailing during the first 6 months after surgery is essential.

Complications, Informed Consent, and Patient Education

The orthodontist and the surgeon are frequently asked to evaluate jaw disharmonies and malpositioned teeth and then to attempt to alter nature's events. With an understanding of the natural progression of the uncorrected bimaxillary dental protrusion associated with dentofacial disharmony and knowledge of how specific interventions will affect head and neck function and facial aesthetics, predictable results are generally achieved. Complications and patient dissatisfaction will always occur in a small percentage of individuals. The clinician's ability to communicate the potential risks, complications, alternative approaches, and benefits of the recommended treatment plan to the patient and the patient's family is essential to achieve a favorable result. When the patient, his or her family, and all treating clinicians are fully informed, shared responsibility for the treatment outcome is more likely (see Chapter 16).

Malocclusion after Orthodontics and Orthognathic Surgery

There are inherent biologic challenges to the orthodontic movement of the teeth in the alveolar process and to the surgical repositioning of the jaws or jaw segments in the individual with bimaxillary dental protrusion and an associated dentofacial disharmony.[51,102,110,114,117] This includes the technical aspects of the repositioning; the initial healing requirements; and the difficulties associated with the long-term maintenance of the mechanically relocated teeth, jaws, and jaw segments. By following sound biomechanical and facial aesthetic principles, clinicians are able to carry out orthodontic treatment and surgical procedures and to effectively maintain the results in most cases. Potential intraoperative technical difficulties associated with sagittal split ramus osteotomies of the mandible and maxillary Le Fort I osteotomies can result in immediate (early) malocclusion, but this is generally preventable (see Chapter 17). Timing the surgery to limit ongoing mandibular growth from occurring afterward is usually possible (see Chapter 17). Condylar resorption after orthognathic surgery is rarely seen in the patient with bimaxillary dental protrusion (see Chapter 36). All of these factors must be taken

into account to best achieve and then maintain the desired result.

Temporomandibular Disorders: The Effects of Orthodontics and Orthognathic Surgery

It is estimated that an average of 32% of the population reports at least one symptom of TMD, whereas an average of 55% of the population demonstrates at least one clinical sign of the condition. TMD includes various symptoms and signs that involve the temporomandibular joint, the masticatory muscles, and the related structures. The symptoms and signs may include a spectrum of referred head and neck pain; joint noise (i.e., popping, clicking, or crepitus); reduced or altered mandibular movements as a result of muscle spasm or disc displacement with or without reduction; condylar head erosion; and pain during the direct palpation of either of the temporomandibular joints or the masticatory muscles. Occlusal factors (i.e., degrees of malocclusion) are often claimed to be associated with TMD. Interestingly, the correction of a skeletal anterior open bite through orthodontics and orthognathic surgery is likely to relieve symptoms and signs of TMD; however, this is not predictable for each individual patient (see Chapter 9).[1]

Conclusions

Bimaxillary dental protrusion is characterized by the proclination of the anterior maxillary and mandibular dentition with an open-bite malocclusion. The correction of the dental disharmony in most individuals is carried out with the extraction of four premolars and conventional orthodontic incisor retraction and uprighting. An alternative approach, which is preferred in only limited and specific circumstances, involves the combination of four bicuspid extractions and anterior maxillomandibular segmental osteotomies with set-back. This dentoalveolar surgical approach may shorten the treatment time, but subtle differences in the expected aesthetic results and the potential for complications must be considered.

In the patient with bimaxillary dental protrusion, facial characteristics associated with significant skeletal disharmony include the following: excessive separation of the lips at rest and effort required to bring the lips into closure; excessive incisor show when the upper lip is at rest and gingiva show with smile; and significant vertical and horizontal facial disproportion. If these skeletal findings are present, then orthognathic surgery will be required to maximize head and neck function and to enhance facial aesthetics.

References

1. Aghabeigi B, Hiranaka D, Keith DA, et al: Effect of orthognathic surgery on the temporomandibular joint in patients with anterior open bite. *Int J Adult Orthodon Orthognath Surg* 16:153, 2001.
2. Alexander TL, Hitchcock HP: Cephalometric standards for American Negro children. *Am J Orthod* 74:298–304, 1978.
3. Altemus LA: Comparative integumental relationships. *Angle Orthod* 33:217–221, 1963.
4. Angelle PL: A cephalometric study of the soft tissue changes during and after orthodontic treatment. *Trans Eur Orthod Soc* 267–280, 1973.
5. Angle EH: *Malocclusion of teeth*, ed 7, Philadelphia, 1907, SS White Dental Mfg Co.
6. Arvystas MG: Treatment of anterior skeletal open-bite deformity. *Am J Orthod* 72:147–164, 1977.
7. Bae SM, Park HS, Kyung HM, et al: Clinical application of micro-implant anchorage. *J Clin Orthod* 36:298–302, 2002.
8. Baek SH, Kim BH: Determinants of successful treatment of bimaxillary protrusion: Orthodontic treatment versus anterior segmental osteotomy. *J Craniofac Surg* 16:234, 2005.
9. Baek SH, Yang WS: A soft tissue analysis on facial esthetics of Korean young adults. *Korean J Orthod* 21:131–170, 1991.
10. Begg PR: Stone age man's dentition. *Am J Orthod* 40:298–312, 373–383, 462–475, 517–531, 1954.
11. Bell WH: Revascularization and bone healing following anterior maxillary osteotomy. *J Oral Surg* 27:249, 1969.
12. Bell WH, Condit CL: Surgical-orthodontic correction of adult bimaxillary protrusion. *J Oral Surg* 28:578–590, 1970.
13. Bell WH: Revascularization and bone regeneration following anterior mandibular osteotomy. *J Oral Surg* 28:196, 1970.
14. Bell WH: Correction of skeletal type of anterior open bite. *J Oral Surg* 29:706, 1971.
15. Bell WH, Dann JJ: Correction of dentofacial deformities by surgery in the anterior part of the jaws. *Am J Orthod* 64:162, 1973.
16. Bloom L: Perioral profile changes in orthodontic treatment. *Am J Orthod* 47:371–379, 1961.
17. Burstone CJ: Lip posture and its significance in treatment planning. *Am J Orthod* 53:262, 1967.
18. Cangialosi T: Skeletal morphologic features of anterior open bite. *Am J Orthod* 85:28–36, 1984.
19. Case CS: Principles of occlusion and dentofacial relations. *Dent Items Int* 27:489, 1905.
20. Chamberland S, Proffit WL: Short-term and long-term stability of surgically assisted rapid palatal expansion revisited. *Am J Orthod Dentofacial Orthop* 139:815–822, 2011.
21. Chung KR, Oh MY, Ko SJ: Corticotomy-assisted orthodontics. *J Clin Orthod* 35:331–339, 2001.
22. Chung KR, Kim YS, Linton JL, et al: The miniplate with tube for skeletal anchorage. *J Clin Orthod* 36:407–412, 2002.
23. Connole PW, Small EW: Combined maxillary and mandibular osteotomies: Discussion of three cases. *J Oral Surg* 29:572–578, 1971.
24. Connor AM, Moshiri F: Orthognathic surgery norms for American black patients. *Am J Orthod* 87(2):119–134, 1985.
25. Connor AM, Moshiri F: Advancement genioplasty: An important part of combination surgery in black American patients. *Am J Orthod Dentofacial Orthop* 93:92–99, 1988.
26. Converse JM, Horowitz SL: The surgical orthodontic approach to the treatment of dentofacial deformities. *Am J Orthod Dentofacial Orthop* 55:217–243, 1969.

27. Converse JM, Horowitz SL: Simianism: Surgical-orthodontic correction of bimaxillary protrusion. *J Maxillofac Surg* 1:8–12, 1973.
28. Cox NH, van der Linden FPGM: Facial harmony. *Am J Orthod* 60:175–183, 1971.
29. Daniel HT, White RP, Proffit WR: Anterior maxillary osteotomy in dental treatment. *J Am Dent Assoc* 83:338, 1971.
30. De Smit A, Dermaut L: Soft tissue profile preferences. *Am J Orthod* 86:67–73, 1984.
31. DeLoach N: Soft tissue facial profile of North American Blacks, a self assessment.
32. Dewan SK, Marjadi UK: Soft tissue changes in surgically treated cases of bimaxillary protrusion. *J Oral Maxillofac Surg* 41:116–118, 1983.
33. Diels RM, Kalra V, DeLoach N, Jr, et al: Changes in soft tissue profile of African-Americans following extraction treatment. *Angle Orthod* 65:285–292, 1995.
34. Drobocky OB, Smith RJ: Changes in facial profile during orthodontic treatment with extraction of four first premolars. *Am J Orthod Dentofacial Orthop* 95(5):220–230, 1989.
35. Drummond RA: A determination of cephalometric norms for the Negro race. *Am J Orthod* 54:670–682, 1968.
36. Dunlevy HA, White RP, Proffit WR, Turvey TA: Professional and lay judgment of facial esthetic changes following orthognathic surgery. *Int J Adult Orthodon Orthognath Surg* 2:151–158, 1974.
37. Enlow DH, Pfister C, Richardson E, Kuroda T: An analysis of black and Caucasian craniofacial patterns. *Angle Orthod* 52:279–287, 1982.
38. Epker BN, Fish LC: Surgical-orthodontic correction of open-bite deformity. *Am J Orthod* 71:278–299, 1977.
39. Epker BN: A modified anterior maxillary osteotomy. *J Maxillofac Surg* 5:35, 1977.
40. Farrow AL, Zarrinnia K, Azizi K: Bimaxillary protrusion in black Americans: An esthetic evaluation and the treatment considerations. *Am J Orthod Dentofacial Orthop* 104:240–250, 1993.
41. Fields HW, Proffit WR, Nixon WL, et al: Facial pattern differences in long-faced children and adults. *Am J Orthod* 85:217–223, 1984.
42. Finnoy JP, Wisth PJ, Boe OE: Changes in soft tissue profile during and after orthodontic treatment. *Eur J Orthod* 9:68–78, 1987.
43. Fonseca RJ, Klein WD: A cephalometric evaluation of American Negro women. *Am J Orthod* 73:152–160, 1978.
44. Foster EJ: Profile preferences among diversified groups. *Angle Orthod* 43:34–40, 1973.
45. Freihofer HPM: Reversing segmental osteotomies of the upper jaw. *Plast Reconstr Surg* 96:86, 1995.
46. Freudenthaler JW, Celar AG, Schneider B: Overbite depth and anteroposterior dysplasia indicators: The relationship between occlusal and skeletal patterns using the receiver operating characteristic (ROC) analysis. *Eur J Orthod* 22:75–83, 2000.
47. Garner LD, Butt MH: Malocclusion in black American and Nyeri Kenyans, an epidemiologic study. *Angle Orthod* 55:139–146, 1985.
48. Garner LD: Soft tissue changes concurrent with orthodontic tooth movement. *Am J Orthod* 66:367–377, 1974.
49. Generson RM, Porter JM, Zell A, Stratigos GT: Combined surgical and orthodontic management of anterior open bite using corticotomy. *J Oral Surg* 36:216–219, 1978.
50. Guinn KE: A soft tissue cephalometric analysis for the black patient. Master's thesis. Howard University School of Dentistry, 1982.
51. Hagler BL, Lupini J, Johnston LE, Jr: Long-term comparison of extraction and nonextraction alternatives in matched samples of African American patients. *Am J Orthod Dentofacial Orthop* 114:393–403, 1998.
52. Hapak FM: Cephalometric appraisal of the open-bite case. *Angle Orthod* 34:65–72, 1964.
53. Holdaway RA: A soft-tissue cephalometric analysis and its use in orthodontic treatment planning, Part I. *Am J Orthod* 84:1–28, 1983.
54. Iino S, Sakoda S, Miyawaki S: An adult bimaxillary protrusion treated with corticotomy-facilitated orthodontics and titanium miniplates. *Angle Orthod* 76:1074–1082, 2006.
55. Im DH, Kim TW, Nahm DS, et al: Current trends in orthodontic patients in Seoul National University Dental Hospital. *Korea J Orthod* 33:63–72, 2003.
56. Ito T, Nakajima M, Arita S, et al: Experimental study on the tooth movement with corticotomy procedure [Japanese]. *Nihon Kyosei Shika Gakkai Zasshi* 40:92–105, 1981.
57. Jacobs JD, Bell WH: Combined surgical and orthodontic treatment of bimaxillary protrusion. *Am J Orthod* 83:321–333, 1983.
58. Jacobson A: The craniofacial skeletal pattern of the South African Negro. *Am J Orthod* 73:681–691, 1978.
59. Kawakami M, Miyawaki S, Noguchi H, Kitita T: Screw-type implants used as anchorage for lingual orthodontic mechanics: A case of bimaxillary protrusion with second premolar extraction. *Angle Orthod* 74:715–719, 2004.
60. Kawamoto HK, Jr: In search of the harmonious face: Apollo revisited, with an examination of the indications for retrograde maxillary displacement (discussion). *Plast Reconstr Surg* 99:1272, 1997.
61. Keating PJ: Bimaxillary protrusion in the Caucasian: A cephalometric study of the morphological features. *Br J Orthod* 12:193–201, 1985.
62. Keating PJ: The treatment of bimaxillary protrusion. A cephalometric consideration of changes in the inter-incisal angle and soft tissue profile. *Br J Orthod* 13:209–220, 1986.
63. Kim JR, Son WS, Lee SG: A retrospective analysis of 20 surgically corrected bimaxillary protrusion patients. *Int J Adult Orthodon Orthognath Surg* 17:23–27, 2002.
64. Kim YH: Overbite depth indicator with particular reference to anterior open-bite. *Am J Orthod* 65:586–611, 1974.
65. Kim YH, Vietas JJ: Anteroposterior dysplasia indicator: An adjunct to cephalometric differential diagnosis. *Am J Orthod.* 73:619, 1978.
66. Kole H: Surgical operation on the alveolar ridge to correct occlusal abnormalities. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 12:515–529, 1959.
67. Kowalski CJ, Nasjleti CE, Walker GF: Differential diagnosis of adult male black and white populations. *Angle Orthod* 44:346–350, 1974.
68. Kowalski CJ, Nasjleti CE, Walker GF: Dentofacial variations within and between four groups of adult American males. *Angle Orthod* 45:146–151, 1975.
69. Kuroda S, Katayama A, Takano-Yamamoto T: Severe anterior open-bite case treated using titanium screw anchorage. *Angle Orthod* 74:558–567, 2004.
70. Lee JK, Chung KR, Baek SH: Treatment outcomes of orthodontic treatment, corticotomy-assisted orthodontic treatment, and anterior segmental osteotomy for bimaxillary dentoalveolar protrusion. *Plast Reconstr Surg* 120:1027–1036, 2007.
71. Lee JS, Park HS, Kyung HM: Micro-implant anchorage for lingual treatment of a skeletal Class II malocclusion. *J Clin Orthod* 35:643–647, 2001.
72. Lew K: Profile changes following orthodontic treatment of bimaxillary protrusion in adults with the Begg appliance. *Eur J Orthod* 11:375–381, 1989.
73. Lew KK, Loh FC, Yeo JF, et al: Profile changes following anterior subapical osteotomy in Chinese adults with bimaxillary protrusion. *Int J Adult Orthodon Orthognath Surg* 4:189– 196, 1989.
74. Lew KK: Orthodontic considerations in the treatment of bimaxillary protrusion with anterior subapical osteotomy. *Int J Adult Orthodon Orthognath Surg* 6:113–122, 1991.
75. Lin WL: A cephalometric appraisal of Chinese adults having normal occlusion and excellent facial types. *J Osaka Dent Univ* 19:1–328, 1985.
76. Martin JC: Racial ethnocentrism and judgment of beauty. *J Soc Psychol* 63:59–63, 1964.
77. Miyawaki S, Koh Y, Kim R, et al: Survey of young adults women regarding men's orofacial features. *J Clin Orthod* 34:367–370, 2000.
78. Miyawaki S, Kotama I, Inoue M, et al: Factors associated with the stability of

titanium screws placed in the posterior region for orthodontic anchorage. *Am J Orthod Dentofacial Orthop* 124:373–378, 2003.
79. Mohnac AM: Surgical correction of maxillomandibular deformities. *J Oral Surg* 23:393, 1965.
80. Moss M, Salentijn L: Differences between the functional matrices in anterior open-bite and in deep-bite. *Am J Orthod* 60:264–280, 1971.
81. Murphey PJ, Walker RV: Correction of maxillary protrusion by ostectomy and orthodontic therapy. *J Oral Surg* 21:275, 1963.
82. Nadkarni PG: Soft tissue profile changes associated with orthognathic surgery for bimaxillary protrusion. *J Oral Maxillofac Surg* 44: 851–854, 1986.
83. Nagasaka H, Sugawara J, Kawamura H, et al: A clinical evaluation on the efficacy of titanium miniplates as orthodontic anchorage. *Orthod Waves* 58:136–147, 1999.
84. Nahoum HI: Vertical proportions and the palatal plane in anterior open-bite. *Am J Orthod* 59:273–282, 1971.
85. Nahoum HI, Horowitz SL, Benedicto EA: Varieties of anterior open-bite. *Am J Orthod* 61:486–492, 1972.
86. Nahoum HI: Anterior open-bite: A cephalometric analysis and suggested treatment procedures. *Am J Orthod* 67:513–521, 1975.
87. Nakanishi H: Experimental study on artificial tooth movement with osteotomy and corticotomy. *Shikwa Gakuho* 82:219–252, 1982.
88. Neger MA: A quantitative method for the evaluation of the soft tissue facial profile. *Am J Orthod* 45:738–751, 1959.
89. Nishida M, Iizuka T, Itoi S, et al: Clinical study of corticotomy by Kole's method. *J Jpn Oral Surg Soc* 3:81–84, 1986.
90. O'Reilly MT: Integumental profile changes after surgical orthodontic correction of bimaxillary dentoalveolar protrusion in black patients. *Am J Orthod Dentofacial Orthop* 96:242–248, 1989.
91. Oliver BM: The influence of lip thickness and strain on upper lip response to incisor retraction. *Am J Orthod* 82(2):141–149, 1982.
92. Park HS, Bae SM, Kyung HM, et al: Micro-implant anchorage for treatment of skeletal Class I bialveolar protrusion. *J Clin Orthod* 35:417–422, 2001.
93. Park S, Kudlick EM, Abrahamian A: Vertical dimensional changes of the lips in the North American black patient after four first-premolar extractions. *Am J Orthod Dentofacial Orthop* 96:152–160, 1989.
94. Peck H, Peck S: A concept of facial esthetics. *Angle Orthod* 40:284–318, 1970.
95. Polk MS, Jr, Farman AG, Yancy JA, et al: Soft tissue profile: A survey of African-American preference. *Am J Orthod Dentofacial Orthop* 108:90–101, 1995.
96. Posen AL: The influence of maximum perioral and tongue force on the incisor teeth. *Angle Orthod* 42:285–309, 1972.
97. Posnick JC, Fantuzzo J, Orchin J: Deliberate operative rotation of the maxillo-mandibular complex to alter the A-point to B-point relationship for enhanced facial esthetics. *J Oral Maxillofac Surg* 64:1687–1695, 2006.
98. Posnick JC, Fantuzzo JJ, Troost T: Simultaneous intranasal procedures to improve chronic obstructive nasal breathing in patients undergoing maxillary (Le Fort I) osteotomy. *J Oral Maxillofac Surg* 65:2273–2281, 2007.
99. Posnick JC, Agnihotri N: Managing chronic nasal airway obstruction at the time of orthognathic surgery: A twofer. *J Oral Maxillofac Surg* 69:695–701, 2011.
100. Prahl-Andersen B, Boersma H, van der Linden FPGM, Moore AW: Perceptions of dentofacial morphology by laypersons, general dentists, and orthodontists. *J Am Dent Assoc* 98:209–212, 1979.
101. Proffit WR, Gamble JW, Christiansen RL: Generalized muscular weakness with severe anterior open bite. *Am J Orthod* 54:104–110, 1968.
102. Proffit WR, Knight JM: Tongue pressures and tooth stability after anterior maxillary osteotomy. *J Oral Surg* 35:798–801, 1977.
103. Proffit WR, Bell WH: Bimaxillary protrusion. In Bell WH, Proffit WR, White RP editors: *Surgical correction of dentofacial deformities*, Philadelphia, 1981, Saunders, pp 1015–1057.
104. Proffit WR, Fields HW, Moray LJ: Prevalence of malocclusion and orthodontic treatment need in the United States: estimates from the N-HANES III survey. *Int J Adult Orthodon Orthognath Surg* 13:97–106, 1998.
105. Reyneke JP, Evans WG: Surgical manipulation of the occlusal plane. *Int J Adult Orthodon Orthognath Surg* 5:99–110, 1990.
106. Ricketts RM: Aesthetic environment and the law of lip relation. *Am J Orthod* 54:272–289, 1968.
107. Rosa RA, Arvystas MG: An epidemiologic survey of malocclusions among American Negroes and American Hispanics. *Am J Orthod* 73:258–273, 1978.
108. Rosen HM: Segmental osteotomies of the maxilla. *Clin Plast Surg* 16:785–794, 1989.
109. Rosen H: Aesthetics in facial skeletal surgery. *Perspect Plast Surg* 6:1, 1993.
110. Rosenquist B: Anterior segmental maxillary osteotomy: A 24-month follow-up. *Int J Oral Maxillofac Surg* 22:210–213, 1993.
111. Rudee DA: Proportional profile changes concurrent with orthodontic therapy. *Am J Orthod* 50:421–434, 1964.
112. Russell DM, Nelson RT: Facial soft tissue profile change in the North American Black with four first pre-molar extractions. *J Md State Dent Assoc* 29(I):24–28, 1986.
113. Sato S, Kaneko M, Sasaguri K, et al: Different mechanics for orthodontic correction of Class II openbite and Class III openbite malocclusions. *Bulletin of Kanagawa Dental College* 35:65–77, 2007.
114. Scheideman GB, Kawamura H, Finn RA, Bell WH: Wound healing after anterior and posterior subapical osteotomy. *J Oral Maxillofac Surg* 43:408–416, 1985.
115. Spradley FL, Jacobs JD, Crowe DP: Assessment of the anteroposterior soft-tissue contour of the lower facial third in the ideal young adult. *Am J Orthod* 79:316–325, 1981.
116. Stoner MM, Lindquist JT: A cephalometric evaluation of 57 cases treated by Dr. CH Tweed. *Angle Orthod* 26:68–98, 1956.
117. Subtelny JD: The soft tissue profile, growth and treatment changes. *Angle Orthod* 31:105–122, 1961.
118. Subtelny JD, Sakuda M: Open bite: Diagnosis and treatment. *Am J Orthod Dentofacial Orthop* 50:337, 1964.
119. Sushner N: A photographic study of the soft tissue profile of the Negro population. *Am J Orthod* 72:373–385, 1977.
120. Swinehart EW: A clinical study of open-bite. *Am J Orthod* 28:18–34, 1942.
121. Tan TJ: Profile changes following orthodontic correction of bimaxillary protrusion with a preadjusted edgewise appliance. *Int J Adult Orthodon Orthognath Surg* 11:239–251, 1996.
122. Thomas RG: An evaluation of the soft tissue facial profile in the North American black woman. *Am J Orthod* 76:84–97, 1979.
123. Trouten JC, Enlow DH, Rakine M, et al: Morphologic factors in open-bite and deep-bite. *Angle Orthod* 53:192–211, 1983.
124. Tweed CH: *Clinical orthodontics*. St. Louis, 1966, Mosby.
125. Umemori M, Sugawara J, Mitani H, et al: Skeletal anchorage system for open-bite correction. *Am J Orthod Dentofacial Orthop* 115:166–174, 1999.
126. Wardlaw DW, Smith RJ, Hertweck DW, et al: Cephalometrics of anterior open bite: A receiver operating characteristic (ROC) analysis. *Am J Orthod Dentofacial Orthop* 101:234–243, 1992.
127. Wilcko WM, Wilcko MT, Bouquot JE, Ferguson DJ: Rapid orthodontics with alveolar reshaping: Two case reports of decrowding. *Int J Periodontics Restorative Dent* 21:9–19, 2001.
128. Worms FW, Meskin CH, Isaacson RJ: Open bite. *Am J Orthod Dentofacial Orthop* 59:589, 1971.
129. Wunderer S: Die prognathieoperation mittels frontal gestieltem maxillafragment. *Oest Z Stomat* 59:98, 1962.
130. Yokoo S, Komori T, Watatani S, et al: Indications and procedures for segmental dentoalveolar osteotomy: A review of 13 patients. *Int J Adult Orthodon Orthognath Surg* 17:254–263, 2002.

25

Dentofacial Disharmonies in Adults: Reconstruction and Rejuvenation

JEFFREY C. POSNICK, DMD, MD

- Special Medical Considerations
- Functional Assessment of Head and Neck Structures
- Temporomandibular Disorders: Effects of Orthodontics and Orthognathic Surgery
- Special Wound Healing Risk Factors
- Comprehensive Dental Rehabilitation
- Unique Orthodontic Treatment Considerations
- Previous Suboptimal Facial Surgery
- Facial Aging and Rejuvenation
- Recovery for the Adult after Orthognathic Surgery
- Conclusions

The techniques for the correction of a dentofacial deformity are basically the same for the middle-aged adult as they are for the teenager and the young adult. To achieve a favorable result in the adult orthognathic patient, it is essential to recognize age-related treatment pitfalls such as medical risk factors; progressive upper airway dysfunction; dental rehabilitation needs, including periodontal and restorative aspects; the effects of facial soft-tissue aging; and the often prolonged physiologic and psychosocial responses to surgery (Figs. 25-1 through 25-11).

Special Medical Considerations

For the adult who is more than 40 years old and who is considering orthognathic surgery, additional medical risk factors may include cardiovascular disease; the effects of obesity; smoking history and pulmonary function; the potential for deep vein thrombosis or pulmonary embolus; the effects of bisphosphonate medication on wound healing; and occult malignancy. As with teenagers and young adults, taking a thorough history, performing a review of the medical records, and completing a physical examination are essential. To clarify areas of concern in these patients, there should be emphasis placed on the need for a thorough clearance examination by a primary care physician; evaluations by other medical specialists; (e.g., cardiologist, pulmonologist); the completion of an electrocardiogram, chest radiography, and laboratory tests (e.g., chemistries, hematology, thyroid tests, coagulation tests); and other special studies (e.g., stress test, echocardiogram, sleep study, pulmonary function tests). Obtaining a hemoglobin A1C level and a serum or urine cotinine level are useful checks to address long-term diabetes effects and smoking compliance, respectively. The adult with a dentofacial deformity and an elevated body mass index is at risk for obstructive sleep apnea (OSA) and therefore more vulnerable to intraoperative and postoperative complications (see Chapter 26).

Functional Assessment of Head and Neck Structures

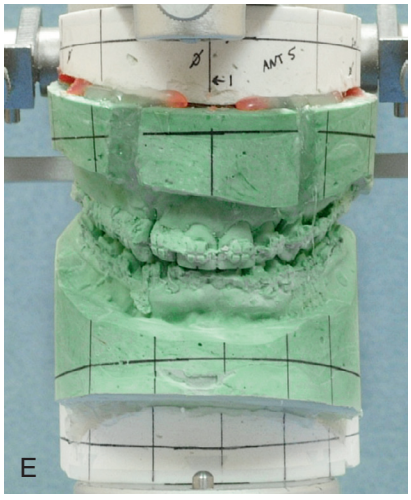
The orthognathic surgeon should evaluate the baseline head and neck functions of their patients, which include speech, swallowing, chewing, breathing, hearing, vision, cognition, and psychosocial competence (see Chapters 7 and 8). The documentation of cervical spine and temporomandibular joint (TMJ) baseline function and of any limitations in neck or mandibular range of motion is also essential (see Chapter 9). Each of these aspects may be negatively affected by the presenting maxillofacial malformations and influenced favorably or unfavorably by the treatment being contemplated. Baseline chronic obstructive nasal breathing as a result of septal deviation, enlarged inferior turbinates, or a tight nasal aperture should not be ignored (see Chapter 10).[103,104,106,107] In the adult, weight gain with adipose hypertrophy within the upper airway in combination with

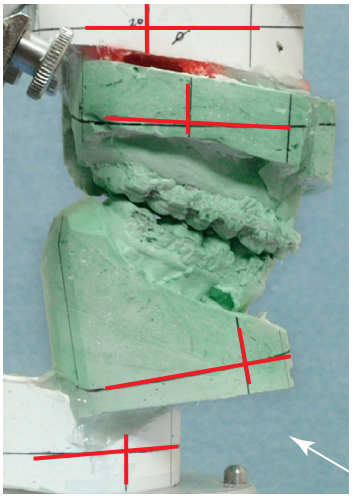
Text continued on p. 981

• **Figure 25-1** A woman in her mid 50s was referred by her general dentist to an orthodontist for the management of a longstanding Class II excess overjet malocclusion that had gradually resulted in the deterioration of the posterior dentition. The orthodontist recognized that the malocclusion resulted from a retrusive mandible and a constricted maxilla. With referral for surgical evaluation, a head and neck examination was completed. In addition to a developmental jaw deformity that involved both the maxilla and mandible, chronic obstructive nasal breathing and a sleep history consistent with obstructive sleep apnea was clarified. Suboptimal facial aging with a desire for an improved neck–chin angle was also discussed. An attended polysomnogram confirmed moderate obstructive sleep apnea. The patient agreed to proceed with orthodontics, including lower first bicuspid extractions to relieve dental compensation in combination with jaw and intranasal surgery. The objectives were to improve the airway, to enhance facial aesthetics, and to achieve improved long-term dental health. The patient's surgical procedures included maxillary Le Fort I osteotomy in segments (horizontal advancement, counterclockwise rotation, arch expansion, and the correction of the curve of Spee); bilateral sagittal split ramus osteotomies (horizontal advancement and counterclockwise rotation); osseous genioplasty (horizontal advancement); an anterior approach to the soft tissues of the neck (cervical flap elevation, neck defatting, and vertical platysma muscle plication); and septoplasty and inferior turbinate reduction. **A,** Facial views before and after reconstruction. **B,** Oblique facial views before and after reconstruction. **C,** Profile views before and after reconstruction. Note the improved A-point to B-point relationship.

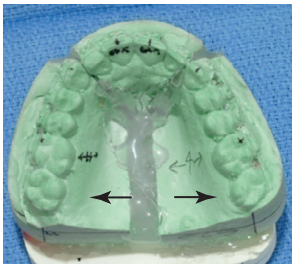
Prior to surgery and after lower bicuspid extractions

After treatment

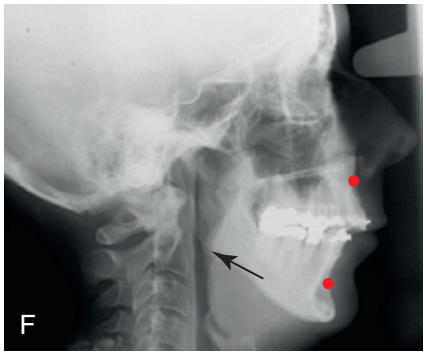


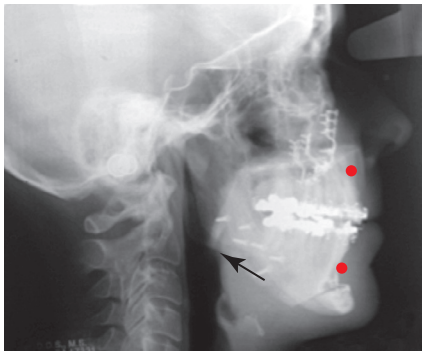


Mandibular counter clockwise rotation



Arch expansion





Pharyngeal airway expansion

• **Figure 25-1, cont'd** **D,** Occlusal views with orthodontics in progress (lower bicuspid extractions) and after treatment. **E,** Articulated dental casts that indicate analytic model planning. **F,** Lateral cephalometric radiographs before and after surgery. Note the improved posterior airway space documented.

• **Figure 25-2** A woman in her early 50s was referred by a prosthodontist for surgical evaluation. There had been a gradual deterioration of her dentition that was at least partially a result of the longstanding Class II excess overjet deep-bite malocclusion. The head and neck evaluation confirmed a retrusive mandible, which also resulted in retroglossal airway obstruction. A desire for improved profile aesthetics was also discussed. A comprehensive approach to dental rehabilitation, improvement of the upper airway, and facial rejuvenation/reconstruction was chosen. Coordinated endodontic, orthodontic, periodontic, prosthodontic, and surgical care was required. Periodontal treatment, extractions, dental implant placement, restorative temporization, and orthodontic alignment were carried out. This was followed by surgery that included bilateral sagittal split ramus osteotomies (horizontal advancement and counterclockwise rotation); osseous genioplasty (vertical lengthening) with interpositional grafting; and an anterior approach to the soft tissues of the neck (cervical flap elevation, neck defatting, and vertical platysma muscle plication). This was followed by crown lengthening and then by the final dental restorations. **A,** Frontal views before and after reconstruction/dental rehabilitation. **B,** Facial oblique views before and after reconstruction/dental rehabilitation. **C,** Profile views before and after reconstruction/dental rehabilitation.

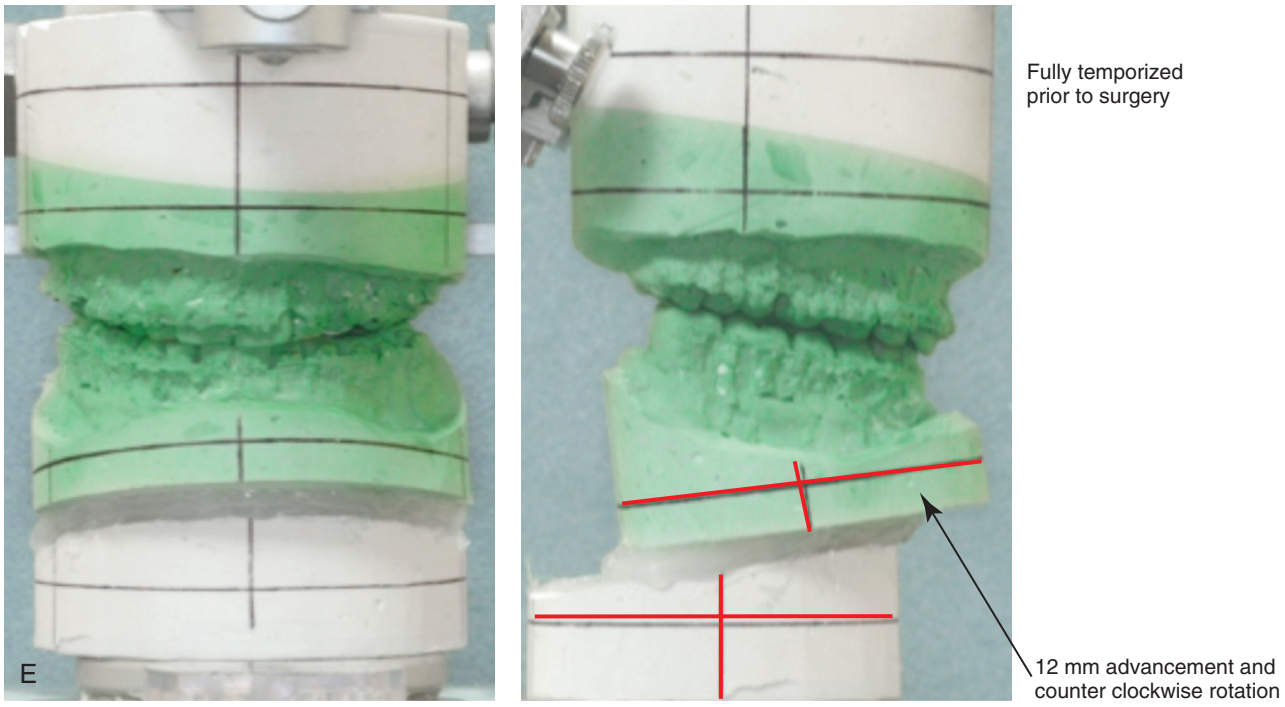
Prior to treatment

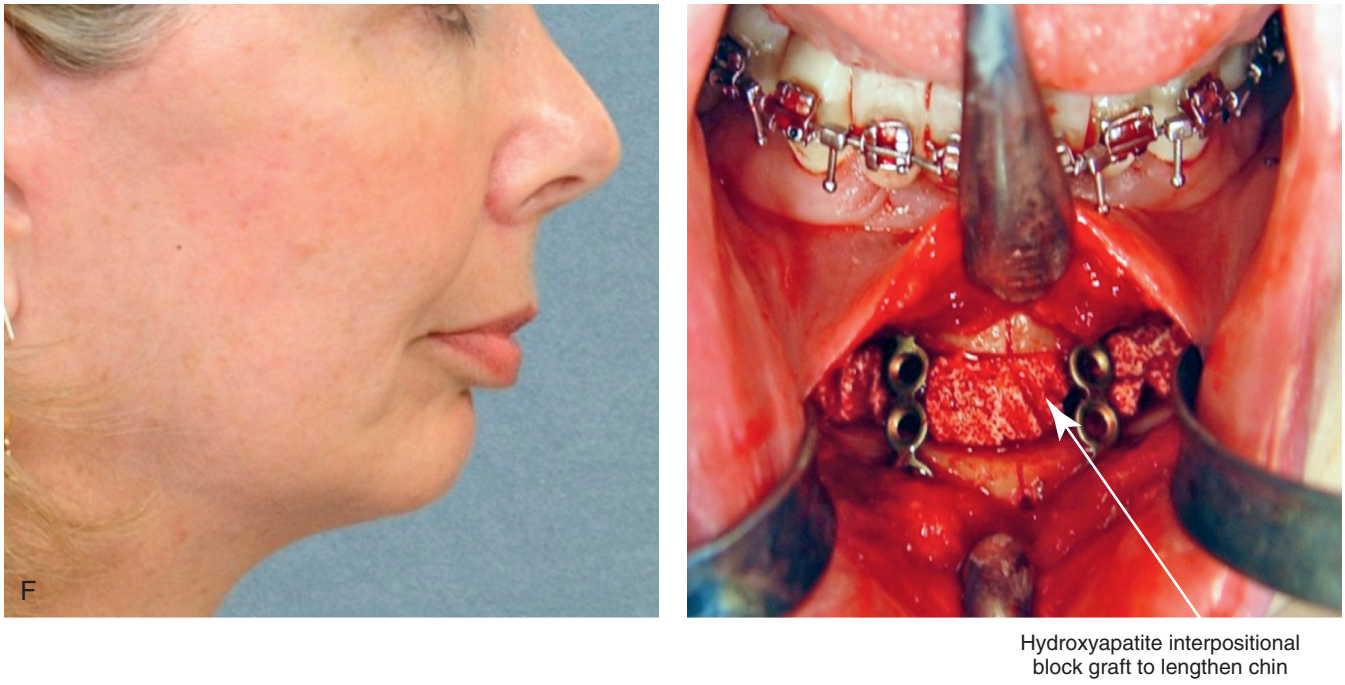
Pre surgery with dental implants in place

After surgery and dental rehabilitation

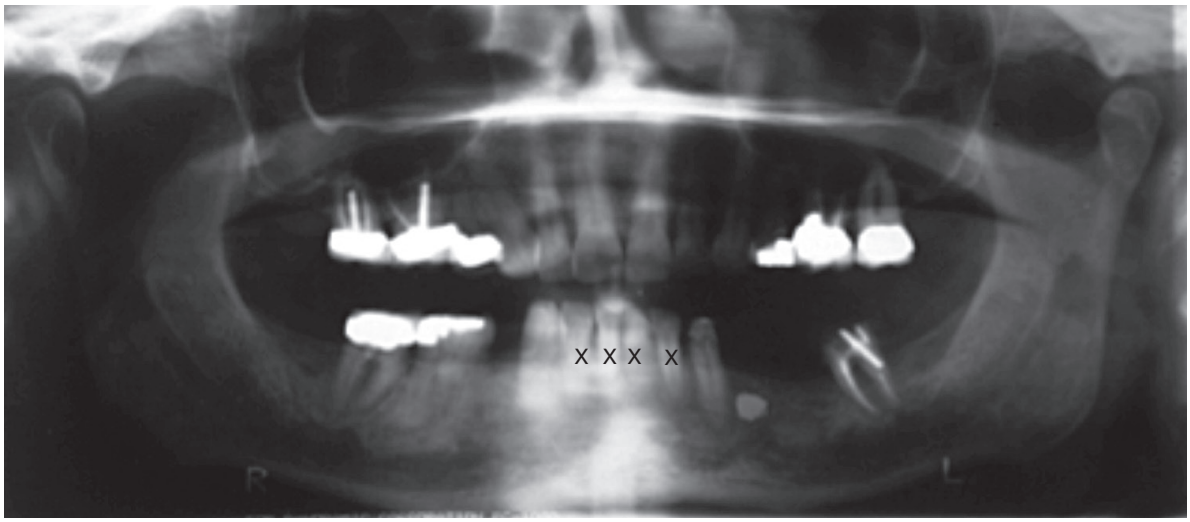
• **Figure 25-2, cont'd D,** Occlusal views at presentation, before surgery, and after reconstruction/dental rehabilitation.

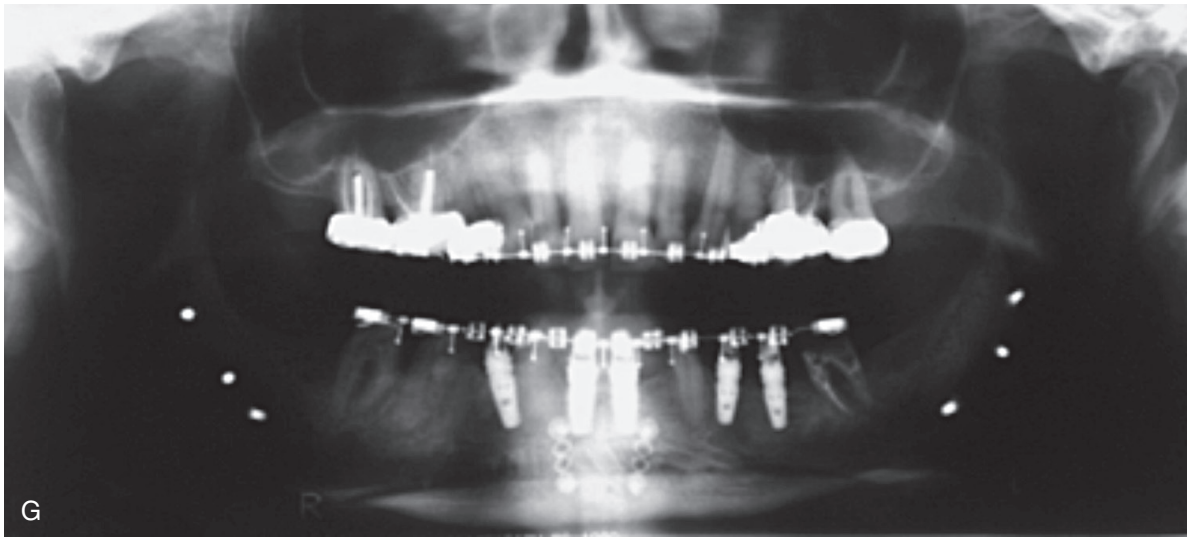
Continued

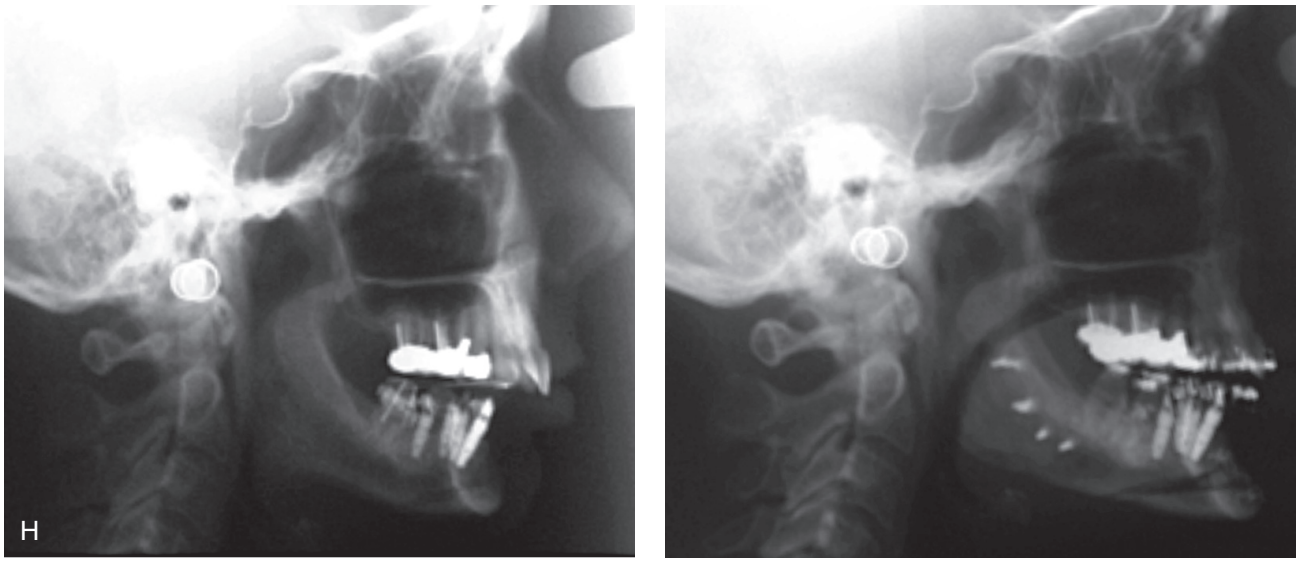




• **Figure 25-2, cont'd E,** Articulated dental casts that indicate analytic model planning. **F,** Profile view of the chin. The intraoperative view of the chin osteotomy indicates vertical lengthening with hydroxyapatite interpositional bloc graft.







• **Figure 25-2, cont'd G,** Panorex radiographs before and after lower jaw and chin surgery with the placement of dental implants. **H,** Lateral cephalometric radiographs before and after reconstruction.

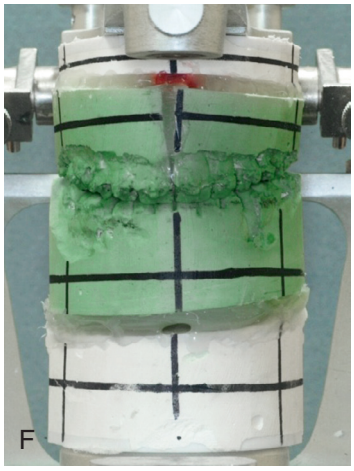
• **Figure 25-3** A woman in her late 40s with a long face growth pattern suffered deterioration of the dentition. Her periodontal risk factors included inadequate attached gingiva; dental root crowding within limited alveolar bone support, nasal airway obstruction with forced mouth breathing, and lip incompetence with drying of the exposed gingiva. An attended polysomnogram confirmed obstructive sleep apnea. Unfavorable soft-tissue envelope distortions and premature facial aging were also present. She agreed to a comprehensive surgical and dental rehabilitative approach. Periodontal treatment was followed by restorative temporization and orthodontic alignment, including four first bicuspid extractions. This was followed by surgery that included maxillary Le Fort I osteotomy in segments (vertical shortening, horizontal advancement, transverse widening, and limited clockwise rotation); bilateral sagittal split ramus osteotomies (horizontal advancement and limited counterclockwise rotation); osseous genioplasty (vertical shortening and horizontal advancement); an anterior approach to the soft tissues of the neck (cervical flap elevation, neck defatting, and vertical platysma muscle plication); and septoplasty, inferior turbinate reduction, and nasal floor recontouring. **A,** Frontal views in repose before and after treatment. **B,** Frontal views with smile before and after treatment.

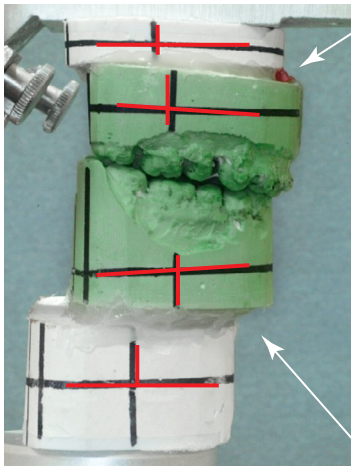
• **Figure 25-3, cont'd C,** Oblique facial views before and after treatment. **D,** Profile views before and after treatment. Note the pretreatment tendency for the patient to hyperextend her neck to achieve an improved airway. After successful surgery and with improved daytime breathing, her neck rests in a natural (neutral) position.

Continued

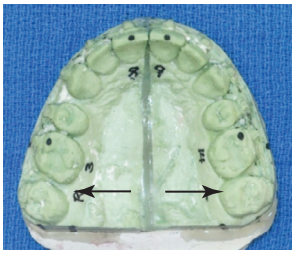
Prior to treatment

After treatment with 4 bicuspid extractions, surgery and dental rehabilitation



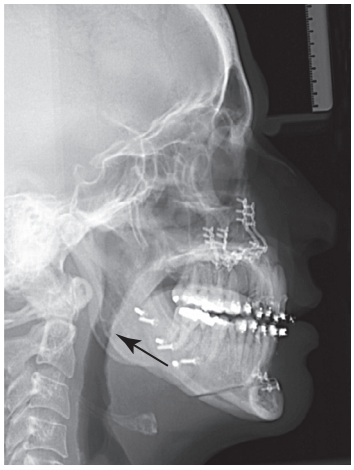


Maxillary clockwise rotation



Arch expansion

Mandibular counter clockwise rotation



Pharyngeal airway expansion

After 4 bicuspid extractions

• **Figure 25-3, cont'd** **E,** Occlusal views before treatment and after reconstruction/dental rehabilitation. **F,** Articulated dental casts that indicate analytic model planning. **G,** Lateral cephalometric radiographs before and after treatment. Note the improved posterior airway space.

©Elsevier Inc.
Orthognathic Surgery; J.C. Posnick

• **Figure 25-4** A woman in her mid 50s was experiencing significant dental deterioration as a result of occlusal trauma. An orthodontic consultation followed by surgical evaluation was carried out. The longstanding developmental jaw deformity was characterized by a short face growth pattern in combination with maxillary transverse constriction. There was a Class II deep-bite excess overjet malocclusion. The patient's head and neck evaluation confirmed a lifelong history of obstructed nasal breathing, restless sleeping at night, and a degree of daytime fatigue that was consistent with obstructive sleep apnea. Unfavorable facial aging that is typical of maxillomandibular deficiency was recognized. A comprehensive surgical and dental rehabilitative approach was selected. The objectives included dental rehabilitation, the enhancement of facial aesthetics, and an improved airway. The patient's procedures included maxillary Le Fort I osteotomy in segments (horizontal advancement, vertical lengthening, and arch expansion); bilateral sagittal split ramus osteotomies (horizontal advancement); osseous genioplasty (vertical lengthening and horizontal advancement); an anterior approach to the soft tissues of the neck (cervical flap elevation, neck defatting, and vertical platysma muscle plication); and septoplasty and inferior turbinate reduction. **A,** Frontal views in repose before and after reconstruction/dental rehabilitation. **B,** Frontal views with smile before and after reconstruction/dental rehabilitation.

Continued

• **Figure 25-4, cont'd** **C,** Oblique facial views before and after reconstruction/dental rehabilitation. **D,** Profile views before and after reconstruction/dental rehabilitation.

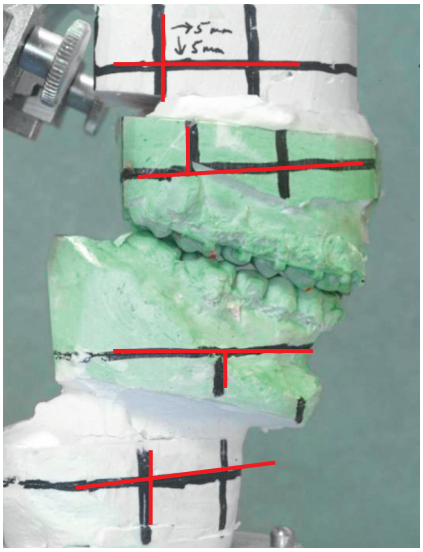
Prior to treatment

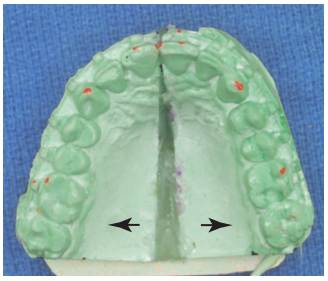
Pre surgery

After surgery and dental rehabilitation

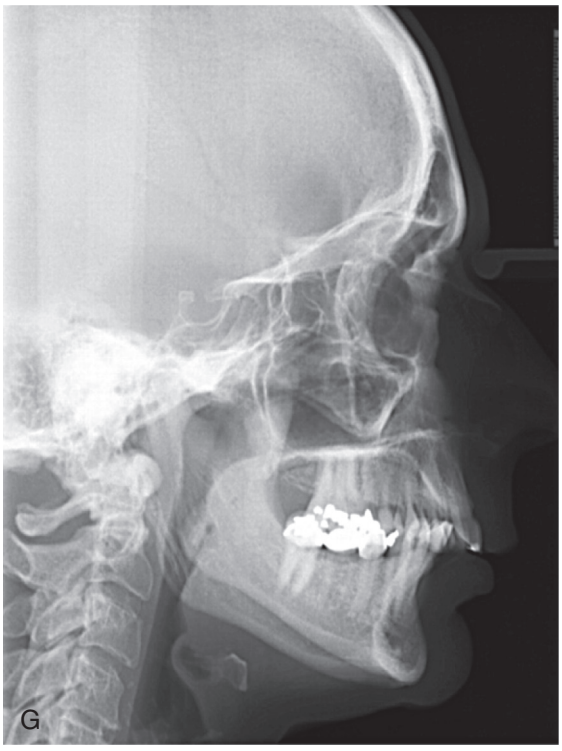
• **Figure 25-4, cont'd E,** Occlusal views before treatment, with orthodontics in progress, and after reconstruction/dental rehabilitation.

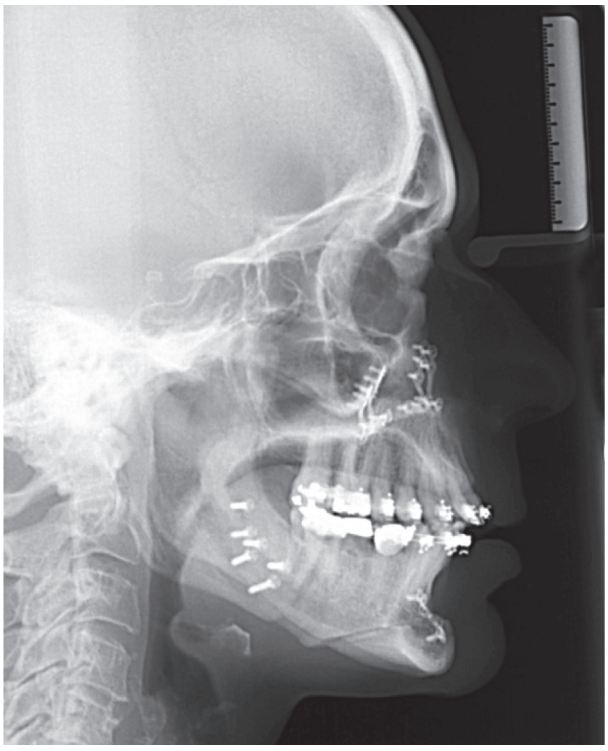
Continued





Arch expansion





• **Figure 25-4, cont'd** **F,** Articulated dental casts indicate analytic model planning. **G,** Lateral cephalometric radiographs before and after reconstruction.

• **Figure 25-5** A woman in her early 40s wished to correct her malocclusion and improve her smile. She was referred to an orthodontist who then requested surgical evaluation. She was found to have a long face growth pattern and a lifelong history of obstructed nasal breathing. There was dental crowding in each arch, mild gingival recession associated with the posterior dentition, and a Class II anterior open-bite malocclusion. The patient was aware of unfavorable facial aesthetics, which included a retrusive chin and an obtuse neck–chin angle. She agreed to a comprehensive surgical and dental rehabilitative approach. She underwent extraction of four bicuspids, orthodontic alignment, and then surgery. The patient's procedures included Le Fort I osteotomy (vertical intrusion, horizontal advancement, and counterclockwise rotation); bilateral sagittal split ramus osteotomies (horizontal advancement and counterclockwise rotation); osseous genioplasty (vertical shortening and horizontal advancement); an anterior approach to the neck (cervical flap elevation, neck defatting, and vertical platysma muscle plication); and septoplasty, inferior turbinate reduction, and nasal floor recontouring. **A,** Frontal views in repose before and after reconstruction. **B,** Frontal views with smile before and after reconstruction.

Continued

• **Figure 25-5, cont'd C,** Oblique facial views before and after reconstruction. **D,** Profile views before and after reconstruction.

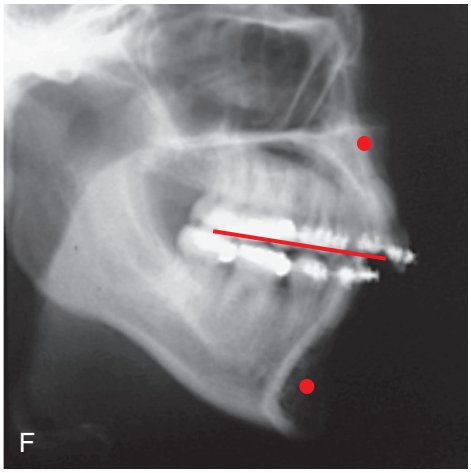
Prior to treatment

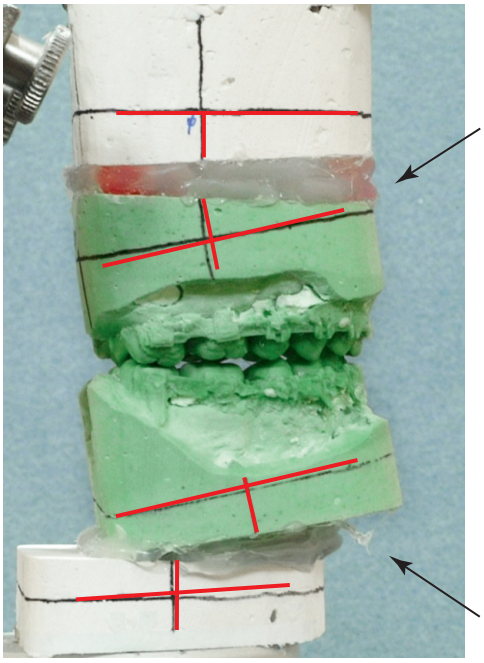
Pre surgery with 4 bicuspid extractions

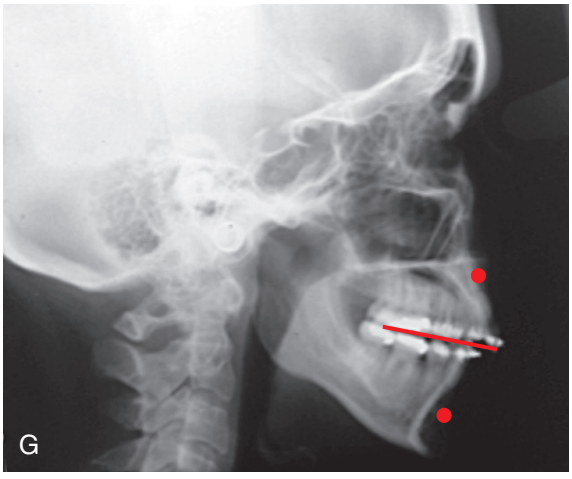
After treatment

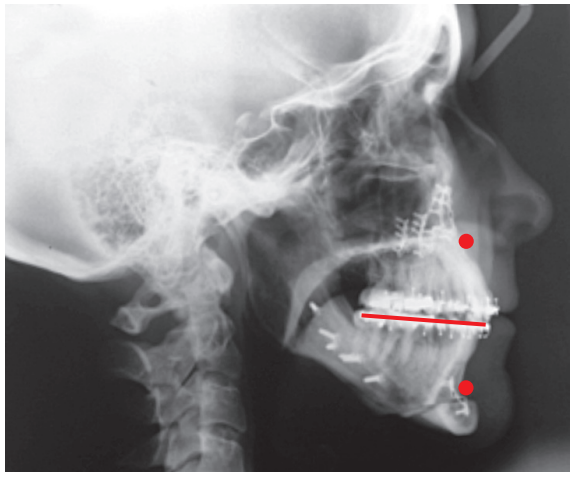
• **Figure 25-5, cont'd E,** Occlusal views before treatment, with extractions and orthodontic decompensation complete, and after treatment.

Continued









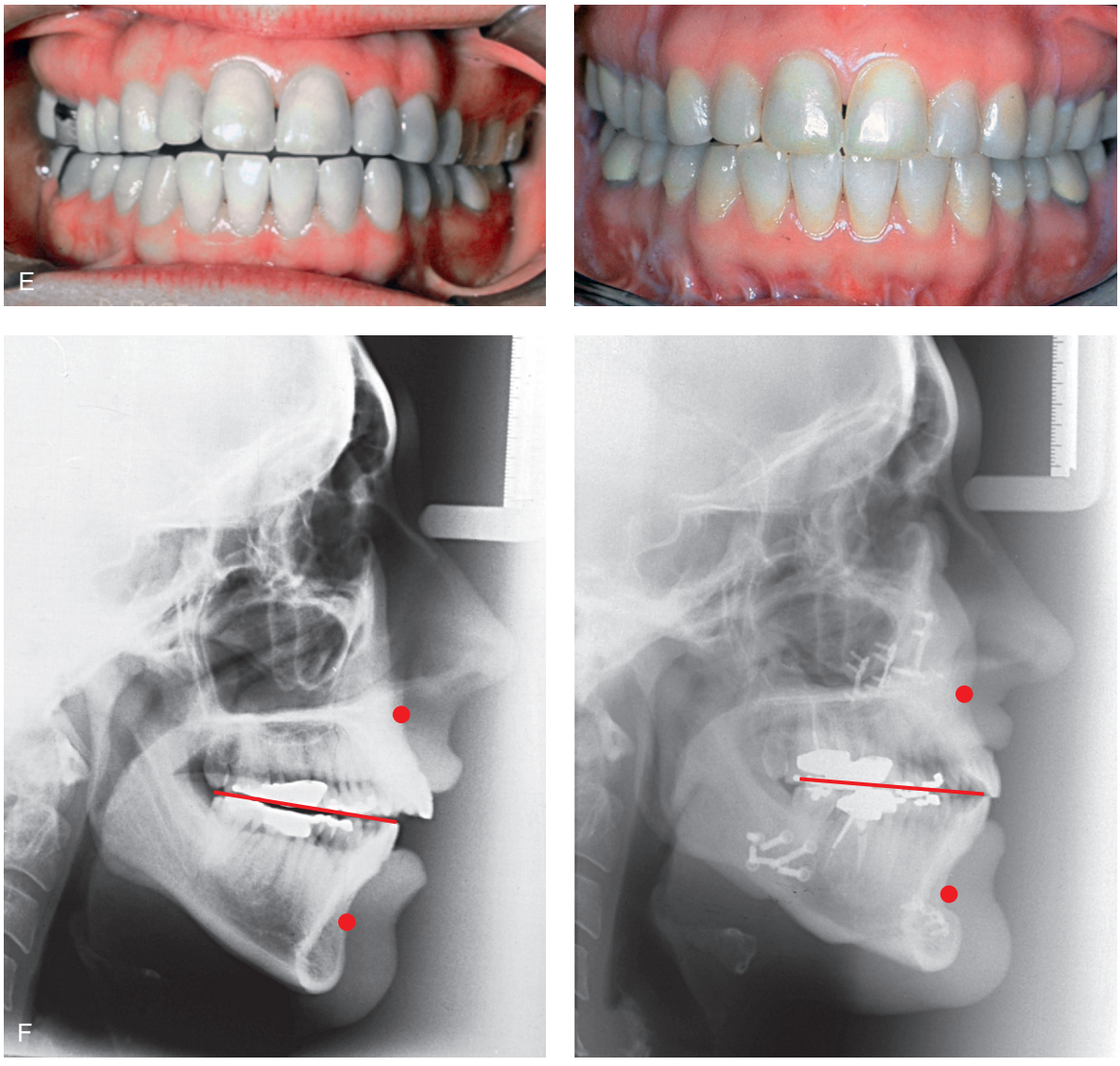
• **Figure 25-5, cont'd F,** Articulated dental casts that indicate analytic model planning. **G,** Lateral cephalometric radiographs before and after surgery.

• **Figure 25-6** A woman in her early 50s arrived for surgical evaluation and requested facial rejuvenation. Her head and neck examination confirmed a lifelong history of obstructed nasal breathing and a long face growth pattern with gummy smile and lip incompetence. Unfavorable soft-tissue envelope facial aging added to the original dentofacial deformity. Early during her life, posterior occlusal equilibration was carried out to close down the anterior open bite; this resulted in dental deterioration that required crowns in the posterior dentition and the need for root canal therapy in many of the teeth. At this time, the patient agreed to a combined surgical and dental rehabilitation approach. After orthodontic preparation, the patient's surgical procedures included maxillary Le Fort I osteotomy (vertical intrusion, horizontal advancement, and counterclockwise rotation); bilateral sagittal split ramus osteotomies (horizontal advancement and counterclockwise rotation); osseous genioplasty (vertical reduction and horizontal advancement); and septoplasty, inferior turbinate reduction, and nasal recontouring. **A,** Frontal views in repose before and after treatment. **B,** Frontal views with smile before and after treatment.

Continued

©Elsevier Inc.
Orthognathic Surgery; J.C. Posnick

• **Figure 25-6, cont'd** **C,** Oblique facial views before and after reconstruction. **D,** Profile views before and after reconstruction.



• **Figure 25-6, cont'd** **E,** Occlusal views before and after treatment. **F,** Lateral cephalometric radiographs before and after surgery.

• **Figure 25-7** A woman in her mid 50s was seen by a prosthodontist for the management of a deteriorating posterior dentition. She was confirmed to have secondary dental trauma as a result of a longstanding malocclusion. She was referred to an orthodontist and then for surgical assessment. Her head and neck evaluation confirmed a short face growth pattern with a Class II excess overjet deep-bite constricted maxillary arch malocclusion. There was a lifelong history of obstructed nasal breathing and the suggestion of obstructive sleep apnea, which was confirmed with an attended polysomnogram. Facial aesthetic concerns included the downturned corners of the mouth, the deep perioral creases, the early jowl formation, the weak profile, the obtuse neck–chin angle, and the loose skin of the neck. The patient agreed to a comprehensive surgical and dental rehabilitative approach. Periodontal evaluation, initial restorations, and orthodontic decompensation preceded surgery. The patient's surgical procedures included maxillary Le Fort I osteotomy (horizontal advancement, counterclockwise rotation, and vertical adjustment); bilateral sagittal split osteotomies of the mandible (horizontal advancement and counterclockwise rotation); osseous genioplasty (horizontal advancement); an anterior approach to the neck (cervical flap elevation, neck defatting, and vertical platysma muscle plication); and septoplasty, inferior turbinate reduction, and nasal recontouring. **A,** Frontal views in repose before and after treatment. **B,** Frontal views with smile before and after treatment.

©Elsevier Inc.
Orthognathic Surgery; J.C. Posnick

• **Figure 25-7, cont'd** **C,** Oblique facial views before and after treatment. **D,** Profile views before and after treatment.

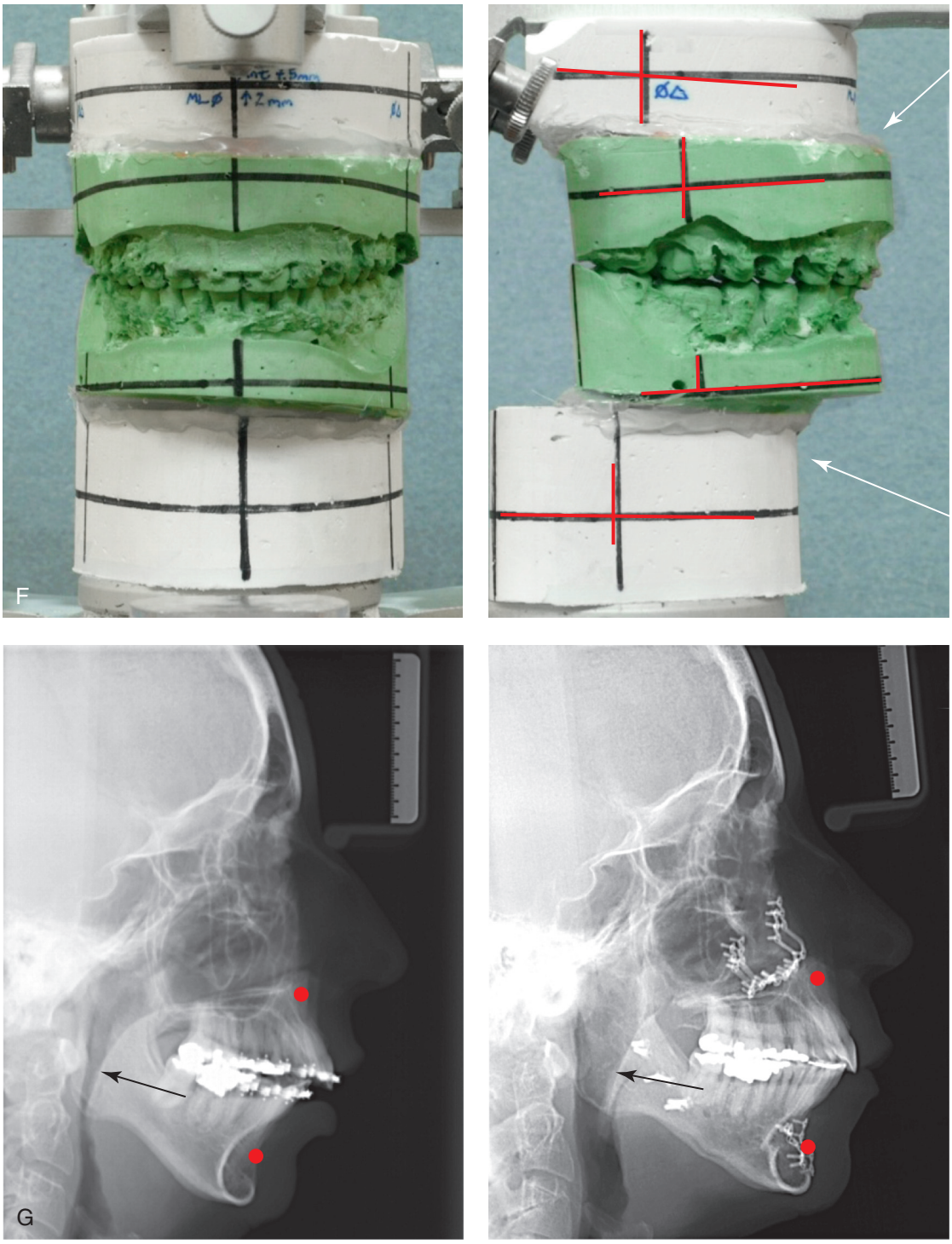
Continued

Prior to treatment

Pre surgery

After treatment

• **Figure 25-7, cont'd E,** Occlusal views before treatment, with orthodontics in progress, and after treatment.



• **Figure 25-7, cont'd** **F,** Articulated dental casts that indicate analytic model planning. **G,** Lateral cephalometric radiographs before and after treatment. Note the improved posterior airway space.

• **Figure 25-8** A woman in her mid 30s was referred by an orthodontist for surgical evaluation. She had a lifelong history of obstructed nasal breathing and a long face growth pattern. Throughout her middle childhood and teenage years, she had undergone attempted growth modification followed by orthodontic camouflage that included four bicuspid extractions in an effort to neutralize the occlusion. This resulted in periodontal deterioration and dental relapse with residual malocclusion. She now agreed to a comprehensive surgical and dental rehabilitative approach. Periodontal treatment was followed by orthodontic decompensation. The patient's surgery included maxillary Le Fort I osteotomy in segments (horizontal advancement, vertical shortening, counterclockwise rotation, arch expansion, and the correction of the curve of Spee); bilateral sagittal split ramus osteotomies in segments (horizontal advancement and counterclockwise rotation); osseous genioplasty (vertical reduction and horizontal advancement); and septoplasty, inferior turbinate reduction, and nasal recontouring. **A,** Frontal views in repose before and after reconstruction/dental rehabilitation. **B,** Frontal views with smile before and after reconstruction/dental rehabilitation.

• **Figure 25-8, cont'd** **C,** Oblique facial views before and after reconstruction/dental rehabilitation. **D,** Profile views before and after reconstruction/dental rehabilitation.

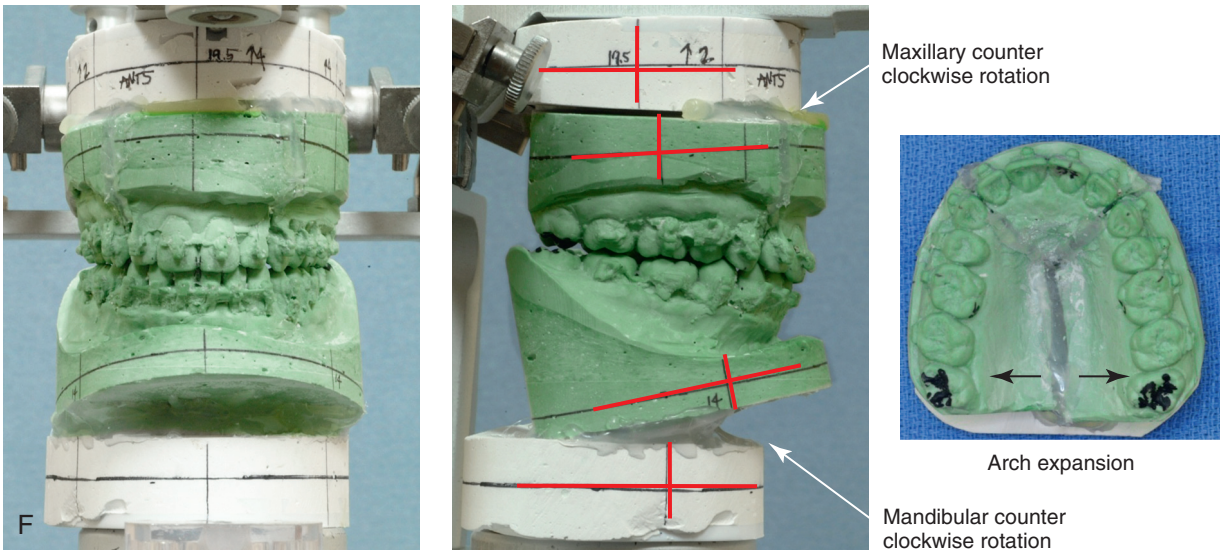
Continued

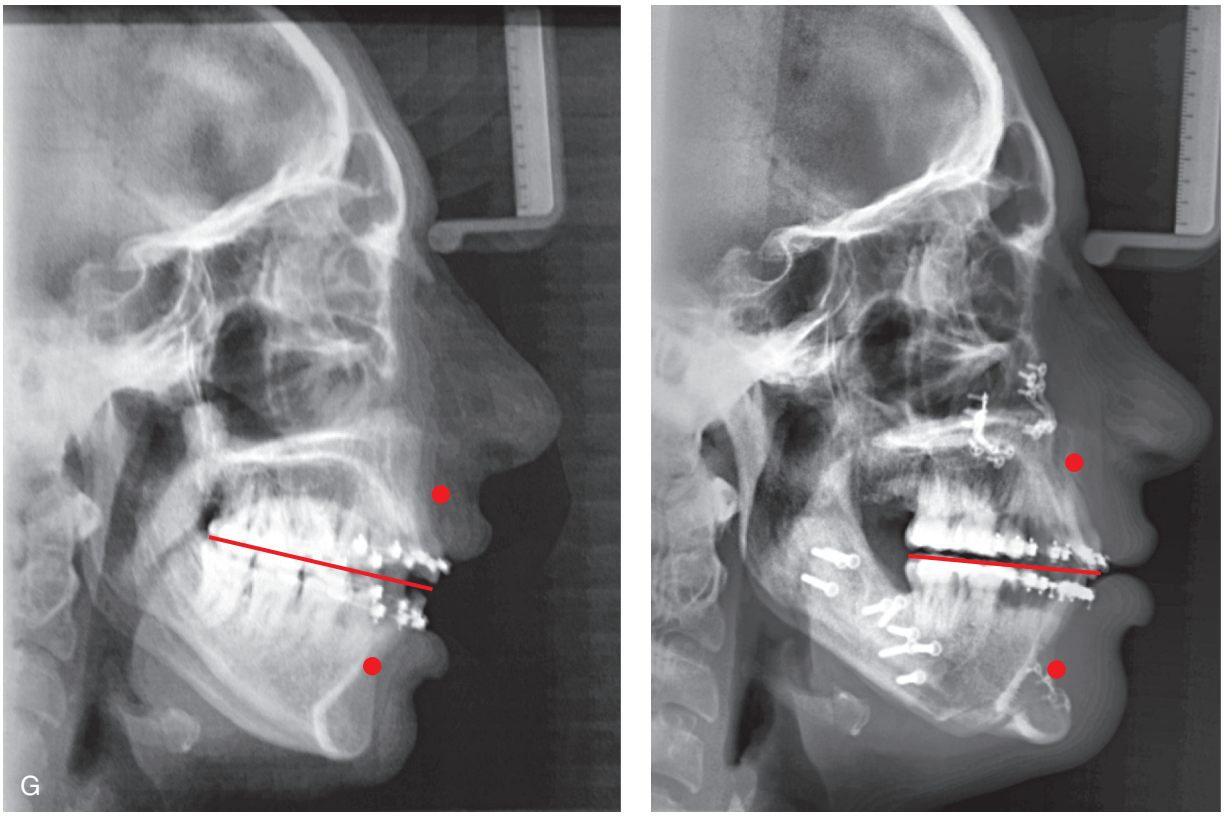
Prior to retreatment with 4 bicuspid extractions as a teenager

Pre surgery

After treatment

• **Figure 25-8, cont'd E,** Occlusal views before retreatment, with orthodontics in progress, and after treatment.



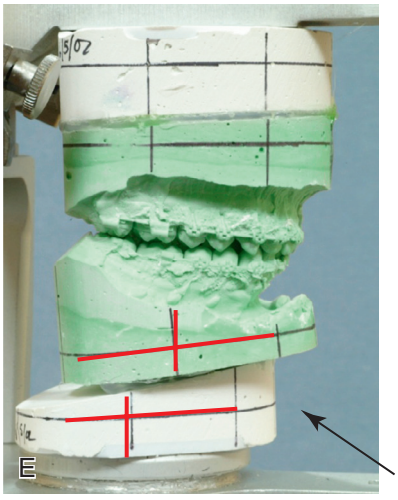


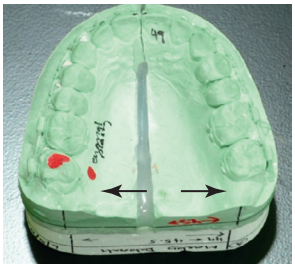
• **Figure 25-8, cont'd** **F,** Articulated dental casts that indicate analytic model planning. **G,** Lateral cephalometric radiographs before and after treatment.

• **Figure 25-9** A woman in her mid 40s requested the correction of her malocclusion and an improved smile. She was referred by a restorative dentist to an orthodontist and then for surgical evaluation. The developmental jaw deformity was characterized by mandibular deficiency in combination with maxillary arch width constriction. A lifelong history of obstructed nasal breathing with a desire for an improved airway was also confirmed. A combined orthodontic and surgical approach was chosen. After orthodontic decompensation, the patient's surgery included maxillary Le Fort I osteotomy in segments (transverse expansion); bilateral sagittal split ramus osteotomies (horizontal advancement and counterclockwise rotation); osseous genioplasty (horizontal advancement); and septoplasty and inferior turbinate reduction. **A,** Frontal views in repose before and after reconstruction. **B,** Facial views with smile before and after reconstruction. **C,** Oblique facial views before and after reconstruction.

Prior to treatment

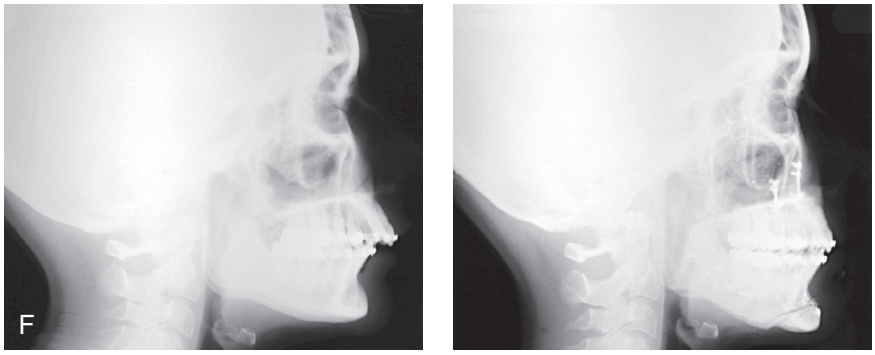
After treatment





Arch expansion

Mandibular counter clockwise rotation



• **Figure 25-9, cont'd** **D,** Occlusal views before treatment and after reconstruction. **E,** Articulated dental casts that indicate analytic model planning. **F,** Lateral cephalometric radiographs before and after reconstruction.

• **Figure 25-10** A 70-year-old woman arrived for surgical evaluation. She desired a stronger chin, less "loose" neck skin, and an improved smile. She initially requested a face lift and a chin implant. She was found to have a retrusive mandible and dental deterioration made worse by dental crowding and a deep-bite negative overjet malocclusion. She underwent orthodontics that included four bicuspid extractions and then surgery that included sagittal split ramus osteotomies (horizontal advancement) and osseous genioplasty (horizontal advancement). **A,** Frontal views with smile before and after treatment. **B,** Oblique facial views before and after treatment. **C,** Profile views before and after treatment.

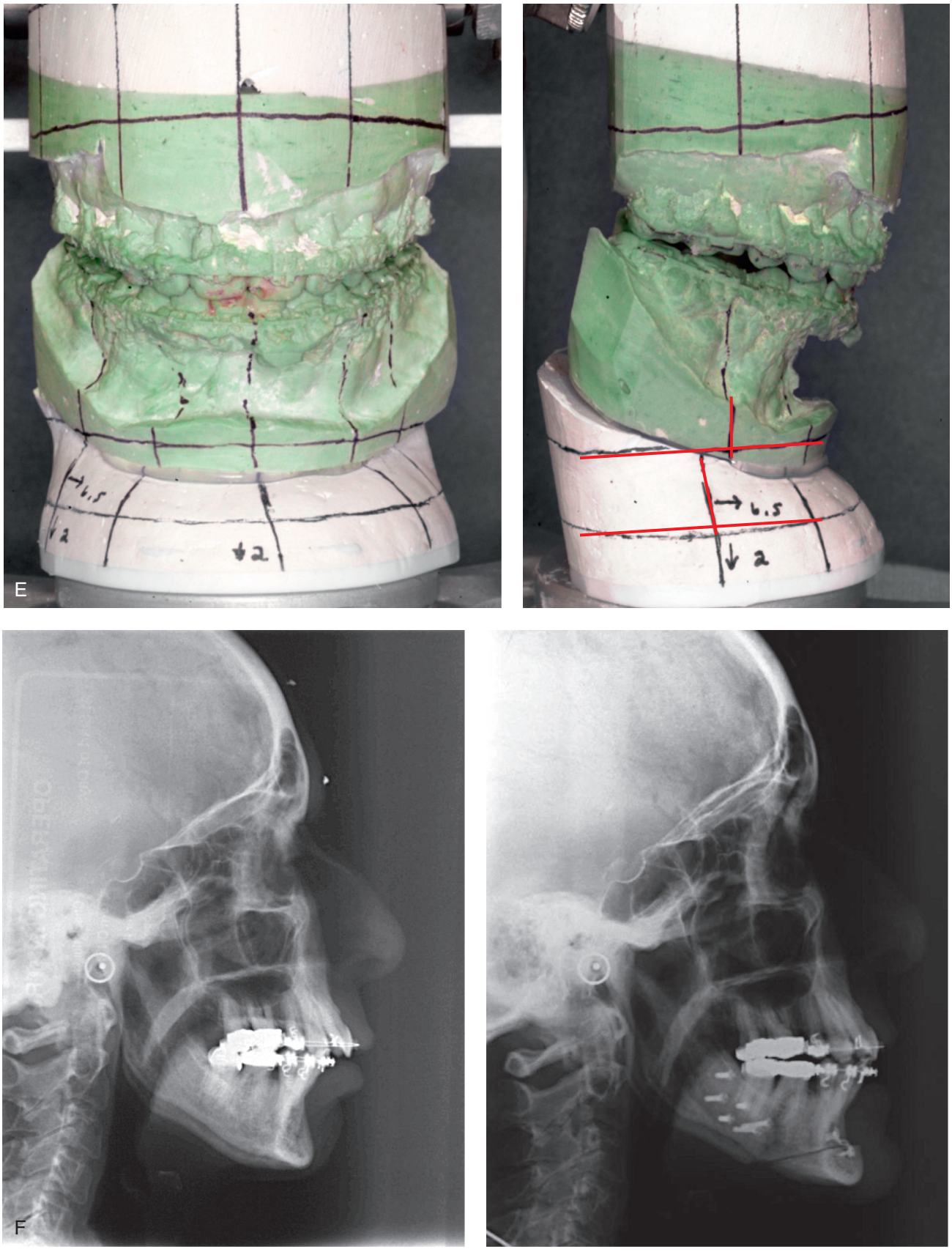
Prior to treatment

Pre surgery with 4 bicuspid extractions

After treatment

• **Figure 25-10, cont'd D,** Occlusal views before treatment, with orthodontics in progress (including four bicuspid extractions), and after treatment.

Continued



• **Figure 25-10, cont'd E,** Articulated dental casts that indicate analytic model planning. **F,** Lateral cephalometric radiographs before and after surgery.

• **Figure 25-11** An Asian woman in her mid 30s had a developmental jaw deformity that was characterized by maxillary deficiency and relative mandibular excess. In the childhood years, she had been treated with growth modification followed by four bicuspid extractions and orthodontic camouflage mechanics. Periodontal deterioration resulted, and malocclusion remained. She also had a lifelong history of obstructed nasal breathing. She now agreed to a comprehensive surgical and dental rehabilitative approach. Periodontal treatment was followed by orthodontic decompensation. The patient's surgery included maxillary Le Fort I osteotomy in segments (horizontal advancement, vertical intrusion, counterclockwise rotation and transverse widening); bilateral sagittal split ramus osteotomies (horizontal advancement); osseous genioplasty (vertical shortening and horizontal advancement); and septoplasty, inferior turbinate reduction, and nasal floor recontouring. **A,** Frontal views with smile before and after treatment. **B,** Oblique facial views before and after treatment. **C,** Profile views before and after treatment.

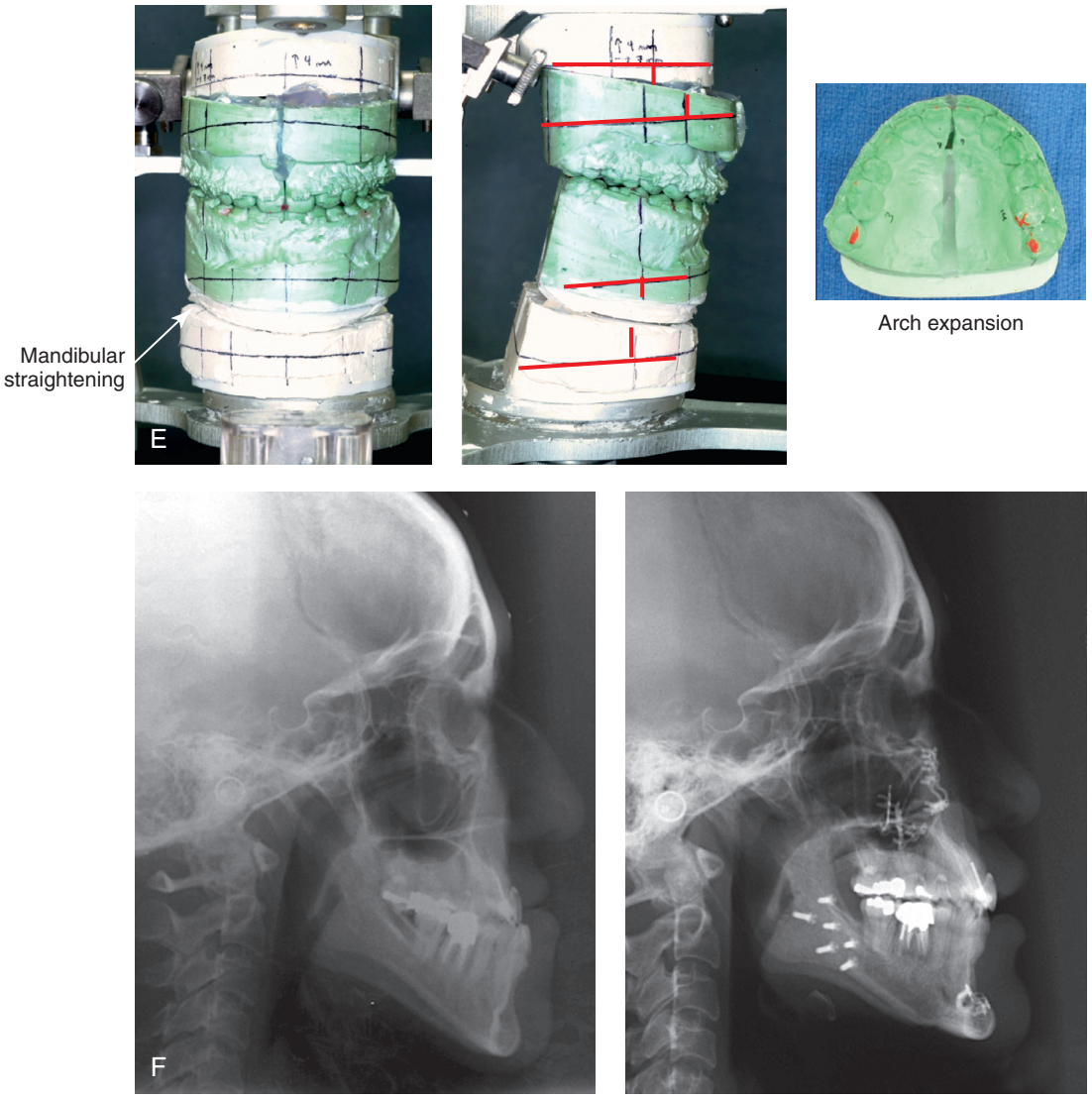
Continued

Prior to retreatment with 4 bicuspid extractions as a teenager

Pre surgery

After treatment

• **Figure 25-11, cont'd D,** Occlusal views before retreatment, with orthodontics in progress, and after reconstruction/dental rehabilitation.



• **Figure 25-11, cont'd E,** Articulated dental casts that indicate analytic model planning. **F,** Lateral cephalometric radiographs before and after treatment.

hypotonia of the pharyngeal muscles in the presence of an uncorrected dentofacial deformity is likely to result in OSA. A formal attended polysomnogram, a complete upper airway evaluation, and a consultation with a sleep specialist are frequently indicated (see Chapter 26).

Temporomandibular Disorders: Effects of Orthodontics and Orthognathic Surgery

It is estimated that an average of 32% of the population report at least one symptom of temporomandibular disorder (TMD) and that an average of 55% demonstrate at least one clinical sign of such a condition.[117] TMDs include various symptoms and signs of the TMJs, the masticatory muscles, and the related structures. The symptoms and signs may include a spectrum of referred head and neck discomforts; joint noise (e.g., popping, clicking, crepitus); reduced or altered mandibular movements as a result of muscle spasm or disc displacement with or without reduction; condylar head erosion; and pain on direct palpation of either TMJ or of the masticatory muscles.

The effects of orthodontics and orthognathic surgery on TMJ function for each specific adult patient cannot be fully predicted. Many clinical studies report improvement in TMJ symptoms and signs in a majority of individuals after successful orthodontics and orthognathic surgery, although some patients will deteriorate after the procedures. The favorable TMJ effects of orthognathic surgery often observed in patients with preoperative TMD may be the result of the improved occlusion, the restoration of normal facial height and jaw projection, or the reduced emotional stress. Adults with longstanding TMD should be cautioned that their pretreatment findings may not respond to occlusal correction and jaw-straightening surgery. Baseline TMD

problems in the patient with a dentofacial may primarily be masticatory muscle disorders (i.e., myofascial conditions) or, less commonly, the result of joint pathology (i.e., internal derangement). These two problems may coexist, and the distinction in some individuals may be difficult. It is unlikely that the correction of an existing dentofacial deformity will correct either internal joint problems or other non-muscular sources of pain. Myofascial pain develops when muscles are fatigued, because they then tend to go into spasm. Myofascial pain generally occurs after clenching or grinding the teeth for many hours each day, presumably as a response to stress. It is also true that some types of occlusal discrepancies predispose the individual who clenches or grinds his or her teeth to TMD. A compelling argument against malocclusion as the primary cause of TMD is the observation that TMD is not more prevalent among individuals with malocclusion as compared with those with a more normal occlusion. Interestingly, for those individuals with myofascial pain, the orthodontic mechanics required as part of the orthognathic correction are often helpful. This is because orthodontic treatment makes the teeth sensitive, which tends to limit grinding and clenching. Therefore, when the parafunctional (i.e. clenching, grinding) activity stops, the myofascial pain is often diminished. The changing occlusal relationships during the orthodontic phase of treatment may also contribute to a break in the cycle that contributed to the initial muscle fatigue and pain. The cycle of clenching and grinding is also broken during the healing phase that takes place just after orthognathic surgery. Unfortunately, it is also recognized that, at some point after successful orthodontics and orthognathic surgery, the individual with a long history of myofascial TMD symptoms may return to the clenching and grinding parafunctional habits. The use of an intra-occlusal splint in this situation will often be the best way to break the cycle and keep symptoms at bay. For all of these reasons, the individual's rationale for the correction of the jaw deformity and the associated malocclusion should not be solely based on a desire for the permanent relief of TMJ symptoms and signs (see Chapters 7 and 9).

Special Wound Healing Risk Factors

Special wound healing risk factors may include previous radiation therapy, diabetes mellitus, smoking or other forms of nicotine use, and past or current bisphosphonate treatment for osteoporosis. Diabetes mellitus, high blood pressure, hyperlipidemia, and OSA are frequent comorbidities of obesity that may complicate wound healing. A significant incidence of postoperative infections among diabetic patients undergoing maxillary or mandibular osteotomies has been reported, despite adequately controlled glucose levels. The inhalation of nicotine via cigarette smoke affects pulmonary function as well as tissue oxygenation and the healing of the surgical wounds. Smoking (nicotine ingestion) has been shown to delay the chondrogenic phase of bone (osteotomy) healing, thereby negatively affecting the circulation of elevated flaps and their healing after wound closure. It is known that bisphosphonate treatment for osteoporosis affects osteoclastic activity and may negatively affect bone healing (see Chapter 16).

Comprehensive Dental Rehabilitation

Recognizing when a patient who is seeking orthognathic surgery has additional dental needs beyond standard orthodontics is essential to the achievement of a favorable outcome (see Figs. 25-1 through 25-11). The need for periodontal evaluation and treatment as well as the need for complex dental restorative work should be considered for every adult patient before the initiation of a coordinated orthodontic and jaw surgical approach. This is especially true for the adult who has suffered dental and periodontal sequelae from a longstanding uncorrected dentofacial deformity and who may have undergone camouflage treatment in an attempt to neutralize the occlusion (e.g., orthodontics, occlusal equilibration, restorative dentistry). The American Board of Orthodontics now requires evidence of the pretreatment periodontal condition for all adult patients.[49] Proceeding with surgical and orthodontic management in an adult without considering a comprehensive dental rehabilitation plan is discouraged and only undertaken with full disclosure to the patient and discussion among the treating clinicians (see Chapters 6 and 16).

Unique Orthodontic Treatment Considerations

There has been a continuous increase in the number of adults seeking orthodontic treatment in North America as well as throughout Europe, South America, and the Asian countries. There is a worldwide increase in facial aesthetic awareness and a desire to maintain the dentition throughout life. The orthodontic movement of teeth in adults has potential effects on the periodontium that depend on the level of orthodontic force; the adequacy of alveolar bone and mucogingival tissue; and the presence of active periodontal disease (see Chapters 5 and 6). With aging, there may be progressive change of the periodontium with an increase in the crown-to-root ratio; a decrease in the resistance to spontaneous tooth migration; and a change in the center of resistance of periodontally affected teeth. The initial response of the periodontal ligament and the alveolar bone to orthodontic loading in the adult may be delayed; however, after tooth displacement has started, it can be carried out in the same way as it is for a teenager or a young adult. Although there is no known age limit to the orthodontic movement of teeth, the periodontium should be healthy when tooth movement is initiated.

As compared with a teenager, a middle-aged adult (i.e., someone >40 years old) with an uncorrected jaw deformity often requests a timely solution to his or her jaw, dental, and airway problems. Interestingly, the anticipation of an extended orthodontic treatment timeframe may be this

patient's greatest personal deterrent to proceeding. The adult is also more likely to present with advanced periodontal disease; dental migration and defective areas; traumatic occlusal problems; dental ankylosis; and the presence of fixed dental elements (e.g., titanium implants). These factors, by definition, will alter orthodontic mechanics. Contemporary orthodontic now offers a spectrum of options to improve the efficiency of treatment, including the use of light continuous forces; computer-aided design of orthodontic devices (e.g., Sure Smile, Insignia); and the regional acceleratory phenomena as described by Wilko and Wilko (see Chapter 5). These options must be viewed in light of their potential risks, complications, and costs (see Chapter 5).

Previous Suboptimal Facial Surgery

It is not uncommon to see an adult with an uncorrected dentofacial deformity who has undergone facial cosmetic procedures in an attempt to camouflage aesthetic concerns.[4,31,132-136] In the individual with an uncorrected jaw deformity, the most commonly seen camouflage procedures include reduction rhinoplasty; a chin implant; cheek implants; and, to a lesser degree, angle of the mandible or perinasal implants.[13,27,34,46,59,79,87,88,110,111,129,137,139] Autogenous and allogenic soft-tissue fillers and Botox injections (e.g., to lower the upper lip and to decrease a gummy smile) are also frequently tried.

A *rhinoplasty* carried out in the patient with an uncorrected jaw deformity patient is often over-reduced in an attempt to "shrink" the nose. This is most frequently seen in association with a horizontally retrusive lower face (e.g., short face growth pattern, long face growth pattern, isolated mandibular deficiency; see Chapters 19, 21, and 23). There may have been over-reduction of the nasal dorsum (i.e., bone and cartilage), over-resection of the lower lateral cartilages, and excess narrowing of the nasal base (i.e., Weir excisions; see Chapter 38). A reduction in nasal airflow as a result of the collapse of the internal or external nasal valves may have been an unanticipated side effect (see Chapter 10).

A *chin implant* placed in the patient with a mandibular deficiency often adds excess horizontal projection without addressing the vertical deformity; this will result in a deepened labiomental crease and an overly prominent pogonion (see Chapter 37). In the individual with a long face Class II open-bite growth pattern, the placement of a standard chin implant (i.e., without shortening the vertical chin height) further increases mentalis strain. Resorption of the bone below the implant and excess stretching of the overlying soft tissues with capsule formation may also occur. This is more frequently seen with the use of non-porous implant materials (e.g., Silastic) and when the implant is not secured in a stable manner (e.g., screw fixation) to the underlying bone (see Chapter 37). These failed camouflage surgical procedures further complicate plans for an anatomic reconstruction and effective facial rejuvenation.

Facial Aging and Rejuvenation

Background

The visual appearance of facial aging may be affected by all tissue layers and cell types, including the surface layer of the skin; the subcutaneous tissue; the superficial and deep mimetic muscles and the investing fascia (i.e., the submuscular aponeurotic system); the superficial and deep adipose tissue; and the retaining ligaments that connect the soft tissues to the skeleton and that separate them into compartments.[10,14,18,67,85,90,114] Although facial aging is visualized through the soft-tissue envelope, it is profoundly affected by the underlying maxillomandibular skeletal structures. Soft-tissue facial rejuvenation procedures generally focus on the following:

1. *The elevation, redraping, and excision of the skin, the subcutaneous tissue, and the submuscular aponeurotic system and platysma layer.* This is to correct gravitational (descent) changes that have occurred over time.
2. *The management of the volumetric changes of adipose tissue that occur with age.* This includes compartment-specific depletion (deflation), hypertrophy (expansion) of the fat cells, and stretching of the retaining ligaments that then affect the facial contours and curvatures.*
3. *The management of the surface layer of the skin.* This layer is affected by both hereditary (genetic) and environmental factors (e.g., chronic exposure to strong ultraviolet light, nicotine/cigarette smoke, dehydration, nutritional deficiencies, temperature extremes, traumatic influences).[15,16,20,23,45,72,78,93,138]
4. *The consideration of expressive muscle alteration with age (i.e., the "facial recurve" concept).* Ligamental relaxation may be a prominent component of facial aging. For example, it may be responsible for malar descent in the midface. In the nasal region, "tip drooping" may be caused by ligament loosening of the lower lateral cartilage and clockwise rotation of the columella.[124]

An important morphologic fact is that, *in the presence of a baseline uncorrected developmental dentofacial deformity, the overlying soft-tissue envelope will be distorted and less attractive* (see Figs. 25-1 through 25-11).[103,115,145] The long-standing skeletal dysmorphology will likely be responsible for a majority of the patient's concerns about premature and unfavorable facial aging. An example of this phenomenon is seen in patients with the short face growth pattern (see Chapter 23). With age, the decreased lower anterior facial height and the diminished horizontal projection associated with this condition result in the premature loss of a

*References 3, 21, 29, 30, 32, 50, 61, 64, 65, 73-77, 81, 82, 86, 122, 123, 131

youthful appearance (see Fig. 40-1). There will be accentuated perioral creases (i.e., marionette lines), early and excessive jowl formation, loose skin in the neck (i.e., a "turkey gobble" appearance), and a further worsening of the already deficient neck–jaw angle. Reconstruction of this pattern of dentofacial deformity in the adult to reflect Euclidean proportions is likely to have a profound rejuvenating effect (see Chapter 23).

The usual modus operandi of the cosmetic surgeon who has been asked to manage facial aging in the individual with a baseline dentofacial deformity involves neck and cheek soft-tissue flap undermining, redraping, and then the excision of skin that is under tension in the horizontal and vertical planes; this is also known as a *face lift.*[5,6,8,52,53,56,57,83,108,125,128,130] Augmentation implants may be considered and occasionally placed (e.g., the chin, the angle of the mandible, the cheek, the pyriform rim) in an attempt to modify underlying skeletal deficiencies.[13,27,34,46,59,60,79,87,88,137,139] In most cases, this approach predictably makes the face look different but not youthful or attractive. A natural-appearing rejuvenation in the adult with a dentofacial deformity requires reconstructing the normal curves and contours to the skeleton; these changes will then be favorably reflected in the overlying soft-tissue envelope. This is most naturally accomplished through orthognathic procedures that restore Euclidian proportions to the maxillomandibular complex.[22,44,103,105,112,113,115,118]

It is also true that facial aging—even in the presence of a proportionate skeleton—tends to gradually result in a transition from a heart-shaped face to a rectangular and then a pear-shaped face as gravity-induced descent and anterior drift of the soft-tissue envelope occur from the malar region superiorly through the nasolabial fold and from the submalar region to the jowls. At some point, as the individual ages, ideal rejuvenation is likely to require restoration of the lax and altered soft-tissue envelope, possibly via a brow lift, an eye lift, and a face lift. Fat volume redistribution frequently occurs with age (e.g., atrophy, hypertrophy) and may also benefit from rejuvenation procedures.[3,21,29,30,32,50,61,64,65,73,77,81,82,86] The surface layer of the skin may have weathered with age and be improved with quality skin care or resurfacing procedures (e.g., chemical peels, laser treatments, dermabrasion; see Chapter 40).[15,16,20,23,45,72,78,93,138]

The term *reconstruct* can be defined as "to bring to normal." The term *rejuvenate* means "to restore the look of the individual at a younger age." In the adult with a baseline dentofacial deformity, the creation of normal facial curvatures—first through skeletal reconstruction and then via soft-tissue rejuvenation procedures—is required to fully capture the appearance of health, vitality and beauty. If successful, the enhancement of facial appearance will be immediately obvious for all to see without the need for quantitative analytic assessment. The maxillofacial surgeon has a unique opportunity to reconstruct the skeletal structures and to rejuvenate the soft tissues of the adult with an uncorrected dentofacial deformity. Attention is also focused on the rehabilitation of the dentition and the periodontium and on the achievement of a more functional upper airway. Adults with dentofacial deformities who undergo successful orthognathic surgery will experience a quantum change: they will look younger and better as compared with how they looked earlier during their lives. A variety of reactions from the patient's social network are to be expected; these will range from positive responses to surprise, bewilderment, and even resentment. The individual's response to these initial reactions will depend on his or her personality type, preoperative preparation, and inner ego stability (see Chapter 7).

Periorbital and Midface Aging

Most cosmetic surgeons focus on the soft tissues when they are considering options for the management of periorbital and midface aging.[1,2,9,19,24,26,33,39,42,51,54,55,119] The traditional approach to aging in this region of the face focuses on the management of the laxity of the soft-tissue envelope and the reduction of protruding lower eyelid fat bags.[40,47,80,116] The spectrum of current theories of periorbital and midface aging as well as methods of management are varied and have strong advocates, including the following.

1. **Lambros** has demonstrated through longitudinal photographic imaging that descent probably does not have as large a role in the aging process as previously thought.[66]
2. **Coleman** believes that the role of fat atrophy with volume loss and secondary sagging of the soft-tissue envelope is critical.[25]
3. **Gosain** evaluated changes in the cheek fat pad with aging through magnetic resonance imaging and found a combination of hypertrophy and ptosis (rather than atrophy) to be an important factor.[48,150-152]
4. **Le Louarn** postulates that the facial mimetic muscles are a predominant cause of the visual appearance of aging in the midface. He believes that repeated motion (i.e., contraction) of these muscles results in the redistribution of the facial fat over time, which eventually creates the aged face.[69]
5. **Jelks** has drawn attention to the importance of the anatomic relationship between the ocular globe, the periorbital soft tissues, and the underlying skeleton with respect to midface aging.[62,63] A positive relationship (i.e., a positive vector) is said to exist when the most anterior projection of the globe in profile lies behind the lower eyelid margin.[91] A negative relationship (i.e., a negative vector) exists when the most anterior projection the globe in profile lies anterior to the lower lid. Individuals with a negative vector relationship are more likely to have scleral show, to exhibit an apparent loss of cheek prominence, and to have a deep nasojugal groove. They are also prone to suboptimal results if traditional soft-tissue eyelid rejuvenation procedures are carried out. To achieve the most favorable result, the underlying skeletal defi-

ciency and the lower lid laxity (i.e., lateral canthopexy) should also be addressed.

6. **Pessa and colleagues** agree with Jelks that skeletal deficiency is responsible for the negative vector relationship documented in adults with unattractive periorbital and midface aging.[95-101] These authors postulate that, with age, the infraorbital rims partially resorb and regress posterior relative to the cornea. They also believe that, with age, the upper aspect of the anterior maxilla is partially resorbed with progressive curve distortion (i.e., concavity). They infer that, to achieve the most favorable result, the underlying skeletal deficiency must be addressed.
7. In his analysis of periorbital and midface aging, **Yaremchuk** agrees with Jelks and Pessa that the negative affects of aging in the periorbital midface region are greatly influenced by the underlying skeleton.[140-149] He believes that a key element for attractive midface rejuvenation is reconstructing the skeleton to achieve convexity and a positive vector relationship. He accomplishes this via augmentation of the zygoma, the infraorbital rim, the anterior maxilla, and the base of the nose, as indicated. He frequently combines skeletal enhancement with lower lid and midface soft-tissue suspension to create and restore youthful and attractive periorbital aesthetics.

NOTE: This author believes that horizontal (sagittal) deficiency of the anterior maxilla—rather than a lack of projection of the zygomatic complex or of the infraorbital rims per se—is a frequent cause of the observed negative-vector eyelid relationship originally described by Jelks.

Maxillary horizontal (sagittal) deficiency that has been present since the teenage years is a frequent cause of the observed negative vector and results in an unattractive midface and lower eyelid appearance that worsens with age. Exceptions may include individuals of Asian decent with a hereditary tendency for excess bizygomatic width and limited zygomatic buttress projection. These individuals are more likely to benefit from zygomatic buttress (sagittal) augmentation to achieve aesthetic advantages.

A critical review of the literature provides little convincing evidence to support the theory put forth by Pessa and colleagues regarding a progressive deterioration or resorption of the infraorbital rim, the zygoma, or the anterior maxilla with advancing age in humans. The more logical explanation for the observed negative vector in some individuals is the presence of a developmental skeletal deformity that changes little after the teenage years (i.e., after skeletal maturity is reached). The common developmental jaw deformities with which maxillary horizontal (sagittal) deficiency is seen include: the long face growth pattern; maxillary deficiency with relative mandibular excess; maxillomandibular deficiency (i.e., the short face growth pattern); and cleft maxillary hypoplasia.

In the past, many cosmetic surgeons have not paid enough attention to these deep-layer skeletal morphologic facts. They tended to focus energy on the soft-tissue details without giving enough thought to the effects of the underlying skeletal framework for facial aging. Attempts to camouflage an underlying maxillary deficiency with techniques such as excess fat grafting to fill the soft-tissue lower eyelid envelope; the removal of excess skin to "smooth out" the eyelids; excessive repositioning or tightening of the deep soft-tissue layer (i.e., the submuscular aponeurotic system) to manage descent; and excessive hard-tissue augmentation of the zygomatic prominence and the infraorbital region are all likely to result in a poorly contoured and unnatural appearing midface and periorbital region. This camouflage approach should be compared to the more favorable direct management of the anterior maxilla and the perinasal regions through orthognathic surgery.

Consideration of Craniofacial Morphologic Change with Age: Does it Occur?

Although it has been much discussed, there is little convincing evidence to suggest that the craniofacial skeletal structures either significantly atrophy or hypertrophy with age after traditional growth maturity is reached.[11,12,17,28,35-38,43,68,84,89,92,109,120,126,127,153] This should not be confused with the documented facial changes that do occur in association with the loss of teeth; occlusal surface erosion; the ongoing eruption of teeth; and disease of the periodontium. With extensive dental loss, significant deterioration of the horizontal projection, the vertical height, and transverse width of the midface and the lower face will unquestionably alter the soft-tissue drape and result in a more aged look. After skeletal maturity is reached (i.e., 14 to 16 years of age in girls and 16 to 18 years of age in boy) and in the absence of dental loss or significant deterioration, neither notable atrophy nor hypertrophy of the craniofacial prominences has been convincingly proven to occur. This specifically includes the following structures:

- The supraorbital ridges
- The lateral orbital rims
- The infraorbital rims
- The zygomatic prominences
- The nasal height and the frontonasal angle
- The pyriform rims and the anterior nasal spine
- The gonial regions of the mandible
- The pogonion prominence

Midface Skeletal Change with Age: Does it Occur?

In 1927, **Hellman** proposed the theory that the facial skeleton continues to grow throughout life[58]; he called this process *morphological differentiation.* **Pessa and colleagues** proposed an algorithm of facial aging to support Hellman's theory on the basis of the review of a limited number of radiographs that suggested to him that bony changes of the midface (i.e., the zygoma and the infraorbital rim) occurred with age.[95] The data that were used to support

this theory were collected from computed tomography scans performed on just 12 men: six with a mean age of 20 years and six with a mean age of 56 years. These scans were not obtained for the same individuals over time, and they were not selected in a controlled fashion. Pessa made four difficult-to-reproduce angular measurements in each midface region on each scan (i.e., the glabellar angle, the orbital angle, the pyriform angle, and the angle of the maxillary wall). He concluded from the limited data that "the craniofacial skeleton continues to change with age resulting in remodeling of the maxilla [clockwise rotation] relative to the cranial base."[95] Despite Pessa's theory, no scientific data confirm significant changes in the midface skeleton with age.

Levine and colleagues have also found fault with the "skeletal-aged" theories of Pessa and colleagues.[70,71] They contend that Pessa's theory of midface skeletal atrophy with age is based on an unsound interpretation of data from a limited number of non-longitudinal computed tomography scans and that it is not consistent with clinical observations or available research. Levine does believe that, in the presence of maxillary deficiency, soft-tissue descent and volume loss with age lead to the frequently observed unattractive changes seen in the midface periorbital region. He goes on to state that the negative vector relationship of the ocular globe and the lower eyelid in this subgroup of adults is more likely the result of a longstanding horizontal (sagittal) maxillary hypoplasia that has been present since the teenage years. It is the longstanding developmental midface skeletal deficiency that does not provide adequate support for the overlying soft tissues.[41] As a result, retaining ligaments eventually give way, the fat may then atrophy or protrude, and the skin becomes lax.

Whether the negative vector eyelid aging characteristics (i.e., rounding of the palpebral fissure, lengthening of the lower lid, and loss of cheek prominence) results from a developmental skeletal midface hypoplasia or from skeletal atrophy with continued aging, most informed clinicians agree that the correction of the baseline bony deficiency is a key aspect of a natural-appearing rejuvenation. **Yaremchuk's analysis** and clinical results are convincing evidence that the underlying skeleton is often where attention should be directed if effective rejuvenation is desired.[141-149] Free fat grafting, the injection of various fillers, and the redistribution of the midface and lower lid soft tissues have more limited roles in the presence of a deficient skeletal framework. Too much attention given to the soft tissues or to imprecise skeletal augmentation will predictably result in an operated look that is visually obvious for all to see.

Mandibular Skeletal Change with Age: Does it Occur?

Pessa and colleagues also hypothesized that the mandible changes in shape during the aging process after normal skeletal maturity.[98] The authors tested this theory in just 16 subjects (eight male subjects and eight female subjects) with the use of acetate tracings of frontal (posteroanterior) cephalograms taken during two different time frames. The first group ranged in age from 5 to 17 years, and the second group ranged in age from 46 to 60 years. Seven difficult-to-reproduce data points were chosen along the inferior border of the mandible on each radiograph; an analysis was carried out with the use of these points. From this data, the authors theorized that the "youthful mandible had a greater degree or curvature whereas the mature [middle-aged] mandible can be described as having a less convex curvature" and that "the mandible increases in size [length] with continued growth." A critical review of Pessa's methods suggests that the posteroanterior cephalometric radiographs of the young study subjects were not standardized for head position (i.e., flexion and extension) as compared with the radiographs taken of the older subjects. Therefore, the accuracy of the comparison measurements taken along the inferior border of the mandible on each of the non-longitudinal radiographs is questionable. This inaccuracy is caused by inherent distortions that result from differences of positioning of the subjects' heads at the time that the two-dimensional radiographs were taken.

Shaw and colleagues recently reviewed computed tomography scans of dentate Caucasian subjects; they included an equal number of women (n = 60) and men (n = 60) in an attempt to identify changes in mandibular morphology with age.[121] The patient population was not evaluated longitudinally, and it was not standardized. The radiographs were taken from a hospital-based data pool and not controlled to exclude those with jaw deformities, significant malocclusions, congenital anomalies, or trauma. The researchers further subdivided the radiographs into three groups in accordance with the age brackets of the patients (i.e., 20 to 40 years, 41 to 64 years, and >65 years). The authors took measurements from each scan related to the width and length of the mandible and at the gonial angles. Interestingly, the authors drew conclusions from these data that are contradictory to those of published studies carried out by other investigators with the similar intention of clarifying the effects of age on mandibular morphology. For example, the study by **Pecora and colleagues** suggests that the mandible continues to grow in length in both men and women during aging.[94] Pessa and colleagues also suggest that there is an increase in mandibular width, height, and length with aging. The analysis by **Bartlett and colleagues** of postmortem human skull data suggest that there is an increase in the width—but not the length—of the mandible with age.[7]

NOTE: Despite differing assertions and opinions offered by a handful of authors, in the absence of the loss of the teeth or the significant alteration of the dentition, no reproducible convincing data confirms significant changes in the shape of the mandible with aging.

Recovery in the Adult after Orthognathic Surgery

Physiologic and Psychosocial Effects

Although the middle-aged adult's basic maxillofacial skeletal and soft-tissue healing capacity is in most cases similar to that of a teenager or young adult, their return of energy level, oromotor function, sensation (i.e., the infraorbital nerve and the inferior alveolar nerve), and psychosocial readiness to resume preoperative daily activities and work-related matters may be more prolonged. For example, the teenager will typically return to schoolwork, computer work, reading, and desk-type activities by 2 weeks after his or her procedures, even after complex jaw and intranasal surgery. These patients may have concerns about the perceptions of their peers, but in general a rapid return to the presurgery environment is expected. For the middle-aged adult, returning to a competitive activity schedule at work (e.g., business telephone conversations, conferences, meeting new clients, engaging in sales-type activities) and feeling comfortable around family and friends is generally more challenging and will require extra preparation, planning, and discussion between the surgeon and the patient. Aspects of greater concern for the middle-aged adult as compared with the teenager include rehabilitation requirements and personal adjustments to the following: 1) facial region sensory loss and recovery; 2) limitations of mouth opening; 3) changes in occlusion; 4) changes in speech articulation; 5) lingering general fatigue; 6) persistent soft-tissue swelling and "tightness" during dynamic expression; and 7) altered body image.

NOTE: This author has found that an adult patient's recovery is best and his or her satisfaction is highest when he or she has an upbeat and optimistic attitude; when immediate family members and friends are available and supportive; when there is a documented need for and advantage to simultaneous improvements in: the upper airway; the occlusion and dental health; and when there are clearly defined tangible aesthetic objectives.

Setting Clear Objectives with Realistic Expectations

All parties involved should agree on the treatment objectives and accept the risks and limitations of the planned surgical procedures. Unfortunately, the patient's preoperative psychosocial assessment, the surgeon's understanding of unique wound healing requirements, and the uncertainties of surgery are not completely predictable. Furthermore, orthognathic surgery should not be carried out with a belief that it will repair broken personal relationships, save jobs, or overcome financial concerns. The reality is that a small percentage of patients will perceive an unfavorable surgical outcome even when the clinicians feel that the results are at least satisfactory. Others will not accept a complication even though the risks were explained in advance and all reasonable precautions were taken. Experience has shown that there are no shortcuts to thorough patient education; comprehensive care must be provided every step of the way (see Chapter 7).

Body Dysmorphic Disorder

To minimize patient disappointment after surgery, an informal (and, in some cases, a more rigorous) psychosocial assessment is indicated. The clinician must keep in mind that some individuals who are seeking treatment will have a true psychiatric disorder. Body dysmorphic disorder is a medical condition that can be difficult for a clinician to recognize before surgery. If this condition is present, it will be a predictable cause of unhappiness after surgery. It is estimated that 7% to 15% of individuals who are pursuing cosmetic surgery may have body dysmorphic disorder (see Chapter 7).

Sensory Loss and Incidence of Dysesthesia

Inferior Alveolar and Mental Nerve Injury and Recovery

During sagittal split ramus osteotomies of the mandible, the inferior alveolar nerve can be directly damaged by a burr on a rotary drill, a blade on a reciprocating saw, or a chisel used to complete the split. The nerve can also be compressed during osteotomy stabilization (i.e., plate and screw fixation). Studies document that, with time, inferior alveolar nerve sensory function is generally sufficient in most patients, although complete recovery is uncommon. The exact reasons why, over time, most patients experience only a degree of simple loss of sensation in the distribution of the inferior alveolar nerve, some continue to experience active sensations that are not normally present but are of little concern, and a small percentage have active sensations that are subjectively uncomfortable or painful (i.e., dysesthesia) is not known. Zuniga and colleagues (see chapter 16) report a 5% incidence of dysesthesia of the inferior alveolar nerve after sagittal split ramus osteotomy (see Chapter 16 for a more complete review of the literature).

Infraorbital Nerve Injury and Recovery

A degree of injury to the infraorbital nerve at the time of Le Fort I osteotomy that results in paresthesia is expected, whereas long-term dysesthesia of the cheek skin, the upper lip, the palatal mucosa, and the gingiva is less common. Infraorbital nerve injury may result from compression, stretching, or direct injury from saw blades, burs, or hand-held instruments. A study by Thygesen and colleagues (see Chapter 16) found continued changes (i.e., less than full recovery had occurred) in somatosensory infraorbital nerve function 12 months after Le Fort I osteotomy in 7% to 60% of patients. This included the regions of the cutaneous skin, the lip, the gingiva, and the palatal mucosa. Fortunately, the authors did not find instances of significant

dysesthesia associated with the documented sensory loss in any of the study patients (see Chapter 16 for a more complete review of the literature).

Patient Age and Sensory Recovery

It is believed that older patients (i.e., >25 years old and especially >40 years old) who sustain the same extent of injury to either the infraorbital nerve or the inferior alveolar nerve after maxillary or mandibular osteotomies, respectively, will not experience the same degree of subjective sensory recovery as younger individuals. It appears that the time required for "acceptable" subjective recovery of facial and oral sensation is longer and that the percentage of middle-aged adults who have undergone these operations and who interpret their lack of full sensory return as dysesthesia (i.e., uncomfortable sensory loss) is higher than that observed among teenagers and young adults (see Chapter 16). Adults are more likely to complain about a lack of sensory return to the mucosa of the palate and the gingiva. They may be annoyed by the lack of proprioception when flossing and when managing food on the roof of the mouth. Interestingly, Pogrel and colleagues documented that, with time, men tend to achieve a more tolerable decrease in these uncomfortable sensory symptoms than women.[102]

Conclusions

The surgical techniques for the correction of a dentofacial deformity are generally the same for the middle-aged adult as for the teenager and the young adult. To achieve a favorable result in the adult orthognathic patient, it is essential to recognize age-related treatment pitfalls such as medical risk factors; progressive upper airway obstruction; dental rehabilitation needs, including periodontal and restorative aspects; effects of facial soft-tissue aging; and the often prolonged physiologic and psychosocial response to surgery. An important observation is that, in the presence of a baseline jaw disharmony, the overlying soft-tissue envelope will tend to show premature and unattractive aging. Adults who undergo successful orthognathic surgery are likely to experience a quantum change when they look younger and more attractive than they did earlier during their lives.

References

1. Aiache AE, Ramirez OH: The suborbicularis oculi fat pads: An anatomic and clinical study. *Plast Reconstr Surg* 95:37, 1995.
2. Anderson RD, Lo MW: Endoscopic malar/midface suspension procedure. *Plast Reconstr Surg* 102:2196, 1998.
3. Ascher BA, Coleman S, Alster T, et al: Full scope of effect of facial lipoatrophy: A framework of disease understanding. *Dermatol Surg* 32:1058–1069, 2006.
4. Bains JW, Elia JP: The role of facial skeletal augmentation and dental restoration in facial rejuvenation. *Aesthetic Plast Surg* 18:243, 1994.
5. Baker DC: Lateral SMASectomy. *Plast Reconstr Surg* 100:509–513, 1997.
6. Baker T, Stuzin J: Personal technique for facelifting. *Plast Reconstr Surg* 100:502–508-513, 1997.
7. Bartlett SP, Grossman R, Whitaker LA: Age-related changes of the craniofacial skeleton: An anthropometric and histologic analysis. *Plast Reconstr Surg* 90:592–600, 1992.
8. Barton FE, Jr: Rhytidectomy and the nasolabial fold. *Plast Reconstr Surg* 90:601, 1992.
9. Barton FE, Jr, Kenkel JM: Direct fixation of the malar pad. *Clin Plast Surg* 24:329, 1997.
10. Bass NM: The aesthetic analysis of the face. *Eur J Orthod* 13:343–350, 1991.
11. Behrents RG, editor: *Growth in the aging craniofacial skeleton. Monograph No 17, Craniofacial Growth Series*, Ann Arbor, Michigan, 1985, Center for Human Growth and Development, The University of Michigan.
12. Behrents RG: *An atlas of growth in the aging craniofacial skeleton. Monograph 18*, Ann Arbor, Michigan, 1985, Center for Human Growth and Development, The University of Michigan.
13. Binder WJ, Bloom DC: The use of custom-designed midfacial and submalar implants in the treatment of facial wasting syndrome. *Arch Facial Plast Surg* 6:394–397, 2004.
14. Bishara SE, Jakobsen JR, Hession TJ, Treder JE: Soft tissue profile changes from 5 to 45 years of age. *Am J Orthod Dentofacial Orthop* 114:698–706, 1998.
15. Bologna JL: Dermatologic and cosmetic concerns of the older woman. *Clin Geriatr Med* 9:209–229, 1993.
16. Brown AM, Kaplan ML, Brown ME: Phenol-induced histological skin changes: Hazards, technique, and uses. *Br J Plast Surg* 13:158, 1960.
17. Burr DB: Muscle strength, bone mass, and age-related bone loss. *J Bone Miner Res* 12:1547, 1997.
18. Burstone CJ: The integumental profile. *Am J Orthod* 44:1–25, 1958.
19. Byrd HS, Andochick SE: The deep temporal lift: A multiplanar, lateral brow, temporal, and upper face lift. *Plast Reconstr Surg* 97:928–937, 1996.
20. Carruthers J, Fagien S, Matarasso SL; Botox Consensus Group: Consensus recommendations on the use of botulinum toxin type A in facial aesthetics. *Plast Reconstr Surg* 114(Suppl):1–22, 2004.
21. Carruthers JDA, Carruthers A: Facial sculpting and tissue augmentation. *Dermatol Surg* 31:1604–1612, 2005.
22. Chemello PD, Wolford LM, Buschang PH: Occlusal plane alteration in orthognathic surgery—Part II: Long-term stability of results. *Am J Orthod Dentofacial Orthop* 106:434–440, 1994.
23. Cheng JT, Perkins SW, Hamilton MM: Collagen and injectable fillers. *Otolaryngol Clin North Am* 35:73–85, 2002.
24. Chiu ES, Baker DC. Endoscopic brow lift: A retrospective review of 628 consecutive cases over 5 years. *Plast Reconstr Surg* 112:628–633, 2003.
25. Coleman SR: Structural fat grafting: More than a permanent filler. *Plast Reconstr Surg* 118(3 Suppl):108S–120S, 2006.
26. Collawn SS, Vasconez LO, Gamboa M, et al: Subcutaneous approach for elevation of the malar fat pad through a prehairline incision. *Plast Reconstr Surg* 97:836, 1996.
27. Constantinides MS, Galli SK, Miller PJ, Adamson PA: Malar, submalar, and midfacial implants. *Facial Plast Surg* 16:35–44, 2000.
28. Daher JC, Cosac OM, Domingues S: Face-lift: The importance of redefining facial contours through facial liposuction. *Ann Plast Surg* 21:1, 1988.
29. Donofrio LM: Fat distribution: A morphologic study of the aging face. *Dermatol Surg* 26:1107–1112, 2000.
30. Donofrio LM: Panfacial volume restoration with fat. *Dermatol Surg* 31:1496–1505, 2005.
31. Ellenbogen R, Motykie G, Youn A, et al: Facial reshaping using less invasive methods. *Aesthet Surg J* 25:144–152, 2005.
32. Ersek RA, Chang P, Salisbury MA: Lipo layering of autologous fat: An improved

technique with promising results. *Plast Reconstr Surg* 101:820–826, 1998.
33. Finger ER: Transmalar subperiosteal midface lift: Early results with a simplified approach. *Aesthet Surg J* 16:261, 1996.
34. Flowers RS: Tear trough implants for correction of tear trough deformity. *Clin Plast Surg* 20:403, 1993.
35. Formby WA: *Longitudinal growth changes in the face from 18 to 42 years [master's thesis]*. Oklahoma City, Okla, 1991, The University of Oklahoma Health Sciences Center.
36. Frost HM: Tetracycline-based histological analysis of bone remodeling. *Calcif Tissue Int* 3:211, 1969.
37. Frost HM: Changing concepts in skeletal physiology: Wolff's Law, the mechanostat and the "Utah Paradigm." *J Hum Biol* 10:599, 1998.
38. Frost HM: Perspective on the estrogen-bone relationship and postmenopausal bone loss: A new model. *J Bone Miner Res* 14:1473, 1999.
39. Furnas DW: Festoons, mounds, and bags of the eyelids and cheek. *Clin Plast Surg* 20:367, 1993.
40. Goldberg RA, Wu JC, Jesmanowicz A, Hyde JS: Eyelid anatomy revisited: Dynamic high-resolution magnetic resonance images of Whitnall's ligament and upper eyelid structures with the use of a surface coil. *Arch Ophthalmol* 110:1598, 1992.
41. Goldberg RA, Relan A, Hoenig J: Relationship of the eye to the bony orbit, with clinical correlations. *Aust N Z J Ophthalmol* 27:98, 1999.
42. Goldberg RA: The three periorbital hollows: A paradigm for periorbital rejuvenation. *Plast Reconstr Surg* 116:1796–1804, 2004.
43. Goldstein MS: Changes in dimensions and form of the face and head with age. *Am J Phys Anthropol* 22:37–89, 1936.
44. Goncalves JR, Bushang PH, Goncalves DG: Postsurgical stability of oropharyngeal airway changes following counterclockwise maxillo-mandibular advancement surgery. *J Oral Maxillofac Surg* 64:755–762, 2006.
45. Gonzalez-Ulloa, M, Castillo A, Stevens E, et al: Preliminary study of the total restoration of the facial skin. *Plast Reconstr Surg* 13:151–161, 1954.
46. Gonzalez-Ulloa M: Building out the malar prominences as an addition to rhytidectomy. *Plast Reconstr Surg* 53:293, 1974.
47. Gosain AK, Amarante MTJ, Hyde JS, Yousif NJ: A dynamic analysis of changes in the nasolabial fold using magnetic resonance imaging: Implications for facial rejuvenation and facial animation surgery. *Plast Reconstr Surg* 98:622, 1996.
48. Gosain AK, Klein MH, Sudhakar PV, et al: A volumetric analysis of soft-tissue changes in the aging midface using high-resolution MRI: Implications for facial rejuvenation. *Plast Reconstr Surg* 115:1143–1152, 2005.
49. Grubb JE, Greco PM, English JD, et al: Radiographic and periodontal requirements of the American Board of Orthodontics : a modification in the case display requirements for adult and periodontally involved adolescent and preadolescent patients. *Am J Orthod Dentofacial Orthop* 134:3–4, 2008
50. Guerrerosantos J: Long-term outcome of autologous fat transplantation in aesthetic facial recontouring: Sixteen years of experience with 1936 cases. *Clin Plast Surg* 27:515–543, 2000.
51. Gunter JP, Antrobus SD: Aesthetic analysis of the eyebrows. *Plast Reconstr Surg* 99:1808, 1997.
52. Hamra ST: The deep-plane rhytidectomy. *Plast Reconstr Surg* 86:53–61, 1990.
53. Hamra ST: Composite rhytidectomy. *Plast Reconstr Surg* 90:1, 1992.
54. Hamra ST: Arcus marginalis release and orbital fat preservation in midface rejuvenation. *Plast Reconstr Surg* 96:354, 1995.
55. Hamra ST: The role of orbital fat preservation in facial aesthetic surgery: A new concept. *Clin Plast Surg* 23:17, 1996.
56. Hamra ST: Frequent face lift sequelae: Hollow eyes and the lateral sweep: Cause and repair. *Plast Reconstr Surg* 102:1658, 1998.
57. Heinrichs HL, Kaidi A: Subperiosteal face lift: A 200-case, 4-year review. *Plast Reconstr Surg* 102:843, 1998.
58. Hellman M: Changes in the human face brought about by development. *Int J Oral Surg Radiol* 20(37):463–465, 1927.
59. Hinderer UT: Malar implants for the improvement of the facial appearance. *Plast Reconstr Surg* 56:157, 1975.
60. Hinderer UT: Nasal base, maxillary and infraorbital implants—alloplastic. *Clin Plast Surg* 18:87, 1991.
61. Jackson IT: Buccal fat pad removal. *Aesthet Surg J* 23:484–485, 2003.
62. Jelks GW, Jelks EB: The influence of orbital and eyelid anatomy on the palpebral aperture. *Clin Plast Surg* 18:193–204, 1991.
63. Jelks GW, Jelks EB: Preoperative evaluation of the blepharoplasty patient: Bypassing the pitfalls. *Clin Plast Surg* 20:213, 1993.
64. Kanchwala SK, Bucky LP: Facial fat grafting: The search for predictable results. *Facial Plast Surg* 19:137, 2003.
65. Kanchwala SK, Holloway L, Bucky LP: Reliable soft tissue augmentation: A clinical comparison of injectable soft tissue fillers for facial-volume augmentation. *Ann Plast Surg* 55:30, 2005.
66. Lambros V: Observations on periorbital and midface aging. *Plast Reconstr Surg* 120:1367–1376, 2007.
67. LaTrenta GS: *Atlas of aesthetic face and neck surgery*, Philadelphia, 2004, Saunders.
68. Lavater JC: *Essays on physiognomy, Vol 1-3*, London, 1789, J & J Robinson.
69. Le Louarn C, Buthiau D, Buis J: Structural aging: The facial recurve concept. *Aesthetic Plast Surg* 31:213–218, 2007.
70. Levine RA, Garza JR, Wang PTH, et al: Adult facial growth: Applications to aesthetic surgery. *Aesthetic Plast Surg* 27: 265–268, 2003.
71. Levine RA: Letter. *Plast Reconstr Surg* 121:698, 2008.
72. Leyden JJ: Clinical features of aging skin. *Br J Dermatol* 122(Suppl):1, 1990.
73. Liang M, Narayanan K, Davis PL, Futrell JW: Evaluation of facial fat distribution using magnetic resonance imaging. *Aesthetic Plast Surg* 15:313–319, 1991.
74. Little JW: Volumetric perceptions in midfacial aging with altered priorities for rejuvenation. *Plast Reconstr Surg* 105:252–266, 2000.
75. Little JW: Three-dimensional rejuvenation of the midface: Volumetric resculpture by malar imbrication. *Plast Reconstr Surg* 105:267–285, 2000.
76. Loeb R: Nasolabial fold undermining and fat grafting based on histologic study. *Aesthetic Plast Surg* 15:61, 1991.
77. Matarasso A: Managing the buccal fat pad. *Aesthet Surg J* 26:330–336, 2006.
78. Matarasso SL, Carruthers JD, Jewell ML, et al: Consensus recommendations for soft tissue augmentation with non-animal stabilized hyaluronic acid (Restylane). *Plast Reconstr Surg* 117(Suppl):3S, 2006.
79. May JW, Jr, Zenn MR, Zingarelli P: Subciliary malar augmentation and cheek advancement: A 6-year study on 22 patients undergoing blepharoplasty. *Plast Reconstr Surg* 96:1553, 1995.
80. Mendelson BC, Muzaffar AR, Adams WP, Jr: Surgical anatomy of the midcheek and malar mounds. *Plast Reconstr Surg* 110:885–896, 2002.
81. Millard DR, Pigott RW, Hedo A: Submandibular lipectomy. *Plast Reconstr Surg* 41:513, 1968.
82. Millard DR, Jr, Yuan RT, Devine JW, Jr: A challenge to the undefeated nasolabial folds. *Plast Reconstr Surg* 80:37, 1987.
83. Miller T: Face lift: Which technique? *Plast Reconstr Surg* 100:501, 1997.
84. Mittleman H: The anatomy of the aging mandible and its importance to facelift surgery. *Facial Plast Surg Clin North Am* 2:301–309, 1994.
85. Mitz V, Peyronie M: The superficial musculo-aponeurotic system (SMAS) in the parotid and cheek area. *Plast Reconstr Surg* 58:80–88, 1976.
86. Mladick RA: Lipoplasty: An ideal adjunctive procedure for the face lift. *Clin Plast Surg* 16:333, 1989.
87. Mladick RA: Alloplastic cheek augmentation. *Clin Plast Surg* 18:29, 1991.
88. Mole B: The use of Gore-Tex implants in aesthetic surgery of the face. *Plast Reconstr Surg* 90:200–206, 1992.
89. Montagna W, Carlisle K: Structural changes in aging skin. *Br J Dermatol* 122(Suppl 35):61–70, 1990.
90. Moss CJ, Mendelson BC, Taylor GI: Surgical anatomy of the ligamentous attachments in

the temple and periorbital regions. *Plast Reconstr Surg* 105:1475–1490, 2000.
91. Mulliken JB, Godwin SL, Pracharktam N, Altobelli DE: The concept of sagittal orbital-globe relationship in craniofacial surgery. *Plast Reconstr Surg* 97:700, 1996.
92. Nanda RS: The rates of growth of several facial components measured from serial cephalometric roentgenograms. *Am J Orthod* 41:658–665, 1955.
93. Oba A, Edwards C: Relationships between changes in mechanical properties of the skin, wrinkling, and destruction of dermal collagen fiber bundles caused by photoaging. *Skin Res Technol* 12:283–288, 2006.
94. Pecora NG, Baccetti T, McNamara JA, Jr: The aging craniofacial complex: A longitudinal cephalometric study from late adolescence to late adulthood. *Am J Orthod Dentofacial Orthop* 134: 496–505, 2006.
95. Pessa JE, Zadoo VP, Mutimer KL, et al: Relative maxillary retrusion as a natural consequence of aging: Combining skeletal and soft-tissue changes into an integrated model of midfacial aging. *Plast Reconstr Surg* 102:205–212, 1998.
96. Pessa JE, Desvigne LD, Lambros VS, et al: Changes in ocular-to-orbital rim position with age: implications for aesthetic blepharoplasty of the lower eyelids. *Aesthetic Plast Surg* 23:337–342, 1999.
97. Pessa JE, Peterson ML, Thompson JW, et al: Pyriform augmentation as an ancillary procedure in facial rejuvenation surgery. *Plast Reconstr Surg* 103:683, 1999.
98. Pessa JE, Zadoo VP, Yuan C, et al. Concertina effect and facial aging: Nonlinear aspects of youthfulness and skeletal remodeling, and why, perhaps, infants have jowls. *Plast Reconstr Surg* 103:635–644, 1999.
99. Pessa JE: An algorithm of facial aging: Verification of Lambros' theory by three-dimensional stereolithography, with reference to the pathogenesis of midfacial aging, scleral show, and the lateral suborbital trough deformity. *Plast Reconstr Surg* 106:479–488, 2000.
100. Pessa JE, Chen Y: Curve analysis of the aging orbital aperture. *Plast Reconstr Surg* 109:756–760, 2002.
101. Pessa JE: Reply. *Plast Reconstr Surg* 121:699, 2008.
102. Pogrel MA, Jergensen R, Burgon E, Hulme D: Long-term outcome of trigeminal nerve injuries related to dental treatment. *J Oral Maxillofac Surg* 69: 2284–2288, 2011.
103. Posnick JC, Fantuzzo J, Orchin J: Deliberate operative rotation of the maxillo-mandibular complex to alter the A-point to B-point relationship for enhanced facial esthetics. *J Oral Maxillofac Surg* 64:1687–1695, 2006.
104. Posnick JC, Fantuzzo JJ, Troost T: Simultaneous intranasal procedures to improve chronic obstructive nasal breathing in patients undergoing maxillary (Le Fort I) osteotomy. *J Oral Maxillofac Surg* 65:2273–2281, 2007.
105. Posnick JC, Wallace J: Complex orthognathic surgery assessment of patient satisfaction. *J Oral Maxillofac Surg* 66:934, 2008.
106. Posnick JC, Agnihotri N: Consequences and management of nasal airway obstruction in the dentofacial deformity patient. *Curr Opin Otolaryngol Head Neck Surg* 18:323–331, 2010.
107. Posnick JC, Agnihotri N: Managing chronic nasal airway obstruction at the time of orthognathic surgery: A twofer. *J Oral Maxillofac Surg* 69:695–701, 2011
108. Psillakis JM, Rumley TO, Camargos A: Subperiosteal approach as an improved concept for correction of the aging face. *Plast Reconstr Surg* 82:383, 1988.
109. Raskin E, LaTrenta GS: Why do we age in our cheeks? *Aesthet Surg J* 27:19–28, 2007.
110. Rees T, Ashley F: Treatment of facial atrophy with liquid silicone. *Am J Surg* 111:531–535, 1966.
111. Rees T, Ashley F, Delgado J: Silicone fluid injections for facial atrophy. *Plast Reconstr Surg* 52:118–127, 1972.
112. Reyneke JP, Evans WG: Surgical manipulation of the occlusal plane. *Int J Adult Orthodon Orthognath Surg* 5:99–110, 1990.
113. Reyneke JP, Bryant RS, Suuronen R, Becker PJ: Postoperative stability following clockwise and counter-clockwise rotation of the maxillomandibular complex compared to conventional orthognathic treatment. *Br J Oral Maxillofac Surg* 45:56–64, 2007.
114. Rogers BO: A chronologic history of cosmetic surgery. *Bull N Y Acad Med* 47:265–302, 1971.
115. Rosen HM: Facial skeletal expansion: Treatment strategies and rationale. *Plast Reconstr Surg* 89:798–808, 1992.
116. Rubin LR, Mishriki Y, Lee G: Anatomy of the nasolabial fold: The keystone of the smiling mechanism. *Plast Reconstr Surg* 83:1, 1989.
117. Rugh JD, Solberg WK: Oral health status in the United States: Temporomandibular disorders. *J Dent Educ* 49: 399–405, 1985
118. Sarver DM, Rousso DR: Plastic surgery combined with orthodontic and orthognathic procedures. *Am J Orthod Dentofacial Orthop* 126:305–307, 2004.
119. Sasaki GH, Cohen AT: Meloplication of the malar fat pads by percutaneous cable suture technique for midface rejuvenation: Outcome study (392 cases, 6 years' experience). *Plast Reconstr Surg* 110:635, 2002.
120. Schubert P, Bailey LJ, White RP, Proffit WR: Long-term cephalometric changes in untreated adults compared to those treated with orthognathic surgery. *Int J Adult Orthodon Orthognath Surg* 14:91–99, 1999.
121. Shaw RB, Kahn DM: Aging of the midface bony elements: A three-dimensional computed tomographic study. *Plast Reconstr Surg* 119:675–681, 2007.
122. Staten MA, Totty WV, Kohrt WM: Measurements of fat distribution by magnetic resonance imaging. *Invest Radiol* 24:345, 1989.
123. Stuzin JM, Wagstrom L, Kawamoto HK, et al: The anatomy and clinical implications of the buccal fat pad. *Plast Reconstr Surg* 85:29–37, 1990.
124. Stuzin JM, Baker TJ, Gordon HL: The relationship of the superficial and deep facial fascias: Relevance to rhytidectomy and aging. *Plast Reconstr Surg* 89:441–451, 1992.
125. Stuzin JM, Baker TJ, Gordon HL, Baker TM: Extended SMAS dissection as an approach to midface rejuvenation. *Clin Plast Surg* 22:295, 1995.
126. Tallgren A: Neurocranial morphology and aging: A longitudinal roentgen cephalometric study of adult Finnish women. *Am J Phys Anthropol* 41:285–294, 1974.
127. Tallgren S, Solow B: Long-term changes in hyoid position and craniocervical posture in complete denture wearers. *J Dent Res* 60:473, 1981.
128. Teimourian B: Face and neck suction-assisted lipectomy associated with rhytidectomy. *Plast Reconstr Surg* 72:627, 1983.
129. Terino EO: Alloplastic contouring in the malar-midface-middle third facial aesthetic unit. In Terino EO, Flowers RS, editors: *The art of alloplastic facial contouring*, St Louis, 2000, Mosby.
130. Tessier P: Subperiosteal face lift. *Ann Chir Plast Esthet* 34:193, 1989.
131. Trepsat F: Volumetric face lifting. *Plast Reconstr Surg* 108:1358, 2001.
132. Van der Dussen FN, Egyedi P: Premature aging of the face after orthognathic surgery. *J Craniofac Surg* 18:335, 1990.
133. Whitaker LA: Facial skeletal contouring for aesthetic purposes. *Plast Reconstr Surg* 69:245, 1982.
134. Whitaker LA: Aesthetic augmentation of the malar-midface structures. *Plast Reconstr Surg* 90:337–341, 1987.
135. Whitaker LA, Bartlett SP: Skeletal alteration as a basis for facial rejuvenation. *Clin Plast Surg* 18:197, 1991.
136. Whitaker LA: Temporal and malar-zygomatic reduction and augmentation. *Clin Plast Surg* 18:55, 1991.
137. Wilkinson TS: Complications in aesthetic malar augmentation. *Plast Reconstr Surg* 71:643, 1983.
138. Wise JB, Greco TG: Injectable treatments for the aging face. *Facial Plast Surg* 22:140–146, 2006
139. Wolfe SA: Malar augmentation using autogenous materials. *Clin Plast Surg* 18:39, 1991.
140. Yaremchuk MJ, Israeli D: Paranasal implants for correction of midface concavity. *Plast Reconstr Surg* 102:1676–1684, discussion 1685, 1998.
141. Yaremchuk MJ: Mandibular augmentation. *Plast Reconstr Surg* 106:697–706, 2000.

142. Yaremchuk MJ: Infraorbital rim augmentation. *Plast Reconstr Surg* 107:1585–1592, discussion 1593-1595, 2001.
143. Yaremchuk MJ: Subperiosteal and full-thickness skin rhytidectomy. *Plast Reconstr Surg* 107:1045–1057, 2001.
144. Yaremchuk MJ: Restoring palpebral fissure shape after previous lower blepharoplasty. *Plast Reconstr Surg* 111:441–450, 2003.
145. Yaremchuk MJ: Improving periorbital appearance in the "morphologically prone." *Plast Reconstr Surg* 114:980–987, 2004.
146. Yaremchuk MJ: Making concave faces convex. *Aesthetic Plast Surg* 29:141–147, discussion 148, 2005.
147. Yaremchuk MJ: Indications, evaluation and planning. In Yaremchuk MJ, editor: *Atlas of facial implants*, Philadelphia, 2007, Saunders-Elsevier, pp 3–22.
148. Yaremchuk MJ, Chen YC: Enlarging the deficient mandible. *Aesthet Surg J* 27:539–550, 2007.
149. Yaremchuk MJ, Doumit G, Thomas MA: Alloplastic augmentation of the facial skeleton: An occasional adjunct or alternative to orthognathic surgery. *Plast Reconstr Surg* 127:2021, 2011.
150. Yousif NJ, Gosain AK, Sanger JR, et al: The nasolabial fold: A photogrammetric analysis. *Plast Reconstr Surg* 93:70–77, 1994.
151. Yousif NJ, Gosain AK, Matloub HS, et al: The nasolabial fold: An anatomic and histologic reappraisal. *Plast Reconstr Surg* 93:60, 1994.
152. Yousif NJ: Changes of the mid face with age. *Clin Plast Surg* 22:213, 1995.
153. Zadoo VP, Pessa JE: Biological arches and changes to the curvilinear form of the aging maxilla. *Plast Reconstr Surg* 106:460–466, 2000.

26

Obstructive Sleep Apnea: Evaluation and Treatment

JEFFREY C. POSNICK, DMD, MD

- Rationale for Treatment
- Diagnosis, Characterization, and Assessment of Obstructive Sleep Apnea Severity
- Obstructive Sleep Apnea during Childhood: Diagnosis and Management
- Managing the Airway during a Surgical Procedure in the Individual with Obstructive Sleep Apnea
- Treatment Options for Obstructive Sleep Apnea
- Conclusions

Researchers are increasingly finding that a lack of sleep is detrimental for our health. Sleepiness has been linked to increased rates of heart disease, obesity, stroke, and even certain cancers. The exact reasons for these effects are still largely unknown, but they give support to the theory that sleep is the time when our bodies naturally repair themselves on a cellular level.[150] Sleep—or the lack of it—is now thought to be a complex process that underpins everything from the ability to learn a new skill to how likely we are to find a novel solution to a problem.[243] It is also considered a vital part of happiness and one of the best forms of preventative medicine.[288]

Sleep disorders include conditions that are characterized by repeated pauses in breathing during sleep, which lead to the fragmentation of sleep and decreases in oxyhemoglobin saturation. The physiologic spectrum of sleep-disordered breathing ranges from partial airway collapse with increased upper airway resistance, which is manifested as loud snoring and episodes of hypopnea, to complete airway collapse and prolonged episodes of apnea. Obstructive sleep apnea (OSA) is clinically defined by frequent episodes of apnea and hypopnea as well as symptoms of functional impairment.[13,14,129,130]

According to the National Sleep Foundation, some form of snoring or OSA occurs in 90 million Americans.[245,246] Approximately 40% of individuals who are more than 40 years old snore, and half of them snore every night. Among habitually loud snorers, the incidence of OSA is at least 17% in men and 15% in women. The Foundation estimates that 16 million of the 20 million Americans with OSA remain undiagnosed. Affected individuals are often aware of snoring, daytime sleepiness, poor sleep patterns, and complaints from bed partners.[352] To the educated clinician, the diagnosis of OSA may seem clear, but the affected individuals and their families are often unaware.[9,106]

Young and colleagues carried out a study to determine the prevalence of sleep-disordered breathing.[393-395] With the use of a random sample of 602 employed men and women who were between the ages of 30 to 60 years old and who were studied via overnight polysomnography (PSG), these authors assessed the frequency of episodes of apnea and hypopnea per hour of sleep. The authors determined that this condition affected up to 9% of middle-aged women and 24% of middle-aged men (i.e., an apnea–hypopnea index [AHI] of 5 or more and daytime hypersomnolence).[185] Both male and female habitual snorers tended to have a higher AHI of 15 or more.

Although obesity is often given as the cause of OSA, evidence supports the idea that a significant percentage of individuals with OSA have an anatomic predisposition to airway collapse (i.e., retrusive jaws and obstructed nasal cavities). Clinical studies confirm that maxillary–mandibular–chin advancement (MMCA), which is often combined with intranasal procedures (i.e., septoplasty, inferior turbinate reduction, and nasal floor recontouring), is relatively safe and highly effective for the treatment of OSA when multiple sites of upper airway narrowing are present. Published studies consistently document that MMCA is the most effective surgical alternative to tracheostomy, with results that are comparable to the therapeutic efficacy of nasal continuous positive airway pressure (CPAP). This chapter will review several aspects of OSA, including its diagnosis, the characterization of its severity and the rationales for its various treatment options.

Rationale for Treatment

The two major rationales for the treatment of OSA are to address 1) pathophysiologic derangements and 2) behavioral derangements.[263]

The *pathophysiologic derangements* related to OSA are primarily cardiorespiratory in nature.* They include increased risks of myocardial infarction, stroke, and sudden death.[153] Sudden death may be caused by tachycardia and tachyarrhythmias (i.e., atrial fibrillation) as well as by bradyarrhythmias. The documented high prevalence of atherosclerotic coronary artery disease among patients with OSA is possibly related to endothelial damage, with platelet activation and the presence of inflammatory mediators also being involved.[153] When OSA is combined with systemic hypertension, an increased incidence of stroke has been reported.[138,140,392] A higher-than-average occurrence of idiopathic dilated cardiomyopathy and more frequent episodes of ST-segment depression have also been recognized.[118,125,153] The physiologic processes that are believed to be involved in OSA and that predispose patients to these risks include hypoxemia, negative intrathoracic pressure, and the disequilibrium of the autonomic nervous system.[42]

The *behavioral derangements* caused by OSA may be the result of excessive daytime sleepiness.[161,367] OSA is associated with nocturnal arousals during sleep that occur in response to airway collapse and that result in poor sleep. Excessive daytime sleepiness manifests as sleepiness or fatigue of such a degree that the individual lacks vigilance and does not function as well as he or she should during the day. Other symptoms typically include heavy snoring, known apnea, headaches, difficulty waking in the morning, fatigue, sleepiness after eating, memory loss, having difficultly with work interactions, stressed relationships and sexual performance complaints. There is a documented increased incidence of motor vehicle accidents and job-related injuries among individuals with OSA (e.g., individuals with OSA are 10 times more likely to die in an automobile accident).[26,85,103,292] OSA is also known to be a cause of difficulty with concentration at school or during work activities. Over time, the hypoxia that is caused by OSA is believed to result in changes in the neurons of the hippocampus and the right frontal cortex of the brain. Neuroimaging studies have revealed evidence of hippocampal atrophy in people who chronically suffer from OSA. Clinically, it is documented that a greater percentage of patients with OSA have problems with mentally manipulating nonverbal information and with executive processing.[100] Interestingly, there is not a direct correlation between these behavioral derangements and the severity of OSA as measured with the AHI or with specific levels of oxygen desaturation. It is important to note that there are multiple causes of excessive daytime sleepiness other than OSA, including volitional sleep deprivation, alcoholism, insomnia, and narcolepsy.

*References 10, 23, 25, 42, 118, 125, 138, 140, 153, 175, 182, 221, 228, 250, 263, 265, 266, 289, 330, 331, 333, 350

Diagnosis, Characterization, and Assessment of Obstructive Sleep Apnea Severity

Pathophysiology

OSA is characterized by symptoms that result from the recurrent sleep-associated collapse of the pharyngeal airway and that lead to hypoxemia, hypercapnia, and fluctuations in intrathoracic pressure caused by increased respiratory effort, with arousal from sleep required to reestablish airway patency. When awake, protective mechanisms increase the activity of the pharyngeal dilator muscles; however, during sleep, these mechanisms may fail and result in the collapse of the pharyngeal airway.[45,73,120,293,329,346,349,387] This is made worse by nighttime horizontal and supine sleep positioning.[244,256] Frequent locations of pharyngeal collapse include the velopharynx (behind the soft palate), the oropharynx (behind the tongue), or both.[240]

Schwab and colleagues have documented an increase in adipose hypertrophy within the floor of the mouth, the soft palate, the pharyngeal fat pads, and the lateral pharyngeal walls in many individuals with OSA.[324] In patients with obesity, there is a direct deposition of fat that causes submucosal adipose volume expansion within the pharyngeal walls (thus narrowing the lumen) and the floor of the mouth (thus causing elevation and retro positioning of the tongue), thereby diminishing the actual upper airway breathing space and, in many cases, explaining the greater extraluminal critical closing pressure documented in patients with OSA. Evidence supports the concept that many individuals with OSA have an anatomic predisposition to airway collapse as a result of retrusive jaws and obstructed nasal cavities. An individual's unique baseline skeletal (i.e., jaw) anatomy may have a dramatic effect on the size (i.e., the luminal diameter) of the upper airway. It is now also appreciated that the variability in upper airway breathing space from one person to the next is influenced by the subject's developmental or congenital maxillofacial skeletal morphology. Deficient maxillomandibular structural support adds to the environmental causes (i.e., weight gain) that thicken the pharyngeal soft tissues, thereby further narrowing the lumen and diminishing the patency afforded by pharyngeal dilator muscle contraction forces that work to maintain and open the airway. Genetics plays a lesser role in the observed volumes of the tongue, the soft palate, the adenoids, the turbinates, and the tonsillar tissues seen in individuals with OSA. These soft-tissue structures are more likely to be affected by acquired conditions such as weight gain, infections, or allergies. With obesity, there may also be alterations of pharyngeal muscle orientation and function. These factors combine to determine overall upper airway breathing space, pharyngeal muscle function, and, as a consequence, the occurrence of apnea events during sleep.

Schwartz and colleagues measured the critical closing pressures during sleep in individuals with OSA and

compared these values to those of controls (i.e., individuals without OSA).[327] Individuals with baseline anatomically "large-volume" airways are less dependent on pharyngeal dilator muscles to maintain airway patency than those with intrinsic "small-volume" anatomic airways. Schwartz and colleagues showed that control subjects (i.e., individuals without OSA) typically have critical closing airway values of less than 0 cm of water, whereas those with severe OSA have values of more than 8 cm of water. Multiple diagnostic modalities (e.g., cephalometric x-rays, computed tomography [CT] scans, acoustic reflection, magnetic resonance imaging) confirm that individuals with OSA generally have "smaller-volume" baseline awake airway lumens as compared with controls.[264]

According to Fogel and colleagues the pharyngeal muscles of primary importance fall into three groups[87]:

1. The muscles that influence the hyoid bone position (i.e., geniohyoid, sternohyoid)
2. The muscles of the tongue (i.e., genioglossus)
3. The muscles of the soft palate (i.e., tensor palatine and levator palatine)

The activity of these muscles is normally increased during inspiration to stiffen and expand (i.e., open) the upper airway. This counteracts the collapsing influences that would otherwise occur with inspiratory negative airway pressure. During expiration, the muscles normally substantially relax while positive pressure is flowing through the respiratory tree. The tensor palatine is an exception in that it must maintain a constant level of tone throughout the respiratory cycle to preserve a patent airway. Fogel and colleagues stated that the activity of the pharyngeal dilator muscles during wakefulness is tightly controlled to maintain pharyngeal patency.[87] In individuals with an anatomically "small-volume" airway, the activation of this negative-pressure reflex (i.e., feedback mechanism) to increase the function of the pharyngeal muscles must occur to prevent or limit apnea. This reflex has been shown to be diminished in apnea-prone individuals for both the genioglossus and the tensor palatine muscles. An individual's use of specific medications (e.g., alcohol, sedatives, narcotics) will also negatively influence the reflex arc, thereby causing the relaxation of the muscles and further diminishing airway patency.

At least until the age of 65 years, age is positively correlated with OSA severity as demonstrated by an increase in the AHI.[152] This increase is substantially greater in the presence of baseline maxillomandibular sagittal deficiency. While a person is awake, neuromuscular mechanisms and body position (i.e., standing or sitting up) can positively influence and compensate for the "small-volume" airway. However, the pharyngeal dilator muscles are less able to effectively contract during sleep, which results in the more frequent collapse of the anatomically small breathing space. The ability of the pharyngeal muscles to compensate for and prevent apnea events becomes progressively more difficult with age and in the presence of weight gain.

Frieberg and colleagues hypothesized that a progressive lesion in the afferent and/or efferent nerve pathways caused by repetitive "snoring trauma" may also contribute to the collapsibility and obstruction of the upper airway that is seen in patients with OSA.[89-91] Their study evaluated the hypothesis of the presence of a progressive neurogenic lesion in the pharyngeal muscles in patients with longstanding upper airway obstruction. Biopsies of palatopharyngeal muscle were obtained from patients (n = 21) with habitual snoring and degrees of upper airway obstruction and compared with those of non-snoring control subjects (n = 10). The degree of abnormality found was significantly increased in the study patients as compared with control subjects. The results of their research support the hypothesis of a progressive local neurogenic lesion that is caused by the "trauma of snoring" as a possible contributory factor in upper airway collapsibility.

Sleep electroencephalograms (EEGs) of patients with OSA will differ from those of individuals with upper airway resistance syndrome (UARS) and from those of control subjects.[117,128] The arousal response of individuals with OSA is delayed, despite the fact that increased respiratory effort is seen during the obstructive phase. This delayed arousal response has been confirmed by differences in the patterns of EEG findings. Guilleminault and colleagues hypothesized that a difference in sensory input may be the responsible factor for the divergent responses to the abnormal breathing patterns that may exist between individuals with UARS and those with OSA.[122] The authors set out to compare the results of a palatal mucosa two-point discrimination response in normal subjects (n = 15), those with OSA (n = 15), and those with UARS (n = 15). The subjects were matched for age, sex, and body mass index. The 45 subjects were submitted to a two-point discrimination study of the palatal mucosa during wakefulness. Individuals with OSA had a clear impairment of their palatal mucosa sensory input with a significant decrement in two-point discrimination. Interestingly, the subjects with UARS and the normal control subjects responded similarly to each other. The responses seen in individuals with UARS indicate that those individuals are more capable of transmitting sensory inputs than individuals with OSA. This may be at least one factor that explains the difference in arousal response that is been documented in patients with UARS as compared with those with OSA.[122]

For the individual with OSA, as apnea events become more frequent, hypoxemic and hypercarbic episodes require increased respiratory effort to ultimately achieve arousal from sleep. With awakening from the apnea event, there is a reestablishment of airway patency, and ventilation proceeds. The individual then returns to sleep, and the cycle repeats itself. This repetitive pattern of sleep disruption not only affects the quality of the individual's life (i.e., behavior derangements), but it also affects their longevity (i.e., pathophysiologic derangements).

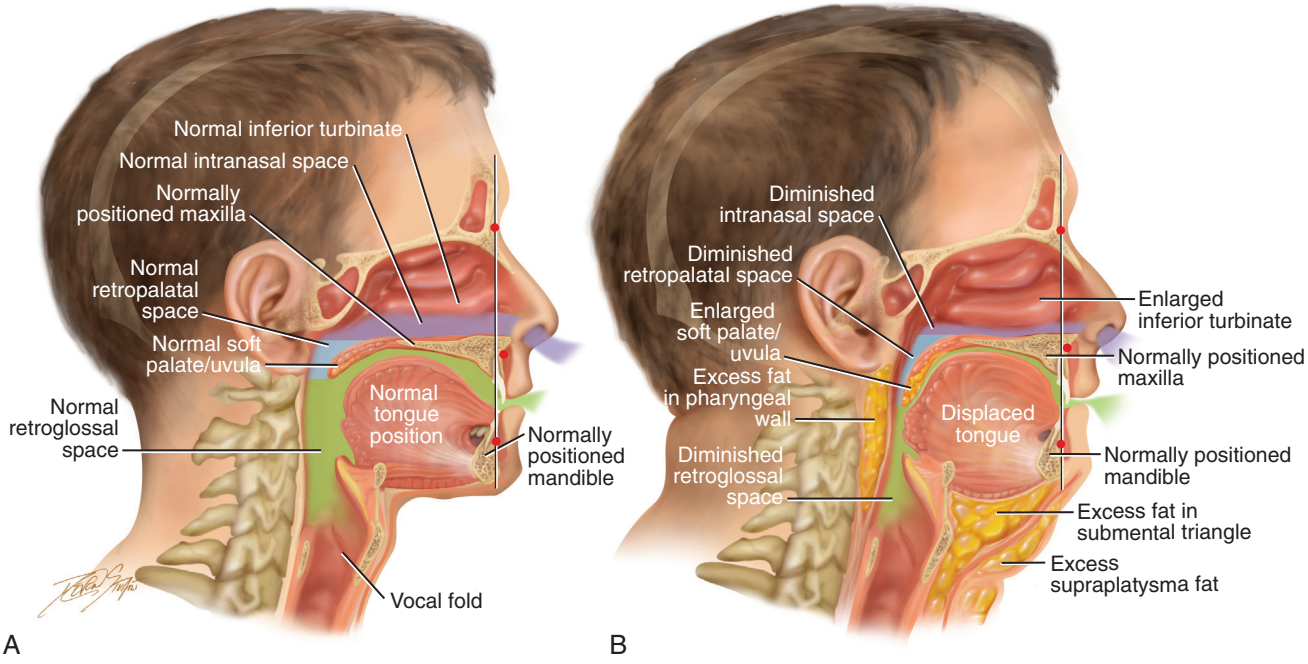
The basic anatomic factors that reduce the size of the upper airway include the following:

1. Regional obesity (i.e., increased volume of the lateral pharyngeal fat pads, the soft palate, and the floor of the mouth
2. Baseline (inherent) abnormalities of the maxillomandibular skeletal structures (i.e., the maxilla, the mandible, and the nasal septum)
3. Volumetric enlargement of upper airway soft-tissue visceral structures (e.g., tongue, soft palate, tonsils, adenoids, inferior turbinates) as a result of congenital anomalies, allergies, infections, or tumors.

The term *pharynx* is used to describe the anatomic region that extends from the esophageal entrance up to the back of the nose. It is further subdivided according to proximity to the nose, mouth, and throat down to the larynx. Together, these pharyngeal structures are involved in critical physiologic functions, including breathing, swallowing, vocalization/speech, and mastication. Each of these functions is potentially influenced by any one of the upper airway anatomic components. The upper airway functions as a continuous cohesive organ; it is commonly defined as the soft-tissue region that is bounded by the nasopharynx superiorly, the epiglottis inferiorly, the maxillomandibular complex anteriorly, and the spinal column posteriorly. The upper airway is somewhat artificially subdivided into the following regions:

1. *The intranasal region.* This portion of the airway extends from and includes the external nasal valves, the internal nasal valves, the turbinates, the nasal septum, and the adenoids.
2. *The retropalatal region.* This portion of the airway is primarily affected by the position of the maxilla (i.e., the hard palate), the size and position of the soft palate, the tonsils, and the parapharyngeal fat pads.
3. *The retroglossal region.* This portion of the airway is primarily affected by the position of the mandible, the size and position of the tongue, the fullness of the soft tissues in the floor of the mouth, the parapharyngeal fat pads, and the tonsils.

An increased body mass index (BMI), which is also known as *obesity,* is a known risk factor for OSA in children, adolescents, and adults (Fig. 26-1).[21,88,241,255,337] Peppard and



• **Figure 26-1** **A,** Midline sagittal cross-section head and neck illustration of a 20-year-old man with a normal body mass index to demonstrate upper airway space and associated maxillofacial anatomy. This individual has a normal upper facial skeleton (cranial vault, orbits, and zygomas), a normal lower facial skeleton (maxilla and mandible), and normal soft tissues (soft palate, tongue, tonsils, and adenoids). The intranasal cavity, which includes the turbinates and the septum, is also normal. **B,** The same individual when he is 50 years old with an elevated body mass index. The upper and lower facial skeleton remain unchanged with age. The adipose cells, which are scattered throughout and within the upper airway soft tissues, have now increased in volume. This includes the retropharyngeal and the lateral pharyngeal tissues, the soft palate, and the floor of the mouth. As a result, the soft palate is long, thick, and in close proximity to the posterior and lateral pharyngeal walls, thereby diminishing the retropalatal space. As a result of increased adipose volume in the floor of the mouth, the tongue has been displaced superiorly and posteriorly, thus decreasing the retroglossal airway space. As a result of chronic allergies, the patient's turbinates have enlarged to cause increased nasal airway resistance (i.e., diminished intranasal airway space). There is also excess adipose volume in both the submental triangle and the supraplatysmal locations, thereby resulting in a full neck and a "double chin" appearance. In review, when the patient was 20 years old, he had normal upper airway space, including the intranasal site; the retropalatal site; and the retroglossal sites. When he was 50 years old, the upper airway space is considerably smaller, including the intranasal space, the retropalatal space, and the retroglossal space. The patient depicted here now has documented obstructive sleep apnea.

colleagues outlined the cardiovascular risks associated with OSA for weight gain and weight loss.[269,268] Excessive body weight was found to be positively associated with OSA,[262] and the stability of the individual's weight was also found to be an important factor. A 10% weight gain predicted a 32% increase in the AHI.[319] The same 10% gain in weight also predicted a six-fold increase in the odds of moderate to severe OSA developing. Interestingly, a weight loss of 10% predicted a 26% decrease in the AHI.[169] Unfortunately, studies consistently document that weight loss programs in patients with OSA frequently fail miserably. Any weight loss that the individual is able to initially achieve tends to be temporary.[167]

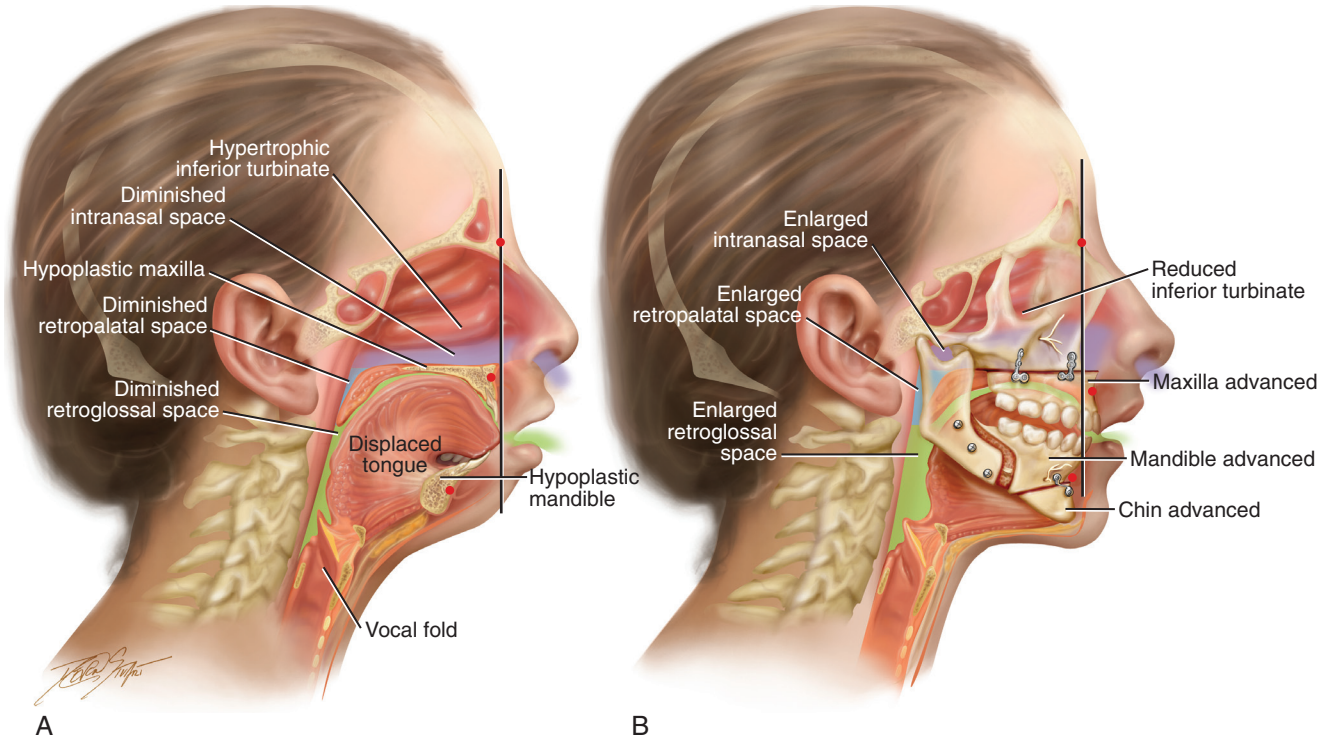
When an individual's BMI is elevated, the volume of fat in the pharynx and the floor of the mouth also increases. It is estimated that more than 66% of individuals with OSA are obese. On the basis of the latest World Health Organization statistics, it is estimated that there are more than 1.6 billion overweight adults (from a total population of 6.5 billion) and at least 400 million obese individuals (BMI ≥ 25 kg/m^2) on Earth.[72] Obesity has both a genetic and an environmental basis.[37,63,102,165,237] A review of population studies suggests that heredity accounts for at least 40% of the variance in BMI.[219,286] Studies have demonstrated a strong relationship between the BMIs of biologic parents and their children. Interestingly, no relationship between the BMI of adoptive parents and their adopted children has been documented. The best indirect indicator of excessive fat accumulation in the pharyngeal region and the floor of the mouth is a person's BMI.

Studies have also documented that relatives of individuals with OSA demonstrate a higher incidence of hypoplastic maxillae and mandibles, longer soft palates, and wider uvulas as compared with matched controls.[219,326] This also speaks to the hereditary component of a small pharyngeal airway space and to an individual's genetic predisposition to OSA.[295,388]

There are specific syndromes and conditions that result in severe maxillomandibular skeleton dysmorphology and that also negatively affect the upper airway (e.g., Crouzon syndrome, Apert syndrome, Treacher Collins syndrome, Pierre Robin sequence).[17,38,270,271,287,341,354,390] It is documented that more than 50% of individuals with Down syndrome suffer from OSA.[66] Interestingly, a similar percentage of individuals with Down syndrome are known to have a baseline intrinsic developmental jaw deformity that is characterized primarily by maxillary deficiency. The surgical correction of the maxillofacial deformities associated with these syndromes have been documented to enlarge the upper airway space and to improve and often resolve breathing difficulties, including OSA (see Chapters 27, 28, 30 and 31). The surgical management of velopharyngeal insufficiency (i.e., superiorly based pharyngeal flap or sphincteroplasty) in individuals with a repaired cleft palate is also known to result in OSA in some cases.[4,39,157,176,187,205,257,258,339,340,343,373,389,396] Interestingly, the non-syndromal developmental dentofacial deformities (e.g., long face growth pattern, short face growth pattern, primary mandibular deficiency, maxillary deficiency with relative mandibular excess, maxillary hypoplasia associated with repaired cleft palate), which affect at least 5% of the general population, also negatively affect the upper airway.[212] Unfortunately, these more common patterns of jaw disharmony frequently go unrecognized as significant etiologic factors for OSA (Figs. 26-2 and 26-3). It is incumbent on the otolaryngologist, the pulmonologist or sleep specialist, the pediatrician or internist, the orthognathic or maxillofacial surgeon, and the general dentist or orthodontist to understand the fundamental relationship between developmental jaw deformities and upper airway obstruction in each patient who is evaluated.

Not infrequently, the individual with "mandibular prognathism" has undergone a lower jaw set-back treatment to improve the occlusion. Historically, such procedures have been preferred by many clinicians for the treatment of skeletal Class III patterns. However, it is now known that this approach is associated with a risk to the airway size and shape.[6,18,41,48,67,68,74,78,84,113,115,171,172,174,314,316,370,386] Demetriades and colleagues confirmed the clinical importance of considering the airway when formulating a treatment plan for the correction of so called "mandibular prognathism."[69] They described 26 patients who presented with developmental dentofacial deformities that were characterized by maxillary deficiencies in combination with relative mandibular excesses. Each patient underwent mandibular ramus osteotomies with at least some set-back. The patients were divided into two groups. The Group 1 patients (n = 13) underwent mandibular setback of at least 5 mm without simultaneous Le Fort I osteotomies. The Group 2 patients (n = 13) also underwent a degree of mandibular set-back, but in conjunction with Le Fort I advancement. All study patients (Groups 1 and 2) underwent preoperative and postoperative lateral cephalometric radiography and portable PSG. The preoperative and postoperative radiographs were compared, and any changes that occurred in the position of the hyoid bone, the distance from the posterior nasal spine to the palate, the retrolingual space, and the hypopharyngeal space were measured. The authors confirmed that mandibular retropositioning of at least 5 mm can significantly decrease the posterior airway space and cause mild to moderate OSA. The study also suggested that, when a limited mandibular set-back is carried out in combination with maxillary Le Fort I advancement, significant negative effects on the airway are likely to be prevented.

In summary, anatomic upper airway risk factors for OSA include the following: 1) regional obesity (i.e., increased upper airway submucosal adipose tissue deposition); 2) retrusive maxillomandibular skeleton (e.g., hypoplasia of the maxilla or mandible); 3) alterations in the intranasal cavity (e.g., septal deviations, hypertrophic inferior turbinates, tight nasal aperture, elevated nasal floor, adenoid hypertrophy); 4) enlargement of other critical upper-airway soft-tissue visceral structures (e.g., soft palate, tonsils,



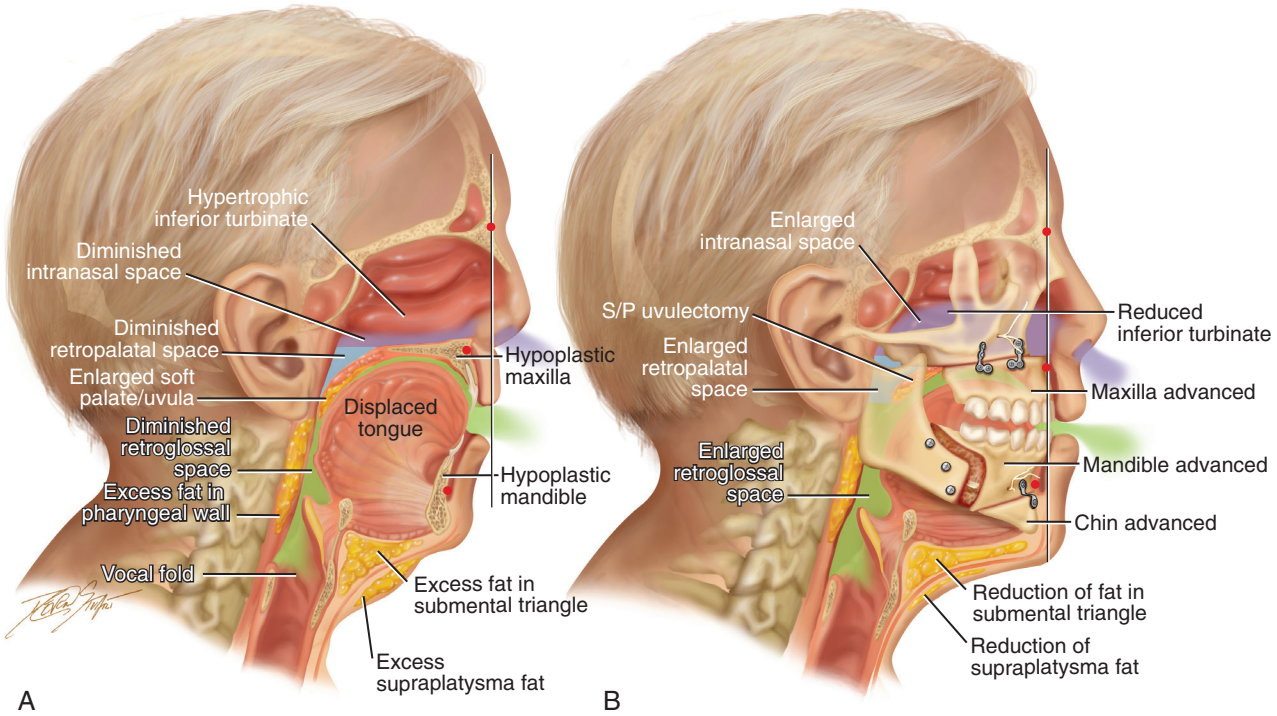
• **Figure 26-2** Midline sagittal cross-sectional head and neck illustrations of a 16-year-old girl with primary mandibular deficiency, maxillary arch constriction, and Class II excess overjet malocclusion (see Fig. 19-4). The illustrations demonstrate the upper airway space and the associated maxillofacial anatomy before and after jaw and intranasal reconstruction. **A,** Illustration when the patient was 16 years old, before intervention. The inferior turbinates are hypertrophic as a result of chronic allergies, and the nasal septum is deviated. These findings result in a diminished intranasal airway space. As part of the developmental jaw deformity, the maxilla is retropositioned, and there is a narrow arch width. Therefore, the soft palate is posterior, which results in diminished retropalatal space. The mandible is horizontally (sagittally) deficient, with a posterior displaced tongue. As a result, there is diminished retroglossal space. The tonsils, the adenoids, the tongue, and the soft palate are displaced but otherwise normal. Although the patient is young and has a normal body mass index, the intranasal and jaw deformities result in diminished upper airway space. The patient has difficulty breathing through the nose while she is awake, and is with heavy snoring at night. Obstructive sleep apnea has been documented. **B,** The patient agreed to a comprehensive orthodontic and surgical approach. Lower bicuspid extractions allowed for the relief of dental compensation. The patient's surgery included maxillary Le Fort I osteotomy in segments (arch expansion and horizontal advancement); bilateral sagittal split ramus osteotomies (horizontal advancement); osseous genioplasty (horizontal advancement); and septoplasty and inferior turbinate reduction. The postoperative illustration indicates maxillofacial changes that resulted from the reconstruction. Now there is an expanded intranasal, retropalatal, and retroglossal airway space. There is improved nasal breathing during the day and reduced snoring and apnea events at night.

tongue); and 5) inadequate pharyngeal muscular tone (i.e., hereditary and environmental factors).[320,332,348,378] Each of these head and neck structural aspects predispose individuals to OSA by causing upper-airway narrowing during wakefulness and airway collapse during sleep. When these aspects occur in combination, the risk is greatly exacerbated. There are also specific medical conditions (e.g., myasthenia gravis, Parkinson disease, amyotrophic lateral sclerosis) as well as certain medications and drugs (e.g. alcohol, narcotics, sedatives, muscle relaxants) that are known to negatively affect the neuromotor tone of the chest wall and the pharyngeal musculature and thus predispose an individual to apnea.

Basic Clinical Evaluation

A thorough medical and sleep history should be taken and a physical examination completed. The following information should be especially considered for an individual with known OSA:

1. The clinician should determine at what specific maxillofacial skeletal locations (e.g., nasal septum, turbinates, maxilla, mandible), if any, there are negative effects occurring in the upper airway during sleep or while awake.
2. If the patient also has a high BMI (i.e., >25 kg/m^2), the clinician should determine at what regional soft-tissue locations (e.g., nasopharyngeal, palatopharyngeal, glossopharyngeal), if any, there is excessive submucosal adipose and other soft-tissue volume that may have a negative impact on the airway.
3. The clinician should determine if there are baseline physiologic dysfunctions of the lungs and chest walls (e.g., chronic obstructive pulmonary disease) that negatively affect the airway.



• **Figure 26-3** Midline sagittal cross-sectional head and neck illustrations of a 59-year-old man to demonstrate upper airway space and associated maxillofacial anatomy before and after jaw, intranasal, and facial surgery carried out primarily to open the airway (see Fig. 26-6). This patient has documented severe obstructive sleep apnea, including a respiratory disturbance index of 51 events/hour with desaturations reaching as low as 82%. The patient has an elevated body mass index and a short face growth pattern that is characterized by maxillomandibular vertical and horizontal deficiency. At the time of presentation to this surgeon, he had already undergone "Phase I" procedures including uvulopalatopharyngoplasty, septoplasty, and inferior turbinate reduction without relief of obstructive sleep apnea. **A,** Illustration of the patient's condition before surgery demonstrates hypertrophic inferior turbinates and septal deviation that result in a diminished intranasal space. The maxilla is vertically short and horizontally deficient. The soft palate is long and thick as a result of excess fat accumulation. For all of these reasons, a diminished retropalatal airway space is present. The mandible is horizontally deficient, and the tongue is elevated as a result of excess fat in the floor of the mouth. The lateral and retropharyngeal walls also show excess fat accumulation. These findings account for the diminished retroglossal airway space. **B,** After the patient was referred to this surgeon, he underwent simultaneous procedures that included maxillary Le Fort I osteotomy (horizontal advancement of 10 mm and vertical lengthening of 5 mm) with interpositional graft; bilateral sagittal split osteotomies of the mandible (horizontal advancement of 12 mm); osseous genioplasty (horizontal advancement); redo septoplasty; and anterior neck soft-tissue procedures (cervical flap elevation, fat removal, and platysma muscle plication). The illustration after maxillary–mandibular–chin advancement demonstrates the enlarged upper airway space, including the intranasal space, the retropalatal space, and the retroglossal space. A 3-month postoperative attended polysomnogram confirms a respiratory disturbance index of less than 5 events/hour.

4. The clinician should consider the effects of baseline neuromotor disorders (e.g., Parkinson disease) and currently used medications (including nonprescription drugs and alcohol) that may negatively affect the airway.

It is also recommended that a subjective questionnaire be used to assess for OSA. The Epworth Sleepiness Scale (ESS) is considered the reference standard.[86,163,164] The survey assesses the respondent's propensity for sleep in eight different situations. Although the survey is not perfect, it does provide useful information about the patient's waking sleepiness.

An airway analysis that is based on CT scan data may also be a useful tool to assess upper airway space. A recent study used three-dimensional computer analysis from cone-beam CT to present normative upper airway size reference data. The collected data confirm that, in humans, the airway size and length increases until the age of 20 years, at which time there is a variable period of stability. After this stable period, the airway at first decreases slowly in size; then, after the age of 50 years, it decreases more rapidly.[322] Li and colleagues demonstrated a correlation between the airway area and the likelihood of OSA.[197] There is a high probability of severe OSA with an airway area of less than 52 mm^2; an intermediate probability if the airway is between 52 mm^2 and 110 mm^2; and a low probability if the airway area is more than 110 mm^2.[190] Cillo and colleagues attempted to correlate specific cephalometric measurements with the AHI in a consecutive series of patients (N = 101) who were documented to have OSA. Unfortunately, no one skeletal or soft-tissue parameter could be linked to OSA.[51] Three-dimensional CT airway analysis has documented quantifiable changes in patients' oropharynges after specific surgical

interventions. This type of imaging may also be used to evaluate changes in the airway after specific medical interventions (e.g., weight loss).[75]

Susarla and colleagues used a case–control study that was designed to analyze the value of the cephalometric upper airway length (UAL) measurement as an indirect predictor of the presence and severity of OSA.[360,361] The study group included adults (n = 96) with PSG-documented severe OSA and a control group of adults (n = 56) with skeletal Class II malocclusion without known OSA. Although significant attention has been devoted to describing and measuring upper airway width, little attention has been devoted to measuring UAL. The theory behind this study is that an important component of the airway resistance is the airway length (i.e., the vertical dimension), which is known to be directly correlated with airway resistance per Poiseuille's law: resistance equals $8\ \eta L/\pi r_4$ (L = length). The results of the study suggest that the UAL (i.e., the distance between the posterior hard palate and the most superior aspect of the hyoid bone on a line parallel to the long access of the airway) is predictive of the presence of OSA in both men and women. The study results indicate that a cephalometric UAL of 72 mm or more in men and 62 mm in women was diagnostic for OSA with high sensitivity and specificity. It would appear that both a narrow posterior airway space and a long UAL predispose individuals to upper airway obstruction and that both conditions are associated with increased odds of OSA.

A reasonable predictor of OSA is a clinical triad of symptoms that includes loud snoring, apnea witnessed by a bed partner, and excessive daytime sleepiness. When the sleep and daytime history is combined with attended PSG, the patient's clinical head and neck examination (including instrumentation), and a focused radiographic analysis, then an overall picture of the upper airway anatomy and function can be seen.*

Attended Polysomnography

Overnight PSG conducted in a sleep laboratory remains the reference standard for the diagnosis of central sleep apnea or OSA.[92,111,155,180,365] PSG requires the use of several monitoring parameters, including EEG, electrooculography, electromyography, electrocardiography, the microphone measurement of snoring, pulse oximetry, respiratory inductance plethysmography, airflow measurement, and the recording of the body position. PSG primarily evaluates sleep-disordered breathing, sleep architecture, and oxygen desaturations. During PSG, episodes of apnea and hypopnea are determined from a reduction in airflow in combination with oxygen desaturation during a 4- to 8-hour time frame.

*References 1, 7, 19, 20, 22, 28, 33, 61, 64, 70, 101, 135-137, 146, 151, 156, 168, 190, 206, 210-212, 260, 281, 309, 315, 323, 338, 359, 362, 364

It is recommended that all sleep studies for a surgical patient be attended overnight PSG. Parameters reviewed after attended PSG should include the following:

- Sleep stages (both rapid eye movement and non–rapid eye movement sleep), including the percentage of sleep in each.
- Apnea index (AI)
- Hypopnea index
- Awake oxygen saturation, including the lowest levels and the stratifications of the percentage of time below specific percentiles (i.e., 90%, 80%, and 70%)
- Heart rate fluctuations (e.g., brachyarrhythmia, tachyarrhythmia)
- Periodic leg movements

To be most accurate as a diagnostic tool, the PSG study should be current (i.e., within the previous 6 to 12 months) and, if possible, not a split-night study. Split-night studies apply CPAP for the second half of the night and may underestimate the severity of OSA. An alternative to an attended sleep study is a home sleep study. Home sleep studies lack all of the monitoring parameters of an attended overnight PSG. However, advantages of home sleep studies include reduced costs and the ability to perform the sleep study in the patient's native sleeping environment. Disadvantages include the gathering of fewer sleep parameters and possibly reduced reliability.

Assessment of Apnea Severity

By convention, the severity of OSA is classically determined by the AHI. The term *obstructive apnea* refers to apnea despite continued respiratory effort. *Apnea* is defined as the cessation of airflow for at least 10 seconds in an adult or for the equivalent of two breaths in a child. *Hypopnea* is defined as 1) a substantial reduction in airflow (i.e., >50%); or 2) a moderate reduction in airflow (i.e., <50%) with oxygen desaturation of more than 3%; or 3) EEG evidence of arousal in combination with oxygen desaturation that lasts for at least 10 seconds in an adult or for the equivalent of two breaths in a child. The AHI is a count of the number of apneas and hypopneas per hour of sleep. The respiratory disturbance index (RDI) is also used to describe OSA. The RDI considers the number of apneas, hypopneas, and respiratory-effort–related arousals per hour of sleep. Respiratory-effort–related arousals are EEG arousals that are not associated with clear-cut apnea or hypopnea but rather with slight changes in airflow or respiratory effort.

In adults, an AHI of 5 to 15 is regarded as mild OSA, an AHI of 15 to 30 is considered moderate OSA, and an AHI of 30 or more is severe OSA. (OSA during childhood is discussed later in this chapter.) Interestingly, the reliability of data procured during the sleep study and its interpretation by sleep specialists has not been verified through intra-observational or inter-observational studies.

Characterization of Apnea

Central sleep apnea is characterized by a lack of drive to breathe during sleep that results in insufficient or absent ventilation with compromised gas exchange. This may occur as a result of a number of known neuromuscular disorders, or it may not have a specific cause. OSA occurs when ongoing respiratory efforts are observed during the cessation of airflow within the upper airway. Central sleep apnea, like OSA, is associated with frequent nighttime awakening; excessive daytime sleepiness; and an increased risk of adverse cardiovascular outcomes. Central sleep apnea as classically defined is relatively uncommon (i.e., it is seen in <5% of patients referred to a sleep clinic).

Interestingly, it is recognized that 10% to 20% of individuals with known OSA will fail to respond to successful anatomic surgical expansion of the maxillomandibular complex. Another difficult to explain subgroup of individuals with documented OSA is that group with apparent normal upper airway morphology (i.e., no significant narrowing). Complex sleep apnea is a newly recognized category in the spectrum of sleep apnea syndromes that helps to explain these outliers. According to Susarla and colleagues, complex sleep apnea often presents with morphologic features that are typical of obstructive disease but that are combined with PSG evidence of strongly chemoreflex-modulated sleep breathing.[360] Complex sleep apnea is distinguished from other forms of OSA by the following:

- Non–rapid eye movement sleep dominance of obstructive disease;
- Oscillations of respiration with or without flow limitation; and
- Induction of central apneas or periodic breathing with the application of positive airway pressure therapy.

Susarla and colleagues believe that a lack of resolution of OSA after successful maxillomandibular advancement surgery may occur if a ventilation control disturbance either remains or is amplified.[360] These individuals with chemoreflex-sensitive apnea can be identified, because they are extremely prone to changes in their carbon dioxide levels. It is important to make an accurate diagnosis, because these patients can be expected to require further management of the central component to their apnea. Helpful treatment for this unique subgroup may include the use of nasal oxygen, pharmacologic agents, or positive-pressure–based adaptive ventilation. Affected individuals require continued reassessment and management by a knowledgeable clinical sleep specialist.

Sites of Functional Obstruction

Studies that attempt to localize the site of upper airway obstruction in an individual with moderate to severe OSA have shown that there is rarely a single anatomic site of blockage (see Figs. 26-1 through 26-3). More commonly, multiple sites of upper airway obstruction occur during episodes of hypopnea and apnea. As stated, the potential upper airway sites of obstruction are somewhat artificially grouped as 1) intranasal; 2) retropalatal; and (3) retroglossal. In addition to taking a history and a physical examination, diagnostic studies to further clarify the anatomic levels of obstruction include the Müller maneuver, which is observed during nasoendoscopic instrumentation; lateral cephalometric radiographs taken during end-tidal volume measurement with the Müller maneuver; helical CT; and magnetic resonance imaging.

Intranasal Site

When a history of chronic obstructive nasal breathing is combined with an intranasal clinical examination, instrumentation (e.g., speculum, endoscope), and radiography (e.g., sinus CT scan), the causes of the intranasal obstruction are generally identified. A thickened, buckled, deviated septum (bone and cartilage), enlarged (hypertrophic) inferior turbinates, an elevated nasal floor, and narrow lateral walls (tight nasal aperture) are frequent causes (see Chapter 10). Occasionally, crooked nasal bones, stenotic external nasal valves (after cleft lip repair), or collapsing internal nasal valves (previous nasal surgery) may also be responsible. Enlarged adenoids or nasal polyps may also be causative factors.

Retropalatal Site

Maxillary hypoplasia either from a known syndrome (e.g., syndromal craniosynostosis, Treacher Collins syndrome, repaired cleft palate) or from a more common developmental dentofacial deformity (e.g., short face growth pattern, long face growth pattern) may be responsible. Enlarged tonsils, excessive lateral parapharyngeal fat pads, and an enlarged floppy soft palate may also be factors.

Retroglossal Site

A retrusive mandible is a frequent component of developmental jaw deformities. As a result of mandibular retrognathia and microgenia, the tongue is retropositioned and elevated. Only rarely is the tongue itself enlarged; rather, it rests in a retro and elevated position as a result of the retrusive mandible. Occasionally an enlarged lingual tonsil will cause retroglossal obstruction. Excess fat deposition in the floor of the mouth will also elevate and retroposition the tongue. Enlarged tonsils and lateral parapharyngeal fat pads may also be contributing factors.

Obstructive Sleep Apnea during Childhood: Diagnosis and Management

"The [American Academy of Pediatrics] guidelines contain the following recommendations for the diagnosis of OSA:

(1) all children should be screened for snoring; (2) complex high-risk patients should be referred to a specialist; (3) patients with cardiorespiratory failure cannot await elective evaluation; (4) diagnostic evaluation is useful in discriminating between primary snoring and OSA, the reference standard being polysomnography; (5) adenotonsillectomy is the first line of treatment for most children, and continuous positive airway pressure is an option for those who are not candidates for surgery or do not respond to surgery; (6) high-risk patients should be monitored as inpatients postoperatively; and (7) patients should be reevaluated postoperatively to determine whether additional treatment is required."[12,57,328]

"This clinical practice guideline is not intended as a sole source of guidance in the evaluation of children with OSA.[15,16,35,46,50,108,110,127,170,216,223,235,296] Rather, it is designed to assist primary care clinicians by providing a framework for diagnostic decision making. It is not intended to replace clinical judgment or to establish a protocol for all children with this condition and may not provide the only appropriate approach to this problem."[223]

Manifestations in Children

Snoring and difficulty breathing during sleep are the most common complaints reported by parents of children with OSA.[54,249,400] However, in contrast with reports of OSA in adults, excessive daytime somnolence is believed to be less common in children, with only 7% of 10% of pediatric patients with OSA experiencing this symptom. Neurocognitive deficits and behavioral manifestations are more frequent in children and may be the result of the chronic exposure to intermittent hypoxemia and sleep disruption caused by sleep fragmentation. Children with OSA are more likely to present with symptoms of hyperactivity, inattentiveness, poor academic performance, and behavioral problems.[30,49,60,112,152,166,229,231,253,254,312] Such symptoms are often attributed to attention-deficit/hyperactivity disorder; treatments prescribed for this disorder (e.g., pharmacologic therapy with stimulants) can actually worsen sleep symptoms.

Diagnostic Criteria in Children

Diagnostic criteria for OSA in adults are typically the product of consensus and often include an AHI of 5 or more as documented by nocturnal PSG as well as evidence of disturbed or unrefreshing sleep, daytime sleepiness, or other daytime symptoms.[313,382] The rationale for specific AHI diagnostic criteria in children as compared with adults suffers from less available data and heterogeneity across studies. Part of the problem is that few studies have been performed to link specified levels of pediatric OSA with adverse outcomes. At present, an AHI or RDI of as low as 1 event/hour is used to identify children with OSA. Pediatric criteria for scoring AHI or RDI are used until the age of 13 years as a matter of routine. According to the most recent American Sleep Disorder Association guidelines, either the pediatric or adult criteria for OSA may be used for adolescent patients who are between 13 and 18 years old at the discretion of the scoring sleep specialist.[12-16] It is also unclear how many events per hour should be considered significant in the adolescent patient as compared with pediatric and adult patients.[5]

Treatment Considerations in Children

Adenotonsillectomy (T&A) is generally considered to be the standard first-line treatment for a child with OSA who does not have complex medical conditions that would make surgery an unacceptable risk.[34,121,124,183,188,220,224,230,232-234,298,317,344,351,355,366,384] A child with OSA should not undergo T&A without receiving a thorough evaluation of the entire upper airway to confirm the actual need for an operation. Nevertheless, the overall cure rate of T&A during childhood is believed to be as high as 82.9%. Studies indicate that, for children with documented OSA and a preoperative RDI of less than 19 events/hour, a T&A will frequently reduce the RDI to less than 5 events/hour. Alternatively, if the preoperative RDI is greater than 19 events/hour, then the T&A has a low probability of reducing the RDI to less than 5 events/hour.[355] Other anatomic causes for the child's OSA should be considered (e.g., maxillomandibular hypoplasia).

A tight nasal inlet is another common cause of upper airway obstruction that may be first recognized during childhood (see Chapter 10). Clinical studies have documented that maxillary arch expansion will improve a tight nasal inlet and thereby decrease nasal airway resistance. Maxillary arch expansion, which is accomplished with the use of a rapid palatal expansion appliance, is commonly carried out during childhood with little morbidity.[8,27,36,79,399] Unfortunately, rapid palatal expansion will only address the one site of obstruction and in a limited way. No studies have confirmed that this approach alone will be effective for OSA. For children in whom T&A or rapid palatal expansion is required but does not lead to the resolution of OSA, evaluation to confirm the additional sites of obstruction should follow and appropriate interventions considered. The option of the administration of CPAP should be entertained, but the overall long-term adherence rate for CPAP is unknown and felt to be problematic in this population. Skeletal reconstruction is likely to be beneficial in a child with a craniofacial anomaly that is known to obstruct the upper airway and in whom OSA has been documented.[17,38,53,270,271,287,341,354,390] For example, maxillary advancement in a child with either Apert or Crouzon syndrome and documented OSA has proven value (see Chapter 30). When a small lower jaw with a retro positioned tongue (i.e., some children with Treacher Collins Syndrome or Pierre Robin sequence) is the cause of OSA, then mandibular advancement can also be helpful (see Chapter 19). Developmental jaw deformities (i.e., long face growth pattern, short face growth pattern, and isolated mandibular

deficiency) occur frequently in the general population, but they often go unrecognized as causes of OSA in children and teenagers (see Chapters 19, 21, and 23). After a developmental jaw deformity has been recognized in a child, OSA should be ruled out.[126,383] Referral to an orthognathic surgeon familiar with OSA is recommended if the PSG is positive (Figs. 26-4 and 26-5).

Managing the Airway during a Surgical Procedure in the Individual with Obstructive Sleep Apnea

A question has been raised regarding the risk of intubation in a morbidly obese individual with a baseline diagnosis of severe OSA. Neligan and colleagues completed a prospective study to assess whether severity of OSA, neck circumference, and BMI were risk factors for difficult intubation in morbidly obese patients.[248] Each study patient (n = 180) underwent preoperative PSG, Mallampati scoring, thyromental distance measurement, mouth opening assessment, and neck circumference measurement. Each study patient was scheduled to undergo a surgical procedure that required orotracheal intubation. A systematic approach to the intubation was developed by the authors that included the placement of the individual into the "ramp" position to facilitate intubation by aligning the three axes: the pharyngeal axis, the laryngeal axis, and the tracheal axis. The ramp position was achieved by placing padding below the individual's back to elevate the head, upper body, and shoulders above the level of the chest. This was judged by positioning the external auditory meatus at the same horizontal level as the sternal notch. Of the 180 patients enrolled in the study, 124 were documented by PSG to have severe OSA (mean AHI, 31.3 events/hour). The median Mallampati score was 2; the mean neck circumference was 43.9 cm; and the mean Cormack and Lehane grade was 1. The incidence of anticipated difficult laryngoscopy, which was defined as a Cormack and Lehane grade of 3 or 4, was 8.3%. Of the study patients with documented OSA (n = 124), only six required three or more intubation attempts, and only 3.3% were judged as being "difficult to intubate." A Mallampati score of 3 or 4 predicted difficult intubation, and a mean neck circumference of 43.9 predicted difficulty with laryngoscopy. The study showed no direct relationship between the diagnosis or severity of OSA and the difficulty of laryngoscopy or intubation. There was no relationship between neck circumference or BMI and difficult intubation. The authors believe that their successful management of intubation requirements in this morbidly obese group related to their meticulous approach to airway management and their technique of using the "ramp" position, which aligns the airway axes and facilitates intubation. Some anesthesiologists prefer to use either a GlideScope (Verathon Medical, Bothell, Wash) or an awake fiber-optic nasotracheal laryngoscopy technique when faced with a morbidly obese individual who requires intubation (see Chapter 11).

Treatment Options for Obstructive Sleep Apnea

Recognizing that a significant subgroup of adults who have been diagnosed with OSA have a chronic illness is essential when considering treatment options. In general, obese adults with OSA should be thought of in a similar way to those with diabetes or hypertension. For these individuals, the total elimination of the disease is not generally feasible.[63,147,169,344] The goal of any treatment modality in this situation is to improve or control the symptoms and the health risks by reducing the severity. A clear exception to this way of thinking is in the individual with a small-lumen upper airway that is primarily the result of retrognathic jaws. For these individuals, who make up approximately one third of patients with OSA, the correction of the jaw deficiency is expected to be successful for most.[29,44,82,144,149,186,197,283,285,311,312]

A review of the literature that addresses the management of OSA confirms that a plethora of publications discuss a spectrum of medical and surgical treatment options.[181,207] More than a dozen "non-surgical" treatment options are mentioned, including palatal, tongue, and jaw training exercises; controlled sleep positioning; spray medications to reduce soft-tissue edema or to improve muscle tone; the use of a spectrum of oral appliances that have been designed to protrude the lower jaw; and the use of CPAP delivered through a variety of occlusive devices over the mouth and nose. The majority of these treatment recommendations (with the exception of CPAP) are empiric and frequently recommended as "simple" options in an attempt to avoid surgery or the need for CPAP. In addition, more than a dozen surgical options are offered with the intention of affecting either a specific region of the pharynx or a tissue type within the head and neck (e.g., external nasal valves, internal nasal valves, nasal septum, nasal turbinates, soft palate, adenoids, uvula, tonsillar pillars, tonsils, tongue, maxilla, mandible, chin, hyoid bone). Ideally, to ensure compliance with the recommended treatment, 1) the medical or surgical approach should have a biologic basis that is directed at the sites of pathology; 2) a reasonable probability of success should be anticipated; 3) an acceptable level of risk should be clarified; and 4) a realistic level of patient acceptance should be expected.

Bariatric Surgery

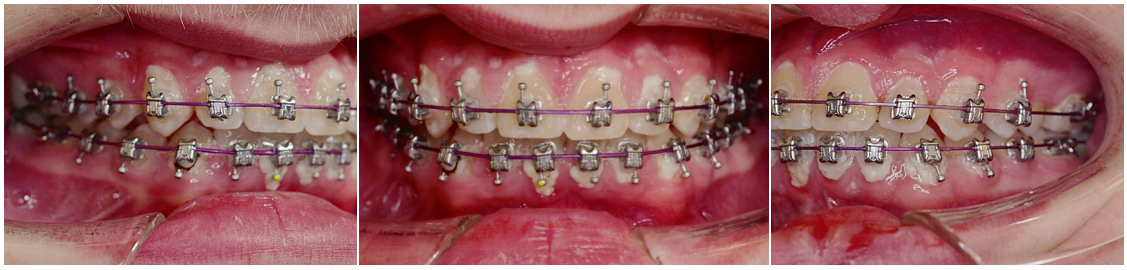
Rasheid and colleagues completed a prospective study in a consecutive series of patients (n = 100) that underwent gastric bypass for the management of obesity to determine the effects of the surgery on OSA.[290] Before surgery, all patients underwent PSG. The mean preoperative RDI was 40 ± 4. Thirteen patients had no OSA, 29 had mild OSA, and 58 had moderate to severe OSA. At 6 months after surgery, patients' BMIs had improved (38 ± 1 kg/m^2 versus 54 ± 1 kg/m^2), and so had their ESS scores (6 ± 1 versus

Text continued on p. 1008

• **Figure 26-4** A 16-year-old boy was referred by his pediatrician for surgical evaluation. He had a history of heavy snoring, restless sleeping, daytime somnolence, attention-deficit/hyperactivity disorder and other behavioral problems, and poor school grades. An attended polysomnogram documented an apnea–hypopnea index of 30 events/hour. Head and neck examination confirmed a normal-sized and functioning soft palate and tongue. The tonsils and adenoids were not enlarged. The intranasal cavity was obstructed (septal deviation and inferior turbinate hypertrophy). The soft palate was resting close to the posterior pharyngeal wall as a result of maxillary retrusion. The tongue was retropositioned as a result of a retrognathic mandible. The patient had previously undergone 2 years of orthodontics and achieved a Class I canine and molar occlusion. The anterior and posterior facial heights were relatively normal. After consultation with his medical and dental team (general dentist, orthodontist, pediatrician, and sleep specialist), the patient agreed to a surgical approach. Orthodontic appliances were placed to protect the periodontium, and he underwent jaw and intranasal surgery. The patient's procedures included maxillary Le Fort I osteotomy (horizontal advancement +10 mm); bilateral sagittal split ramus osteotomies (horizontal advancement +10 mm); osseous genioplasty (horizontal advancement +10 mm); and septoplasty and inferior turbinate reduction. Three months after surgery, he underwent an attended polysomnogram that confirmed the resolution of the obstructive sleep apnea with an apnea–hypopnea index of 2.6 events/hour. He no longer snored or experienced restless sleep. Daytime fatigue improved, as did his behavioral problems and his schoolwork. **A,** Frontal views with smile before and after surgery. **B,** Oblique facial views before and after surgery. **C,** Profile views before and after surgery.

Continued

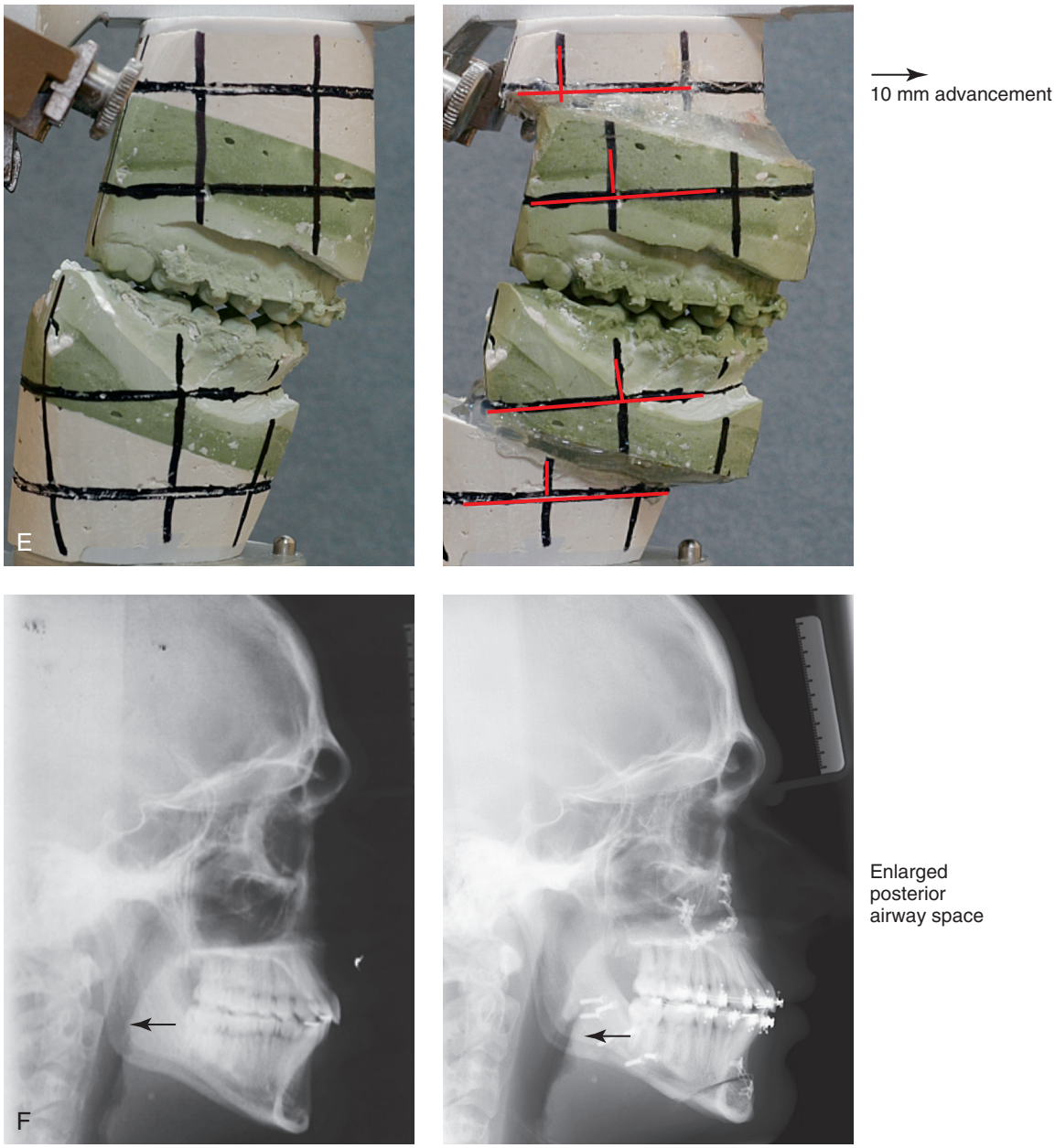
Prior to retreatment



Pre surgery

After treatment

• **Figure 26-4, cont'd D,** Occlusal views before retreatment, with orthodontic appliances in place, and after treatment.



• **Figure 26-4, cont'd** **E,** Articulated dental casts indicate analytic model planning. **F,** Lateral cephalometric radiographs before and after treatment.

• **Figure 26-5** A 15-year-old boy was referred by his pediatrician and arrived with his parents for the surgical evaluation of documented obstructive sleep apnea. He had a history of heavy snoring, restless sleeping, and daytime somnolence. He was diagnosed with attention-deficit/hyperactivity disorder when he was 3 years old, and his school grades are poor. An attended polysomnogram confirmed an overall apnea–hypopnea index of 17 events/hour. In the supine position, his apnea–hypopnea index was 39 events/hour with desaturation to 78%. Head and neck examination confirmed a normal-sized and functioning soft palate and tongue. The tonsils and adenoids had previously been removed, and the intranasal cavity was obstructed (septal deviation and inferior turbinate hypertrophy). The soft palate was resting close to the posterior pharyngeal wall, and the tongue was retropositioned. Both the maxilla and the mandible were deficient in horizontal projection, which explained the reduced airway space. The anterior facial height was mildly deficient. The patient previously underwent orthodontics and had achieved a Class I canine and molar occlusion. In consultation with medical and dental specialists (general dentist, orthodontist, pediatrician, and pulmonologist), the patient agreed to a surgical approach. Orthodontic appliances were placed to protect the periodontium, and he underwent jaw and intranasal surgery. The patient's procedures included maxillary Le Fort I osteotomy (horizontal advancement +10 mm); bilateral sagittal split ramus osteotomies (horizontal advancement +10 mm); osseous genioplasty (horizontal advancement); and septoplasty and inferior turbinate reduction. Three months after surgery, he underwent an attended polysomnogram that confirmed the resolution of the obstructive sleep apnea with an apnea–hypopnea index of 0.9 events/hour and a maximum desaturation of 92%. He was no longer snoring or experiencing restless sleep. His daytime fatigue, behavioral problems, and schoolwork all improved. **A,** Frontal views with smile before and after surgery. **B,** Oblique facial views before and after surgery. **C,** Profile views before and after surgery.

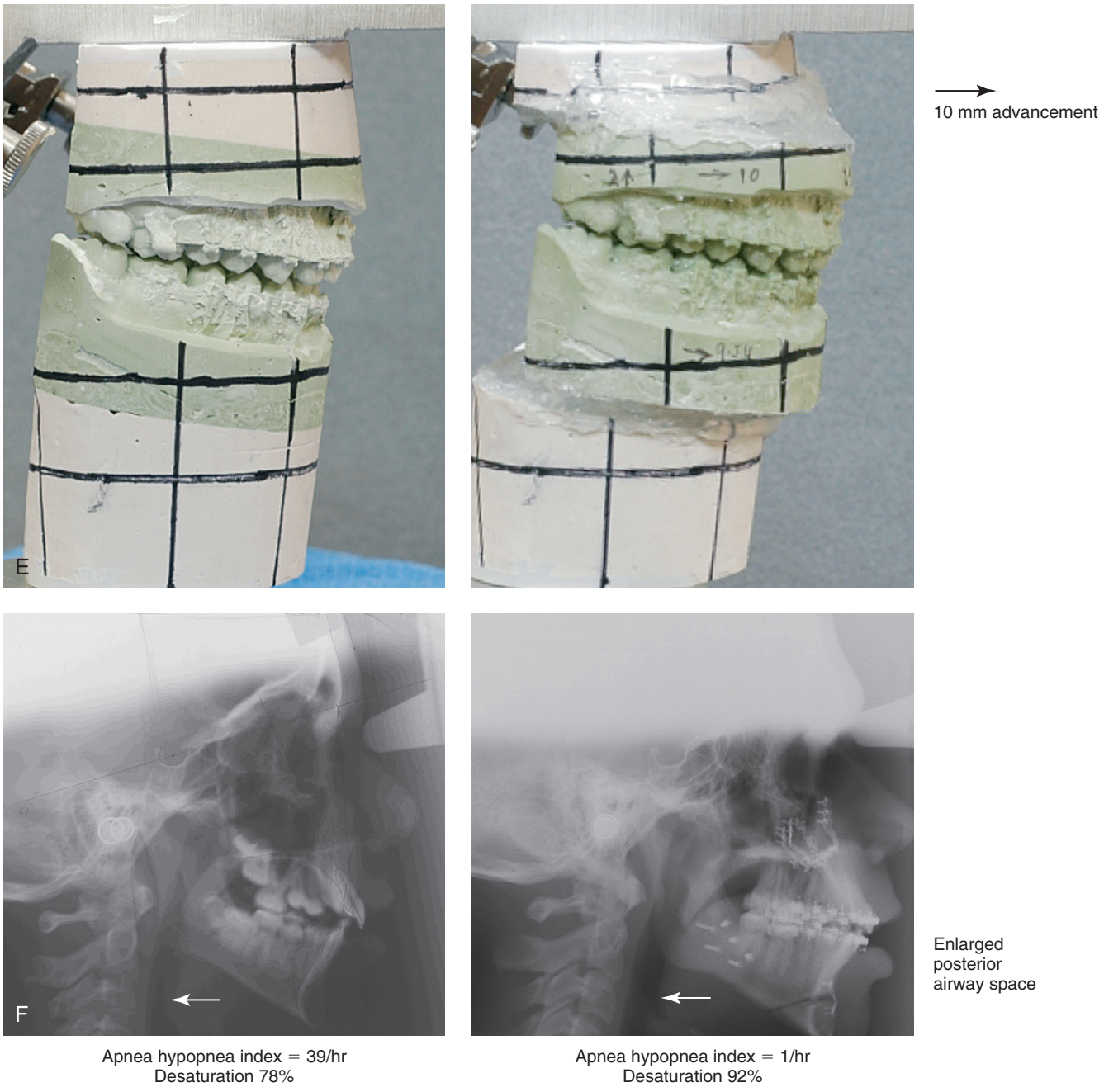
Prior to retreatment

Pre surgery

After treatment

• **Figure 26-5, cont'd D,** Occlusal views before retreatment, with orthodontic appliances in place, and after treatment.

Continued



• **Figure 26-5, cont'd** **E,** Articulated dental casts indicate analytic model planning. **F,** Lateral cephalometric radiographs before and after treatment.

12 ± 0.1). There were also a significant reductions in minimum desaturations during sleep as well as improvements in sleep efficiency. A shortcoming of the study was its conclusion at just 6 months after surgery.

A study by Pillar and colleagues describes the long-term follow up of patients with regard to both weight loss and upper airway findings after bariatric surgery.[273] At 4 months after surgery, there was a significant improvement in the AI from 45 events/hour to 11 events/hour. Over a 7-year time frame, the AI regressed from a mean of 11 events/hour back up to 24 events/hour; however, the AI remained at a much lower level (24 events/hour) than it was before surgery (45 events/hour). In their report, the effectiveness of bariatric surgery was to reduce the mean BMI from 40 kg/m^2 (i.e., morbid obesity) to a mean BMI of 35 kg/m^2 (i.e., obesity) at 7 years after surgery. During the same time frame, the mean AI was reduced from 45 events/hour down to 24 events/hour. Other researchers have also studied the long-term effects of bariatric surgery on patient health.[52,97,116,134]

Continuous Positive Airway Pressure Option

The use of CPAP for OSA was first reported in 1981. Since its introduction, it has become the reference standard for non-surgical management.[43,65,66,71,105,107,114,132,148,159,160,178,181,217,236,267,356,357] With this method, positive pressure can be continuously delivered through a sealed nasal or mouth mask that the patient wears while sleeping. The positive pressure has the potential to pneumatically splint open the whole pharyngeal airway by preventing the soft palate and the tongue from making a seal with the posterior pharyngeal mucosa (i.e., it acts as a counteracting force to the aforementioned collapsing force in the patient with an anatomic predisposition for airway obstruction). The pressure required to achieve this goal is titrated at a sleep laboratory during PSG. CPAP is a sound, evidence-based treatment option that has been shown to be effective when it is used correctly and consistently throughout the night as well as night after night throughout the patient's life. If CPAP is used in this way, it will decrease daytime sleepiness and therefore likely improve the patient's mood and quality of life. The discomfort and difficulty associated with the use of CPAP (e.g., drying of the nasal and oral mucous membranes, dislodgment during sleep, noise, inconvenience when transporting the unit, social displeasure, feelings of claustrophobia) makes long-term compliance a clear problem. Even when defining an acceptable CPAP compliance rate of just 4 hours per night for 70% of nights, only a limited percentage of patients (depending on the study reviewed) who consider themselves users of CPAP are adherent. This does not take into account the significant number of those with OSA who either will not consider trying CPAP or those who have tried it but then refuse to use it. Furthermore, this "acceptable compliance rate" represents only approximately 50% of functional sleep time.[77,252,291,318,385] Sleep medicine clinicians continue to appreciate the complexity of the disorder (i.e., complex chemoreceptor apnea and apnea-related behavioral derangements), and they now acknowledge the inability of a majority of patients to stick with CPAP for the long term, even if they do use it for the short term. Nevertheless, all individuals should be advised of the availability of CPAP and encouraged to consider it.

The American Heart Association/American College of Cardiology Foundation released a position statement about the effects of sleep apnea on cardiovascular disease.[133] The organizations summarized published studies that confirmed the moderate and variable effects of CPAP therapy for OSA on blood pressure. They concluded that individuals with more severe OSA, difficult-to-control hypertension, and better CPAP compliance are likely to have more substantial blood pressure reduction with CPAP. They also recommended that, for those hypertensive patients who were either not willing to use CPAP or not compliant, other effective methods of managing OSA should be considered (e.g., MMCA).

A sleep medicine specialist consult is often beneficial for the patient to maximize his or her education about the disease process and its effects (both behavioral and pathophysiologic) and to explore the CPAP option. Likewise, all patients should be advised of potential surgical alternatives that may apply to them. An evaluation by a surgeon who is familiar with current literature and treatment modalities is beneficial to fully explore non-CPAP options. Clearly, not all surgical options have equal success for each patient. A frank discussion with a knowledgeable surgeon regarding the patient's specific potential benefit-to-risk ratio for each surgical procedure is essential (see discussion to follow).

Oral Appliance Option

The objective of an oral appliance (OA) for the treatment of OSA is to mechanically reposition and hold the mandible in a protruded (forward) location during sleep.[259] As the mandible moves forward, so does the tongue. An effective OA must prevent the sealing of the tongue to the posterior pharyngeal mucosa, thereby limiting obstruction at that site.[374] The upper airway response to oral appliances varies among individuals. It has been stated that up to 50% of individuals with OSA who use an oral appliance will "respond to some extent."[218] Unfortunately, no matter how technically precise the OA is, it can only affect the retroglossal site.[213,325] Most individuals with moderate to severe OSA have obstruction at multiple sites.[377] According to published research, the median compliance rate with an OA after 1 year is 39% with a range of 4% to 82%, depending on the study reviewed.[96] Frequently stated potential long-term negative effects of OAs include: tooth movement with changes in occlusion; injury to the periodontium; excessive salivation or dry mouth; and temporomandibular disorders including masticatory muscle discomfort.[308,310] Other stated reasons for non-compliance include the insufficient reduction of snoring, continued OSA, or social concerns during use. Almeida and colleagues reviewed a series of patients (n = 70) who used OAs to treat snoring or OSA for more than 5 years.[294] The authors found that 44% of those patients had suffered unfavorable occlusal, dental, or temporomandibular joint changes. In research that was carried out by Pancer and colleagues, 14% of study patients (n = 106) were found to have evidence of occlusal changes with the use of an OA for the treatment of OSA.[261] The most common pathologic tooth movements were lingual tipping of the maxillary incisors, proclination of the mandibular incisors, and anterior shifting of the lower first molars. Although some patients were unaware of the occlusal changes, many were acutely bothered by the "shifts" (alterations) in their bites. Bondenmark and Bondenmark and Lindman observed patients (n = 32) after 2 years of continuous OA use.[31,32] Occlusal changes were documented on lateral head radiographs in the study patients. The authors hypothesized that temporomandibular joint remodeling had occurred. To

review, the use of OAs is not generally therapeutic for moderate to severe OSA, and OAs are often not well tolerated in the long term.

An often-stated objective of an OA is for use as a therapeutic trial to determine if a patient would be a candidate for lower jaw surgical advancement. If patients respond to an effective OA, they are said to likely respond in a similar way to mandibular advancement surgery. This is not necessarily the case, because many OAs are designed to position the mandible into a full Class III malocclusion. To achieve the same degree of mandibular advancement, both upper and lower jaw surgery would be required.

Indications for Upper Airway Surgery and Spectrum of Options

Surgery for OSA is indicated when applicable conservative therapies are unsuccessful, intolerable, or unacceptable to the patient with underlying specific anatomically correctible abnormalities that are causing the OSA.[76,80,81,139,193-195,198,202,280,282,285,304,307,358,375] Unfortunately, the use of either CPAP or an OA requires continued personal application of a mechanical device. Likely for this reason, neither is consistently well tolerated or found to be acceptable to the patient in the long run. Indications for surgery to treat OSA are often stated to be a failure of medical management in the individual with one of the following symptoms:

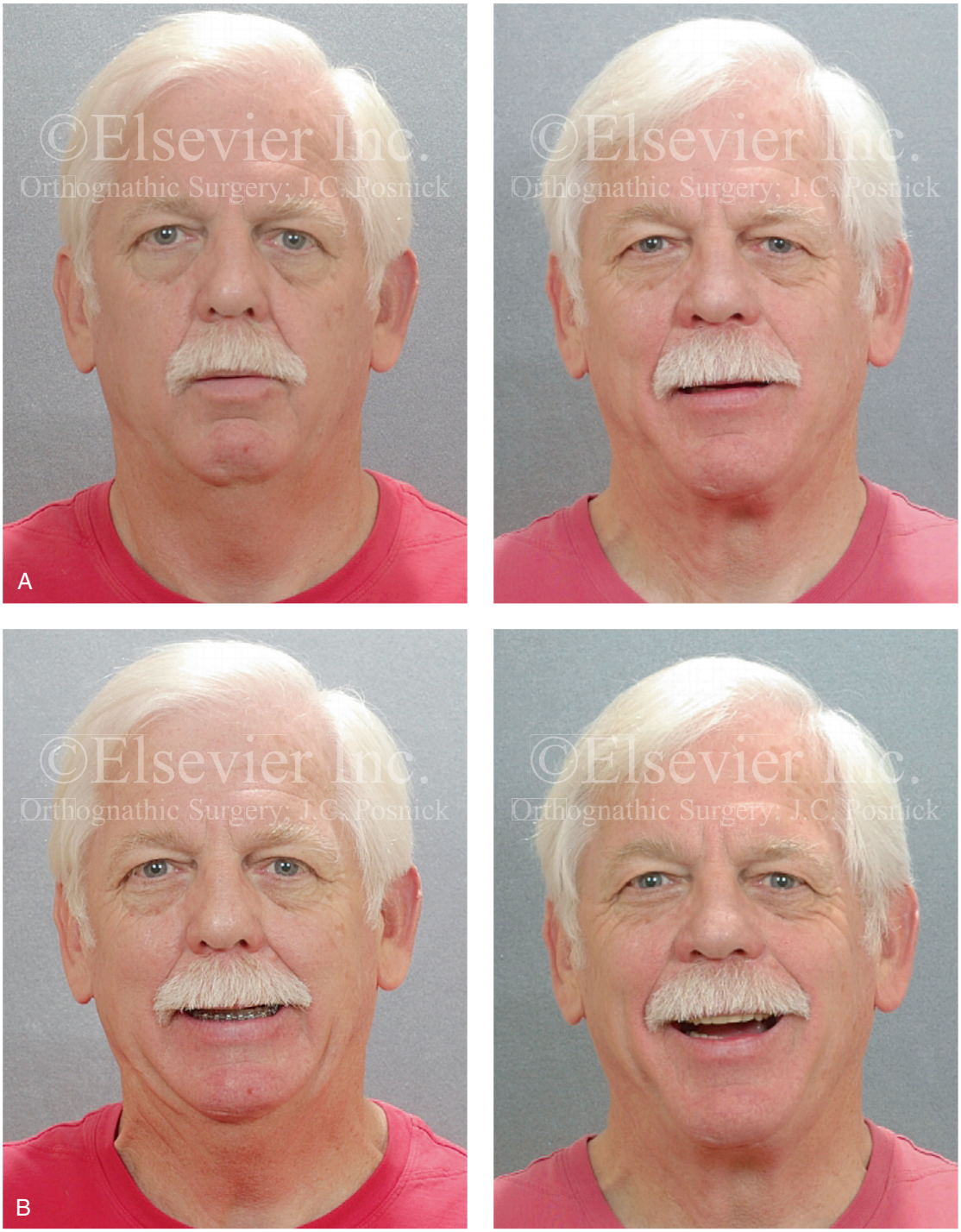
- Excessive daytime sleepiness as a result of OSA
- RDI of more than 20 events/hour
- RDI of less than 20 events/hour but with marked excessive daytime sleepiness
- Oxygen desaturations of less than 90% with apnea events
- Documented hypertension, arrhythmia, or both believed to be triggered by OSA
- Negative esophageal pressures (i.e., >−10 cm of water)
- Anatomic abnormalities of the upper airway (i.e., jaw deformity, fixed intranasal obstructions, chronically enlarged tonsils, enlarged floppy soft palate) that result in apnea events

Although tracheostomy may be considered a way of achieving a "cure" for OSA because it completely bypasses all obstructions of the upper airway, secondary medical and social problems as a result of the tracheostomy cannot be overlooked.[242,368,369] In general, this option is only considered for extreme cases (e.g., newborns or infants with congenital deformities such as the severe retrognathia seen with Treacher Collins syndrome) or as a temporary solution.

Uvulopalatopharyngoplasty (UPPP)—the surgical removal of the uvula, additional parts of the soft palate, the pharyngeal pillars, and the tonsils—to open the airway was originally described by Fujita in 1981.[98,99] Although the effectiveness of this approach for the resolution of OSA is limited, it continues to be an often mentioned treatment option, because it is considered by some to be minimal surgery or a first treatment step (see Figs. 26-3 and 26-6).[2,40,58,93,119,189,272,298,334,342] The reason for its limited success is because it addresses only the retropalatal site, and it does so in an incomplete way.[209] In general, it will shorten the soft palate, but it may also result in a thicker and narrower retropalatal pharyngeal airway as a result of scarring. Interestingly, although improvements in snoring generally occur after UPPP, over time, the AHI actually increases in approximately 30% of patients. This is called *silent apnea;* the relief of the snoring may lead the individual to falsely believe that his or her apnea has also gone away. Realistically, UPPP is expected to be therapeutic only for those with massive soft-tissue redundancy of the pharynx and without concomitant intranasal and retroglossal obstruction.[95,192,204,336,335,391] When either the adenoids, the tonsillar tissue, or the soft palate is significantly hypertrophic or redundant and causing physical blockage of the upper airway, surgery to remove this excessive tissue should be considered. In the absence of a specific syndrome or anomaly that causes increased tongue volume (e.g., Proteus syndrome, vascular anomalies, Beckwith-Wiedemann syndrome), this is rarely a cause of OSA.[47,353] Frequent concerns after intrapharyngeal soft-tissue reduction procedures (e.g., UPPP, tonsillectomy, tongue reduction) include perioperative edema (airway compromise); moderate to severe pain for 2 to 3 weeks; limited success with a single-site approach for the resolution of OSA; velopharyngeal reflux; velopharyngeal insufficiency; pharyngeal scarring (stenosis); and silent apnea.[173,226]

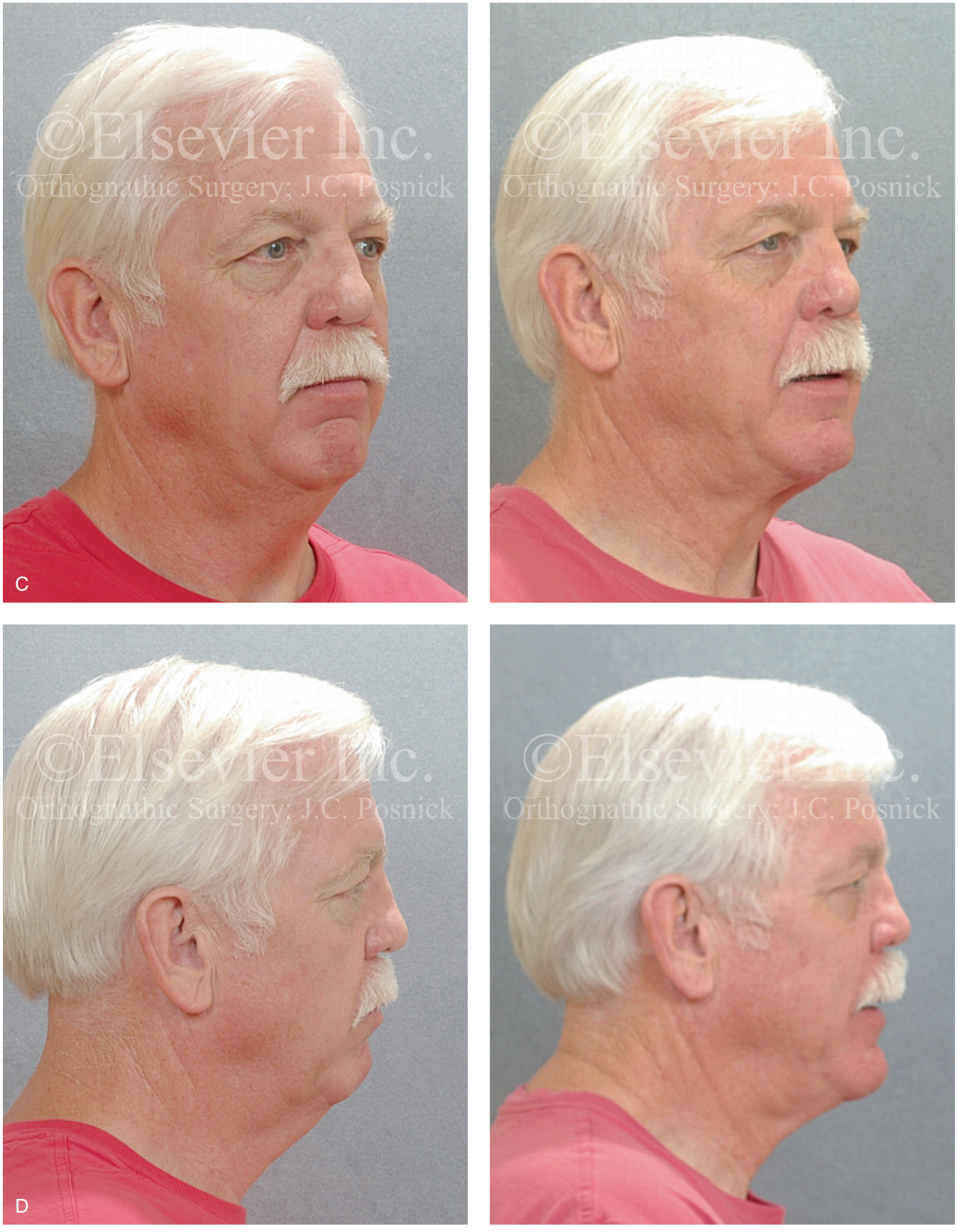
If the maxilla and the mandible are retrusive then consideration should be given to the surgical advancement of these structures to correct the baseline anatomic deformity .[193-195,201,215,277,345,398] MMCA has been shown to enlarge the pharyngeal and hypopharyngeal airway by physically expanding the whole facial skeletal framework (see Figs. 26-4 and 26-7 through 26-14). Because the lateral pharyngeal walls are dynamic, the skeletal expansion also tightens the soft tissues; this subsequently decreases lumen collapsibility. This skeletal approach address's the "small airway" via an extrapharyngeal operation with minimal risk of early postoperative edema-induced upper airway compromise or long-term pharyngeal dysfunction. This operation is also thought to enhance the neuromuscular tone of the pharyngeal dilator musculature (e.g., tensor veli palatini, genioglossus). These anatomic facts explain the highly successful outcomes that have been documented after MMCA. Frequent short-term (perioperative) concerns in the adult patient after MMCA are most often related to: altered occlusion; dysesthesia (sensory loss); limited mouth opening; adjustment to the change in facial appearance; and the downtime required for recovery (see Chapter 25). If septal deviation or thickening or inferior turbinate enlargement or hypertrophy results in significant nasal airway resistance, then these abnormalities can be addressed simultaneously with MMCA (see Chapter 10).[94,191,247,275,276,278] The medical risks associated

Text continued on p. 1045



• **Figure 26-6** A 59-year-old man was referred by his otolaryngologist/head and neck surgeon to discuss further surgical options for the management of documented obstructive sleep apnea. He had already undergone uvulopalatopharyngoplasty, septoplasty, and inferior turbinate reduction. The procedures helped his snoring, but a postsurgery attended polysomnogram confirmed a respiratory disturbance index of 51 events/hour, with desaturations reaching as low as 82%. The patient was also seen by a pulmonary sleep specialist and a cardiologist; mild concentric left ventricular hypertrophy and mild pulmonary hypertension we found. The patient was unable to tolerate continuous positive airway pressure on a regular basis. He lost 20 pounds and was exercising regularly, but this did not resolve his obstructive sleep apnea. Examination confirmed a short face growth pattern with maxillomandibular deficiency (decreased vertical height and limited horizontal projection). During his teenage years, the patient had undergone orthodontic treatment that included four bicuspid extractions. He was now showing signs of periodontal deterioration, and he had a posterior crossbite on the left side. He underwent evaluation by a periodontist, a restorative dentist, and an orthodontist. He agreed to a comprehensive dental rehabilitation approach, with a primary interest in achieving an improved upper airway with the resolution of the obstructive sleep apnea. The sleep specialist and the otolaryngologist were also supportive of this approach. The patient's procedures included maxillary Le Fort I osteotomy (horizontal advancement +10 mm and vertical lengthening +5 mm) with interpositional graft; bilateral sagittal split osteotomies of the mandible (horizontal advancement +12 mm); osseous genioplasty (horizontal advancement); redo septoplasty; and anterior arch soft-tissue procedures (cervical flap elevation, fat removal and platysma muscle plication). Three months after surgery, an attended polysomnogram was carried out, which confirmed a respiratory disturbance index of less than 5 events/hour with the lowest desaturations at 90%. Subjectively, the patient feels that his snoring has resolved, his nighttime sleeping is improved, and his daytime somnolence is greatly diminished. **A,** Frontal views in repose before and after surgery. **B,** Frontal views with smile before and after surgery.

Continued



• **Figure 26-6, cont'd C,** Oblique facial views before and after surgery. **D,** Profile views before and after surgery.

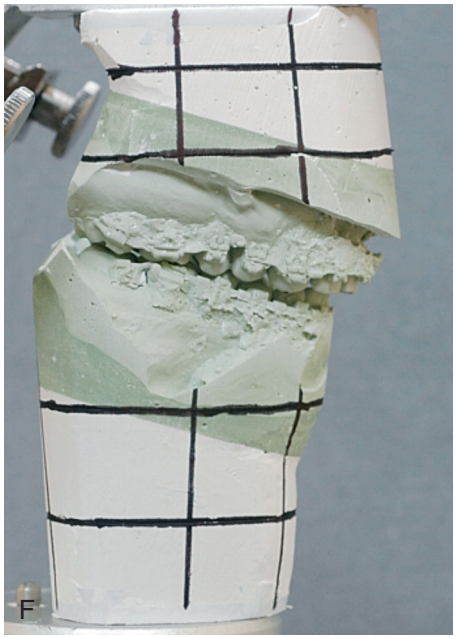
Prior to retreatment after four bicuspid extractions as a teenager

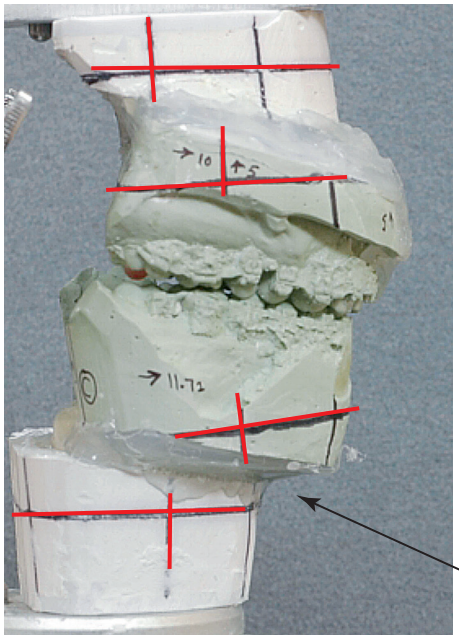
Pre surgery

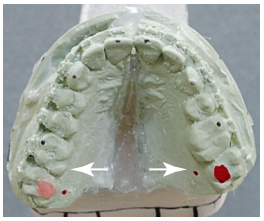
After treatment

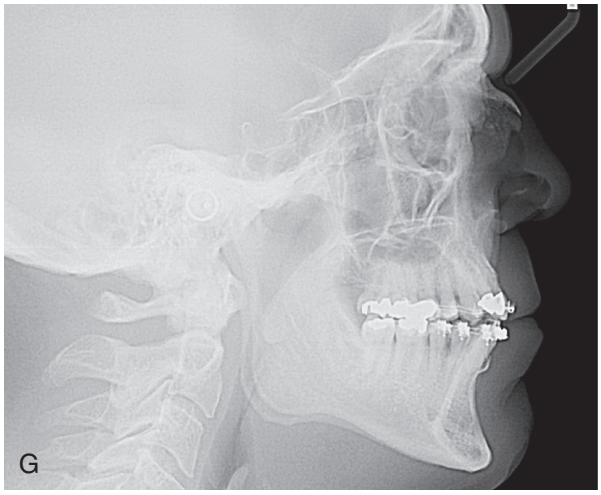
• **Figure 26-6, cont'd E,** Occlusal views before retreatment, with orthodontic appliances in place, and after treatment.

Continued

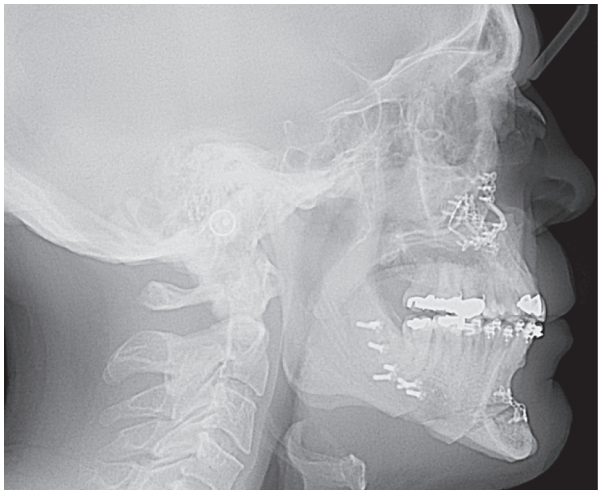








Respiratory disturbance index = 51/hr

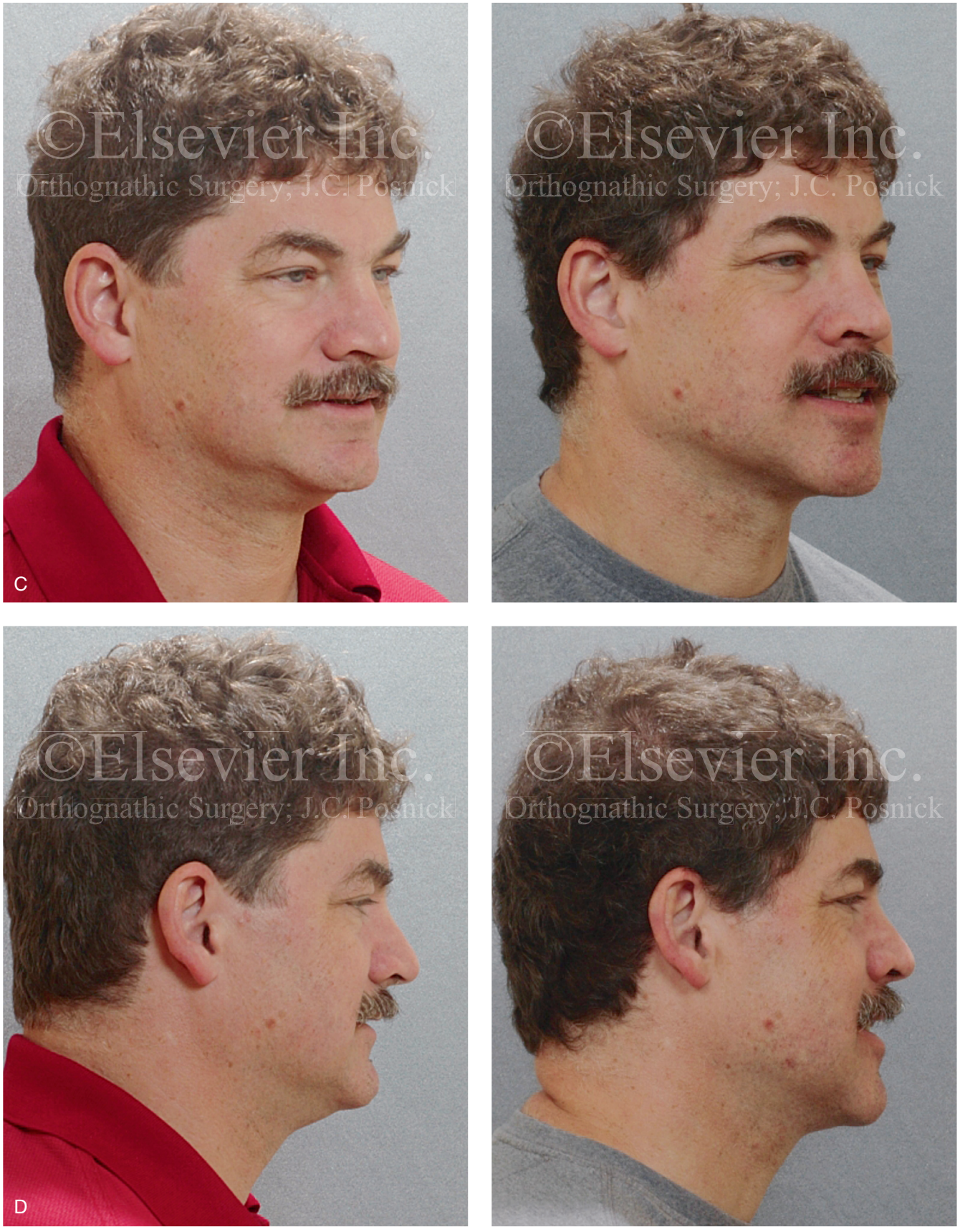


Respiratory disturbance index = 3/hr

• **Figure 26-6, cont'd** **F,** Articulated dental casts that indicate analytic model planning. **G,** Lateral cephalometric radiographs before and after surgery.

• **Figure 26-7** A 51-year-old man was referred by a neurologist to discuss surgical options for the management of documented obstructive sleep apnea. He had heavy snoring, restless sleeping, and significant daytime somnolence. A recent attended polysomnogram confirmed a respiratory disturbance index of 41 events/hour with oxygen desaturation of as low as 80%. A continuous positive airway pressure titration study was effective at 12 cm of pressure; however, the patient was not able to tolerate continuous positive airway pressure. Intranasal examination confirmed a deviated septum and hypertrophic inferior turbinates. The patient's tonsils and adenoids were minimal in size. The soft palate functioned well, but the uvula was enlarged. The tongue was of normal size and function. During his teenage years, the patient had undergone orthodontic treatment, and he had a Class I deep bite. Examination confirmed a short face growth pattern that involved deficient vertical height and limited horizontal projection. Consultations with a sleep specialist, an internist, a general dentist, an otolaryngologist/head and neck surgeon, and an orthodontist were carried out. The patient agreed to a surgical approach that included orthodontics as needed to protect the periodontium. The procedures included maxillary Le Fort I osteotomy (horizontal advancement +10 mm and vertical lengthening +8 mm) with interpositional allograft; bilateral sagittal split osteotomies of the mandible (horizontal advancement); osseous genioplasty (horizontal advancement); uvulectomy; and septoplasty and inferior turbinate reduction. After surgery, the patient's snoring was resolved, his nighttime sleeping improved, and his daytime somnolence was greatly diminished. A 1-year postoperative polysomnogram confirmed a respiratory disturbance index of 13 events/hour. **A,** Frontal views in repose before and after surgery. **B,** Frontal views with smile before and after surgery.

Continued



• **Figure 26-7, cont'd C,** Oblique facial views before and after surgery. **D,** Profile views before and after surgery.

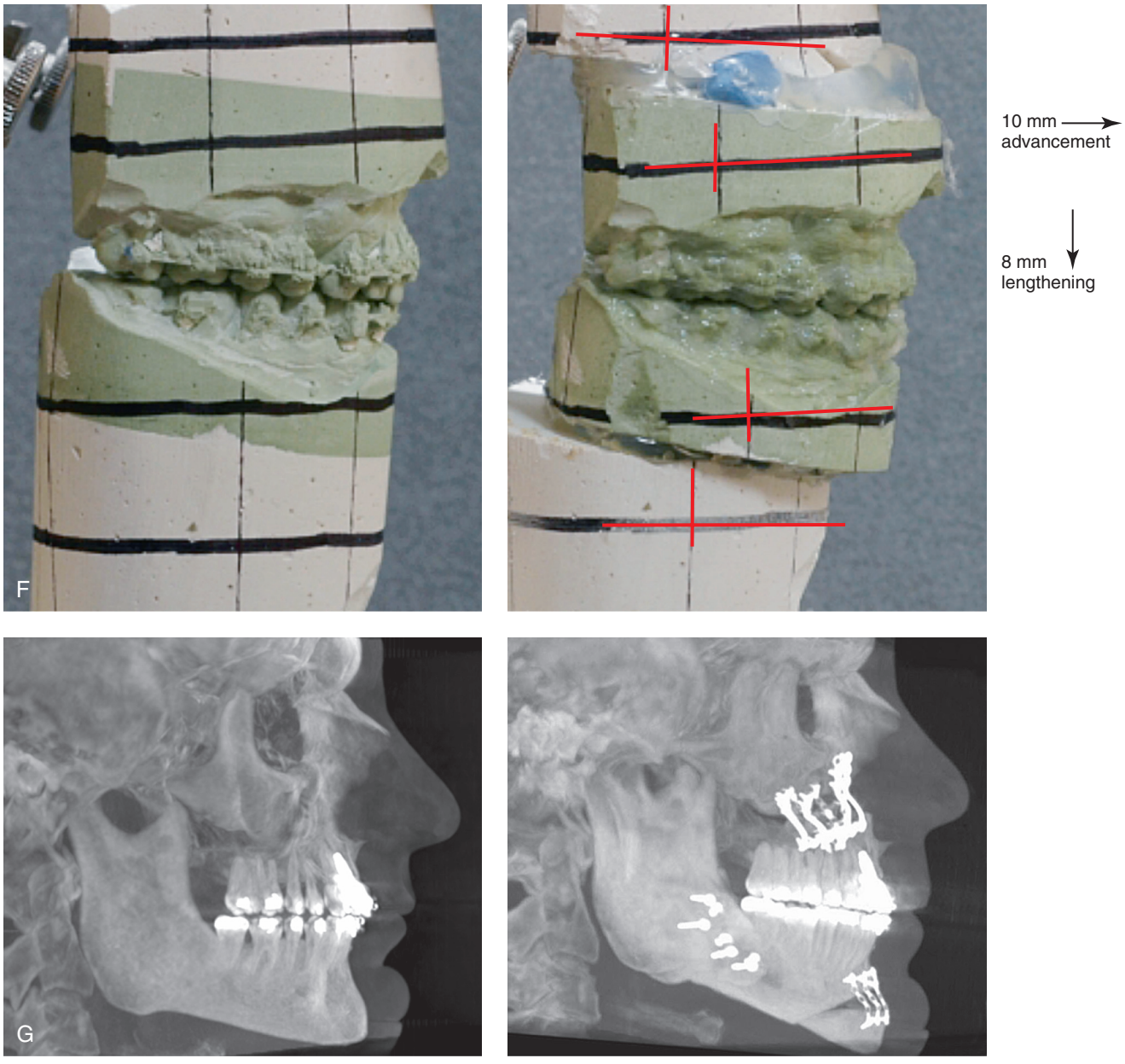
Prior to treatment

Pre surgery

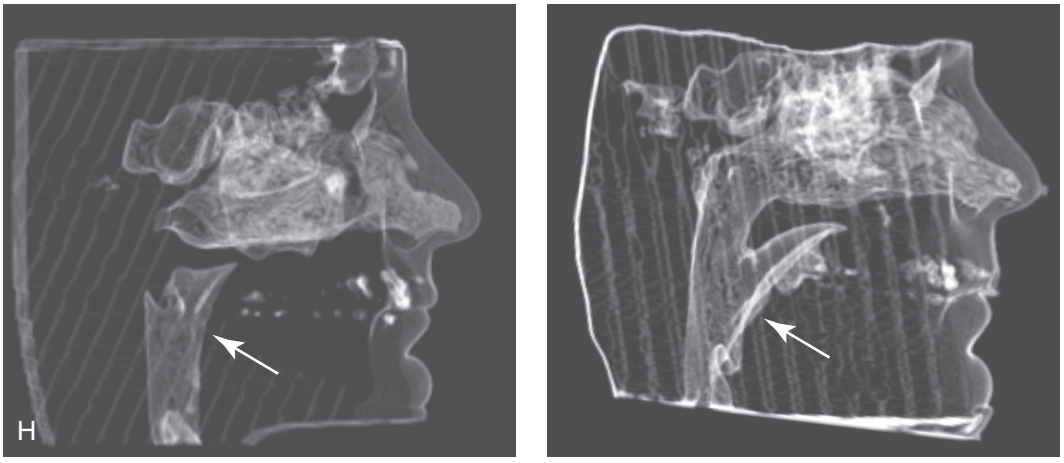
After treatment

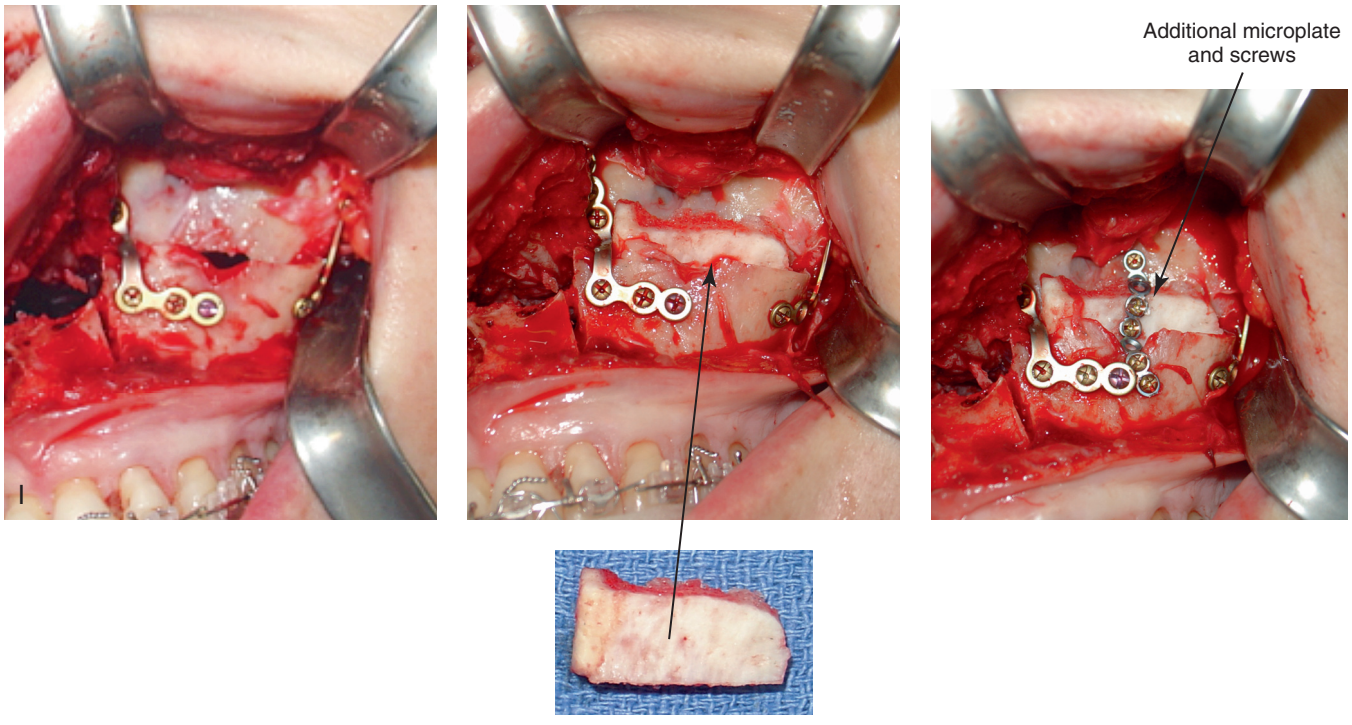
• **Figure 26-7, cont'd E,** Occlusal views before retreatment, with orthodontic appliances in place, and after treatment.

Continued



• **Figure 26-7, cont'd** **F,** Articulated dental casts that indicate analytic model planning. **G,** Lateral cephalometric radiographs before and after surgery.





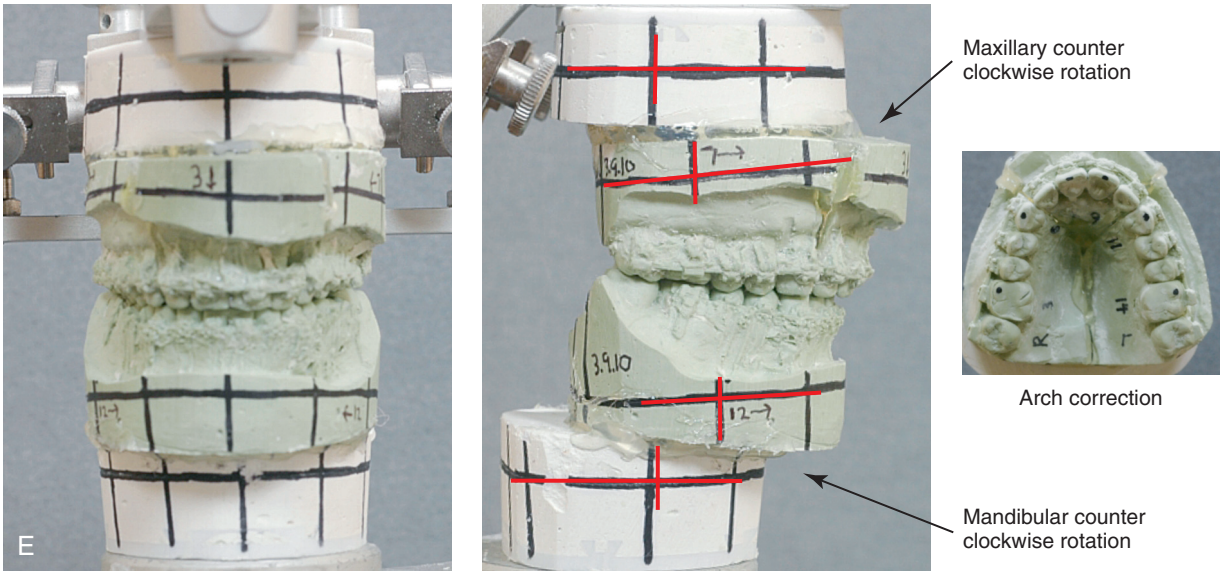
• **Figure 26-7, cont'd H,** Computed tomography scans reformatted to indicate pharyngeal airway space before and after surgery. **I,** Close-up intraoperative view of the left side Le Fort I osteotomy site with plate and screw fixation in place (zygomatic buttress and piriform aperture regions). Also shown is the corticocancellous iliac graft that was crafted and then inset in the defect; this was followed by additional plate and screw fixation of the graft.

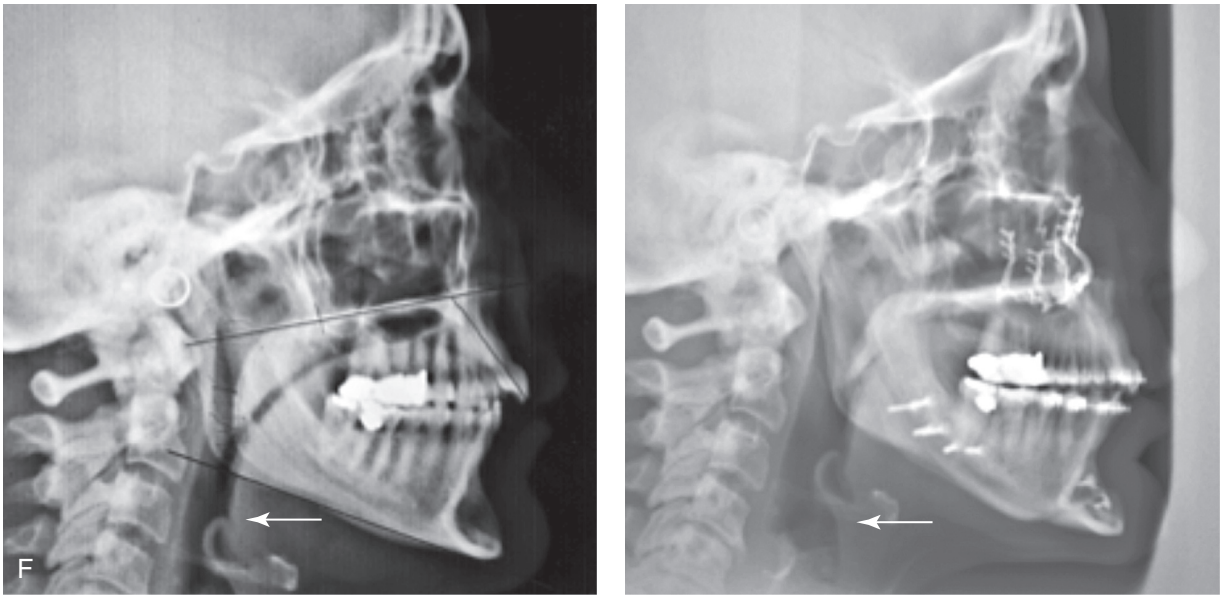
Continued

• **Figure 26-8** A 50-year-old woman was referred by her general dentist to an orthodontist and then for surgical evaluation. She had a mandibular deficiency and a constricted upper jaw width growth pattern that resulted in a Class II deep-bite excess overjet malocclusion. Over the years, there was destruction to the dentition and the periodontal apparatus with generalized bone loss and gingival recession. She was now under the care of a restorative dentist and a periodontist. To resolve the malocclusion, maxillary expansion and mandibular advancement in combination with orthodontics were recommended. As part of her referral to this surgeon, a history was taken that documented heavy snoring, restless sleeping, and daytime somnolence. An attended polysomnogram confirmed a respiratory disturbance index of 42.7 events/hour. There was intranasal site obstruction as a result of septal deviation and inferior turbinate enlargement. There was also retropalatal obstruction caused by maxillary retrusion and a retropositioned tongue that resulted from mandibular retrusion. The soft palate and the tongue were of normal size and function. There were minimum tonsil and adenoid structures. Continuous positive airway pressure was tried but not well tolerated. Surgery was agreed to in combination with orthodontics to improve the malocclusion and open the airway. The procedures that were carried out included maxillary Le Fort I osteotomy (horizontal advancement +7 mm, vertical lengthening +3 mm, and arch form correction); bilateral sagittal split osteotomies of the mandible (horizontal advancement +12 mm); osseous genioplasty (horizontal advancement); septoplasty and inferior turbinate reduction; and an anterior approach to the neck (cervical flap elevation, supraplatysmal defatting, and vertical platysma muscle plication). Subjectively, there was good relief of snoring, restless sleeping, and daytime fatigue. One year after surgery, an attended polysomnogram confirmed the resolution of the obstructive sleep apnea, with a respiratory disturbance index of 1.9 events/hour. **A,** Frontal views in repose before and after treatment. **B,** Oblique facial views before and after treatment. **C,** Profile views before and after treatment.

• **Figure 26-8, cont'd D,** Occlusal views before treatment, with orthodontics in progress, and after treatment.

Continued





Respiratory disturbance index = 42.7/hr

Respiratory disturbance index = 1.9/hr

• **Figure 26-8, cont'd** **E,** Articulated dental casts that indicate analytic model planning. **F,** Lateral cephalometric radiographs before and after surgery.

• **Figure 26-9** A 25-year-old man was referred by his general dentist to an orthodontist and then to this surgeon for the evaluation of a dentofacial deformity. Examination confirmed a maxillary deficiency and a relative mandibular excess growth pattern; this resulted in a severe Class III negative overjet anterior open-bite malocclusion. The maxillomandibular deformities had a negative impact on the patient's speech articulation, swallowing, chewing, breathing, and lip closure/posture. The patient's history confirmed lifelong obstructed nasal breathing, restless sleeping, heavy snoring, and daytime fatigue. An attended polysomnogram was carried out and documented a respiratory disturbance index of 14 events/hour, with desaturations of as low as 87%. The patient underwent additional evaluations by a sleep specialist, an otolaryngologist, a speech pathologist, a restorative dentist, and a periodontist. He agreed to a comprehensive surgical and dental rehabilitative approach, and his periodontal treatment was followed by orthodontic decompensation. The patient's procedures included maxillary Le Fort I osteotomy in segments (horizontal advancement +10 mm, arch expansion and minimal clockwise rotation) with interpositional graft; bilateral sagittal split osteotomies of the mandible (horizontal advancement +4 mm and counterclockwise rotation); osseous genioplasty (horizontal advancement); and inferior turbinate reduction and septoplasty. The patient experienced relief of his snoring, improvements in his sleep quality, less daytime fatigue, and improved breathing during the day. Interestingly, the 4-month postoperative attended polysomnogram indicated only mild reduction of the sleep indexes. Reevaluation by the sleep specialist recommended the avoidance of supine sleep, with no need for further treatment. **A,** Frontal views in repose before and after surgery. **B,** Frontal views with smile before and after surgery.

Continued

• **Figure 26-9, cont'd C,** Oblique facial views before and after surgery. **D,** Profile views before and after surgery.

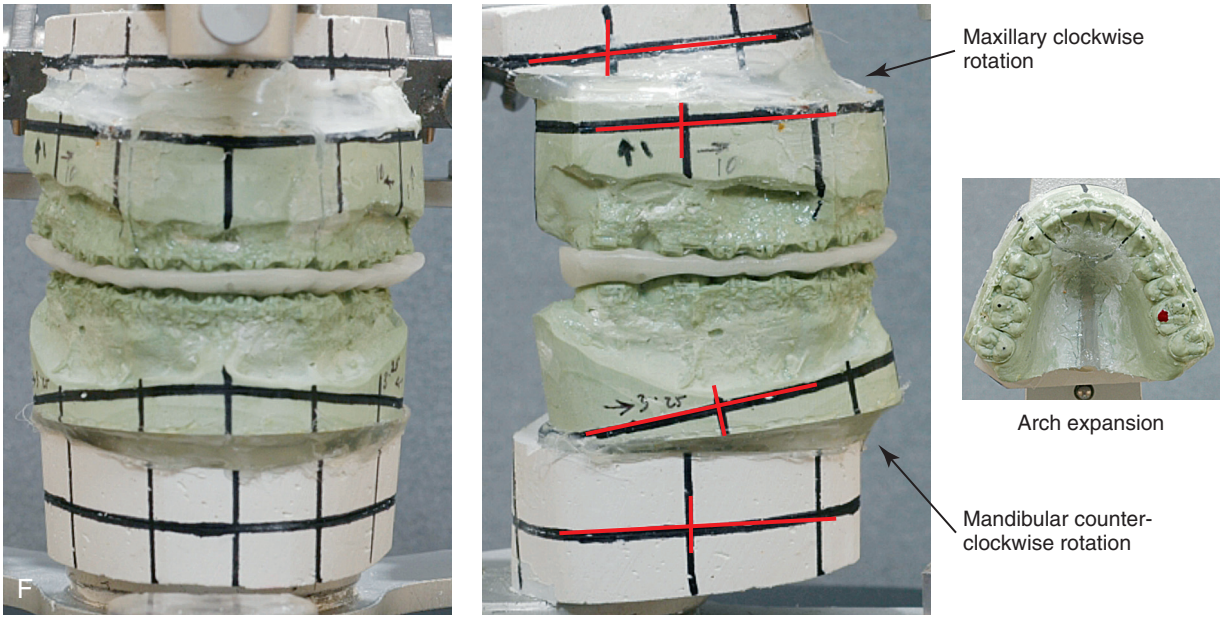
Prior to treatment

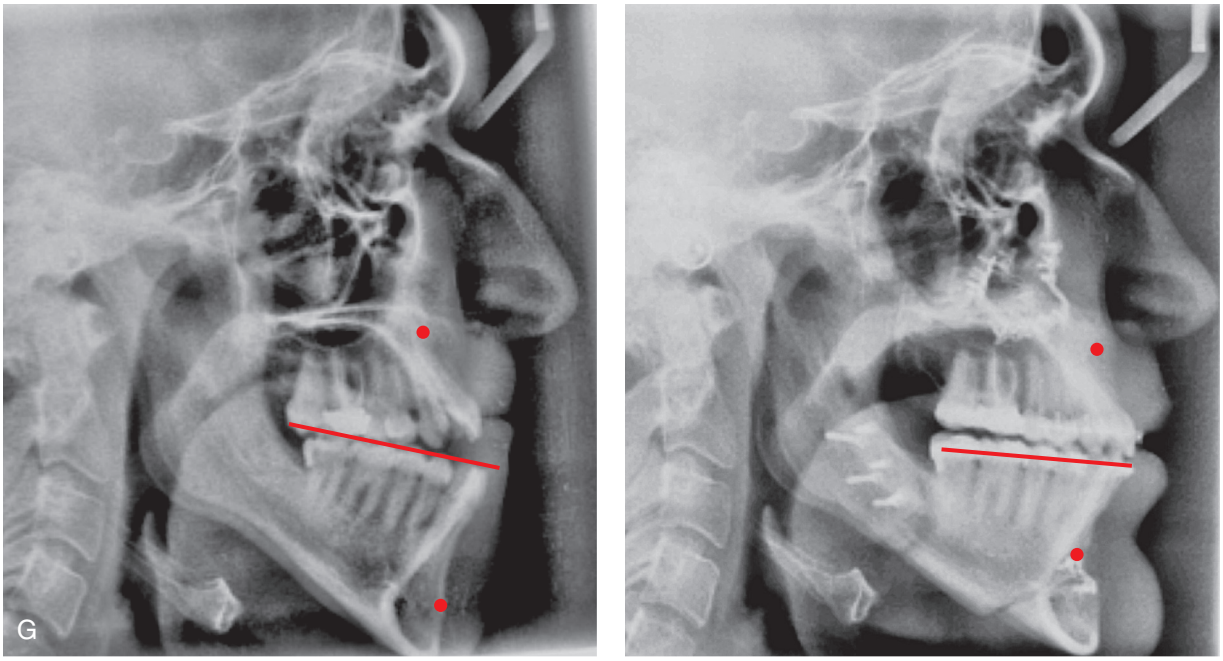
Pre surgery

After treatment

• **Figure 26-9, cont'd E,** Occlusal views before treatment, with orthodontic appliances in place, and after treatment. Note the pulpal necrosis of the right maxillary lateral incisor, which required root canal therapy and crown coverage.

Continued





Apnea hypopnea index = 14/hr

Apnea hypopnea index = 14/hr

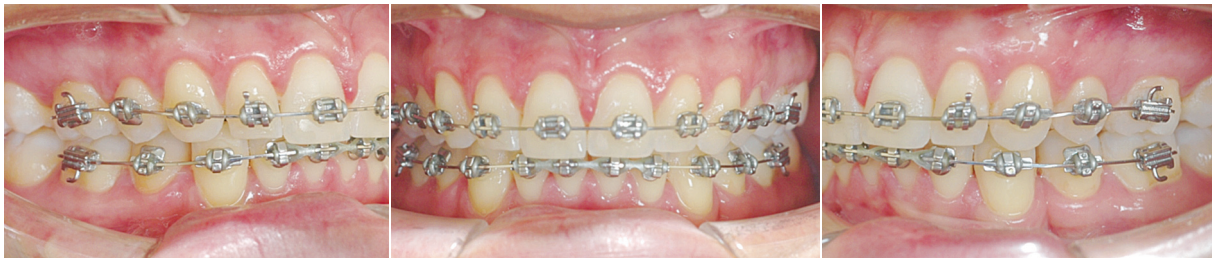
• **Figure 26-9, cont'd** **F,** Articulated dental casts that indicate analytic model planning. **G,** Lateral cephalometric radiographs before and after surgery.

• **Figure 26-10** A 37-year-old man was referred to this surgeon with a request to have his chin implant removed because he was displeased with the aesthetic results. When he was 20 years old, he had undergone a chin augmentation (silicone implant) in an attempt to camouflage the small lower jaw profile. Ten years later, he underwent four bicuspid extractions and orthodontic alignment to improve the occlusion. He had undergone a septorhinoplasty procedure, but he was still experiencing continued difficulty breathing through the nose. During the patient's referral to this surgeon, an examination confirmed maxillomandibular deficiency. He was recognized as having a short face growth pattern with decreased lower anterior facial height and limited facial projection. There was an accentuated labiomental "lip curl" and a "button appearance" of the chin. It was clear that the patient suffered with snoring, restless sleeping, and excessive daytime fatigue. An attended polysomnogram confirmed a respiratory disturbance index of 16 events/hour, with desaturations of 89%. A titration study confirmed the need for 12 cm of water pressure to reduce the respiratory disturbance index to 3 events/hour. **A,** Frontal views in repose before and after surgery. **B,** Frontal views with smile before and after surgery.

Continued

• **Figure 26-10, cont'd** The use of continuous positive airway pressure was attempted, but the patient found it to be uncomfortable and difficult to use. Consultations with a sleep specialist, an otolaryngologist, and an orthodontist confirmed the advantage of an intranasal and orthognathic surgical approach. The patient agreed to perioperative orthodontics and surgery to improve the airway and facial appearance. The patient's procedures included maxillary Le Fort I osteotomy (horizontal advancement +9 mm, vertical lengthening +5 mm) and interpositional grafting; bilateral sagittal split osteotomies of the mandible (horizontal advancement +12 mm); removal of the chin implant; osseous genioplasty (horizontal advancement and vertical lengthening) with interpositional grafting; reduction of the inferior turbinates; redo septoplasty; and an anterior approach to the neck (cervical flap elevation, suprapla-tysmal defatting, and vertical platysma muscle plication). As a result of the procedures, the patient experienced relief of his snoring, improved sleep quality, diminished daytime fatigue, and improved breathing during the day. Six months after surgery, he underwent an attended polysomnogram that indicated a normal sleep study, with no snoring or apnea events. **C,** Oblique facial views before and after surgery. **D,** Profile views before and after surgery.

Prior to retreatment after 4 bicuspid extractions as a teenager

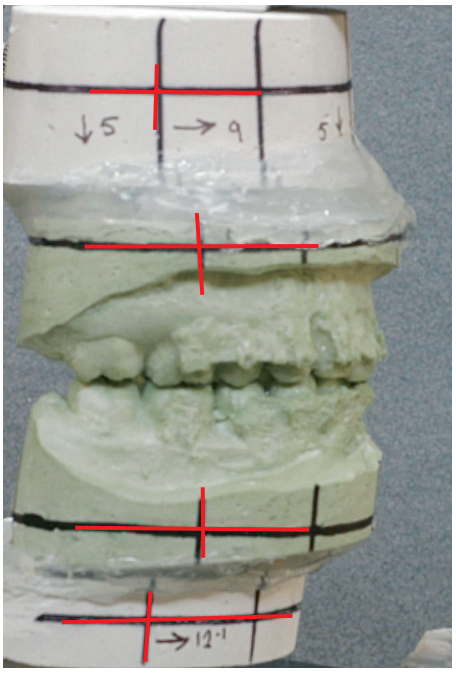


Pre surgery

After treatment

• **Figure 26-10, cont'd E,** Occlusal views before retreatment, with orthodontic appliances in place, and after treatment.

Continued



→ 9 mm maxillary advancement

Respiratory disturbance index = 16/hr

Respiratory disturbance index = 3/hr

Silicone chin implant to be removed

• **Figure 26-10, cont'd F,** Articulated dental casts that indicate analytic model planning. **G,** Lateral cephalometric radiographs before and after surgery (note the preoperative chin implant).

• **Figure 26-11** A 33-year-old man was referred by his orthodontist for the surgical evaluation of a dentofacial deformity. Examination confirmed a long face growth pattern with a Class III anterior open-bite malocclusion. During the mixed dentition, the patient underwent growth modification and orthodontic treatment that included the use of a Frankel appliance, a chin cup, and rapid palatal expansion. By the end of high school, he was left with a residual anterior open bite, a constricted maxillary arch, and a gummy smile. When he was 29 years old and had a lifelong history of heavy snoring, difficulty sleeping at night, and daytime fatigue, he underwent a sleep study. The polysomnogram revealed moderate obstructive sleep apnea with a respiratory disturbance index of 16 events/hour and oxygen desaturations down to 86%. A continuous positive airway pressure titration study confirmed the need for 10 cm of water pressure to resolve the respiratory events. The current head and neck examination documented a deviated septum; enlarged inferior turbinates; an elevated nasal floor; narrow pyriform rims; a soft palate close to the pharyngeal wall; and a retropositioned tongue. The tonsils were not enlarged, and the tongue and soft palate were of normal size. **A,** Frontal views in repose before and after surgery. **B,** Frontal views with smile before and after surgery.

Continued

• **Figure 26-11, cont'd** The patient hoped to achieve improved breathing during the day, better sleeping at night, and resolution of his obstructive sleep apnea. He also wanted correction of his malocclusion, improvements in his lip closure/posture, and an enhanced profile and smile. He agreed to a comprehensive orthodontic and surgical approach. With the orthodontic relief of his dental compensation, intranasal, orthognathic, and facial surgery was carried out. The patient's procedures included maxillary Le Fort I osteotomy in three segments (horizontal advancement +8 mm, vertical shortening –4 mm, and arch expansion); bilateral sagittal split osteotomies of the mandible (horizontal advancement +12 mm and counterclockwise rotation); osseous genioplasty (horizontal advancement and vertical shortening); reduction of the inferior turbinates, septoplasty, and recontouring of the nasal floor; and an anterior approach to the neck (cervical flap elevation, supraplatysmal defatting, and vertical platysma muscle plication). He experienced the relief of his snoring, improvement in his quality of sleep, the ability to breathe through the nose during the day, and less daytime fatigue. He had not yet undergone a formal posttreatment polysomnogram. **C,** Oblique facial views before and after surgery. **D,** Profile views before and after surgery.

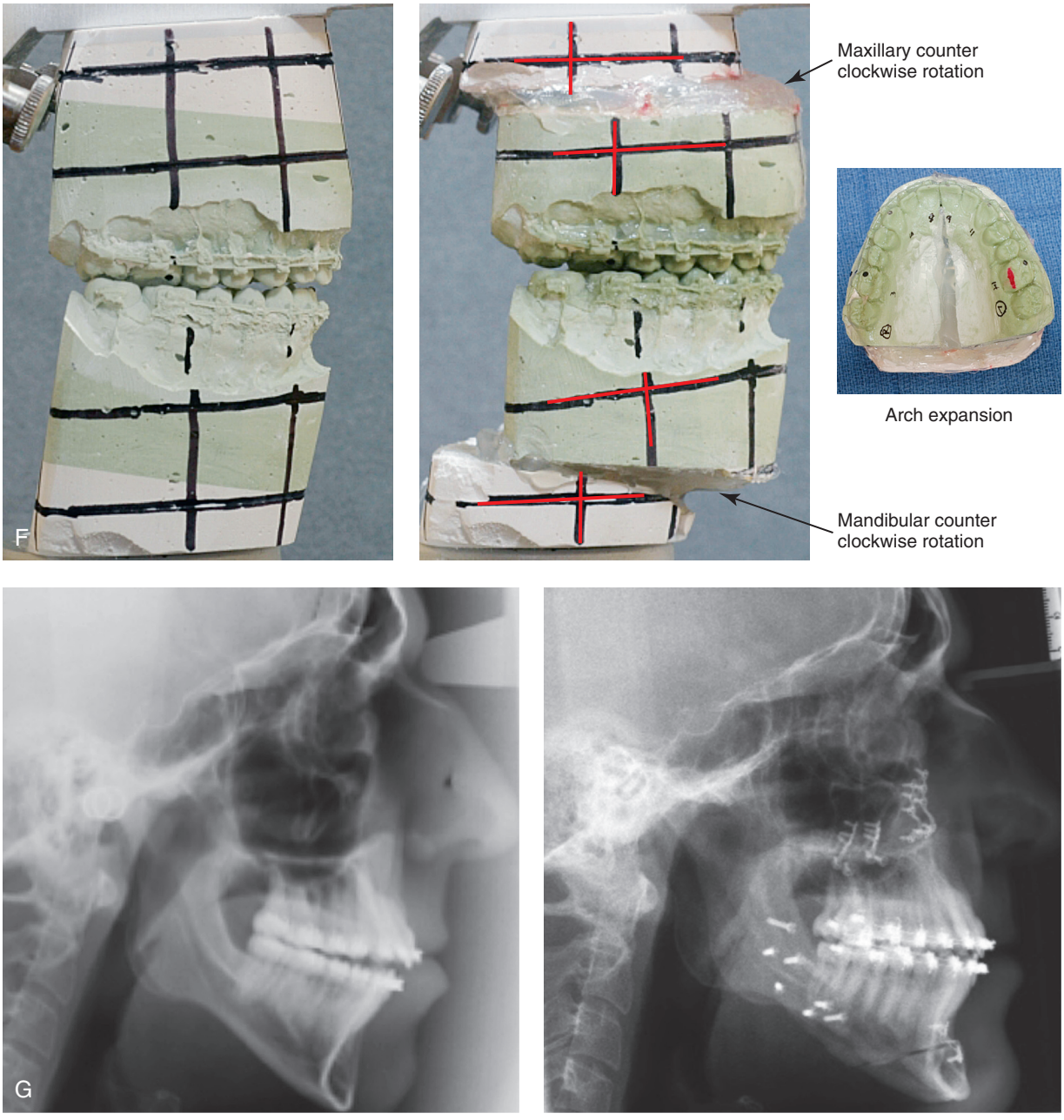
Prior to retreatment

Pre surgery

After treatment

• **Figure 26-11, cont'd E,** Occlusal views before retreatment, with orthodontic appliances in place, and after treatment.

Continued



• **Figure 26-11, cont'd** **F,** Articulated dental casts that indicate analytic model planning. **G,** Lateral cephalometric radiographs before and after surgery.

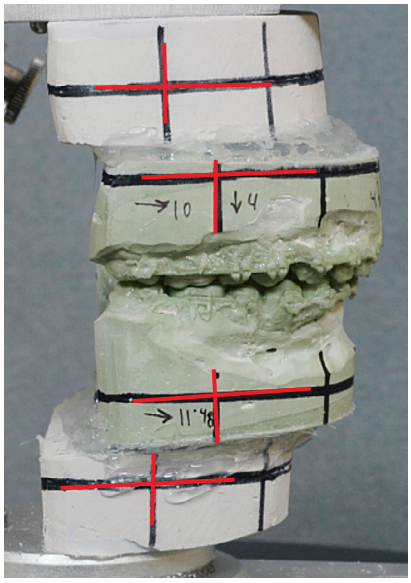
• **Figure 26-12** A 32-year-old man was referred by his internist and otolaryngologist/head and neck surgeon to discuss further surgical options for the management of document obstructive sleep apnea. An attended polysomnogram confirmed a respiratory disturbance index of 56 events/hour, with oxygen desaturations reaching as low as 84%. The patient's sleep efficiency was reduced to 46%. He underwent a split study that confirmed the need for 8 cm of continuous positive airway pressure to significantly reduce the respiratory disturbance index. He was unable to effectively use the continuous positive airway pressure device as a result of claustrophobia, excessive noise, and poor mask fit. Since his early childhood years, the patient was known to have a jaw deformity with malocclusion. He underwent 2 years of orthodontic treatment when he was 12 to 14 years old; this resulted in improvement of the occlusion but residual jaw deformity. Examination confirmed a short face growth pattern with maxillomandibular deficiency (decreased vertical height and limited horizontal projection). The tonsils were not inflamed, but they were moderately enlarged, and the uvula was mildly long and floppy. The tongue was of normal size, the septum was straight, and the inferior turbinates were enlarged. **A,** Frontal views in repose before and after surgery. **B,** Frontal views with smile before and after surgery.

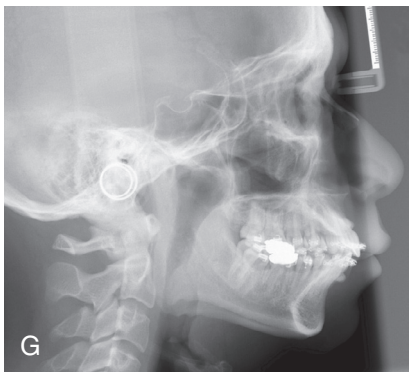
Continued

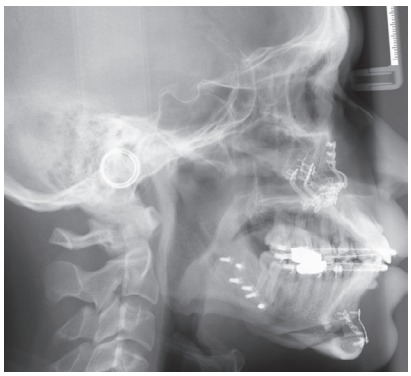
• **Figure 26-12, cont'd** The patient underwent evaluation by a restorative dentist, an orthodontist, a sleep specialist, an otolaryngologist/head and neck surgeon, and an internist. His primary motivations were to achieve an improved airway and to resolve his obstructive sleep apnea. He was also interested in improved profile aesthetics and long-term dental health. We discussed potential benefits of tonsillectomy and of the reduction of the size of the soft palate, but he preferred a skeletal approach, thus reserving the option of soft-tissue reduction if needed as a secondary procedure. With orthodontic appliances in place, the patient's procedures included maxillary Le Fort I osteotomy (horizontal advancement +10 mm and vertical lengthening +4 mm) with interpositional allogenic graft; bilateral sagittal split osteotomies of the mandible (horizontal advancement +12); osseous genioplasty (horizontal advancement); and inferior turbinate reduction. He experienced relief of his snoring, improvement in his quality of sleep, better breathing during the day, and less daytime fatigue. Three months postoperatively, he underwent an attended polysomnogram. Interestingly, after surgery, he chose to sleep with his lips taped together out of the belief that this improves sleep hygiene, including air humidification. Thus, the sleep study was carried out with the patient's lips taped together. An overall respiratory disturbance index of 15 events/hour was documented, with average desaturations to 95%. **C,** Oblique facial views before and after surgery. **D,** Profile views before and after surgery.

Pre surgery

After treatment







Respiratory disturbance index = 56/hr

Respiratory disturbance index = 15/hr

• **Figure 26-12, cont'd** **E,** Occlusal views before and after treatment. **F,** Articulated dental casts that indicate analytic model planning. **G,** Lateral cephalometric radiographs before and after surgery.

• **Figure 26-13** A 43-year-old man was referred by a restorative dentist to an orthodontist and then for surgical evaluation of a dentofacial deformity characterized by maxillomandibular deficiency. Despite orthodontic treatment (including four bicuspid extractions when the patient was a teenager), there remained a Class III negative overjet bilateral posterior cross-bite malocclusion. A degree of labial bone loss and gingival recession was appreciated. Earlier during his life, the patient's nose was broken during a sports injury, and he had since experience chronic obstructive breathing. Examination confirmed a deviated septum and inferior turbinate enlargement. The tonsils, the tongue, and the soft palate were essentially normal; the uvula was somewhat enlarged. He admitted to heavy snoring, restless sleeping, and excessive daytime fatigue. A sleep study confirming a respiratory disturbance index of 37 events/hour with desaturations reaching a low of 86%. The patient was fitted with a continuous positive airway pressure device (8 cm of water pressure per hour) as temporary treatment while his orthodontics were initiated. After the orthodontic relief of dental compensation, surgery was carried out. The patient's procedures included maxillary Le Fort I osteotomy in two segments (horizontal advancement +10 mm, vertical lengthening –2 mm, and arch expansion) with allogenic interpositional grafting; bilateral sagittal split osteotomies of the mandible (horizontal advancement +13 mm and counterclockwise rotation); osseous genioplasty (horizontal advancement); inferior turbinate reduction and septoplasty; and an anterior approach to the neck (cervical flap elevation, supraplatysmal defatting, and vertical platysma muscle plication). After surgery, there was an immediate relief of the patient's snoring as well as improvements in his quality of sleep, his ability to breathe through his nose, his concentration, and his daytime fatigue. The 6-month postoperative attended polysomnogram confirmed a reduction in the respiratory disturbance index to 10 events/hour. **A,** Frontal views in repose before and after surgery. **B,** Frontal views with smile before and after surgery.

• **Figure 26-13, cont'd C,** Oblique facial views before and after surgery. **D,** Profile views before and after surgery.

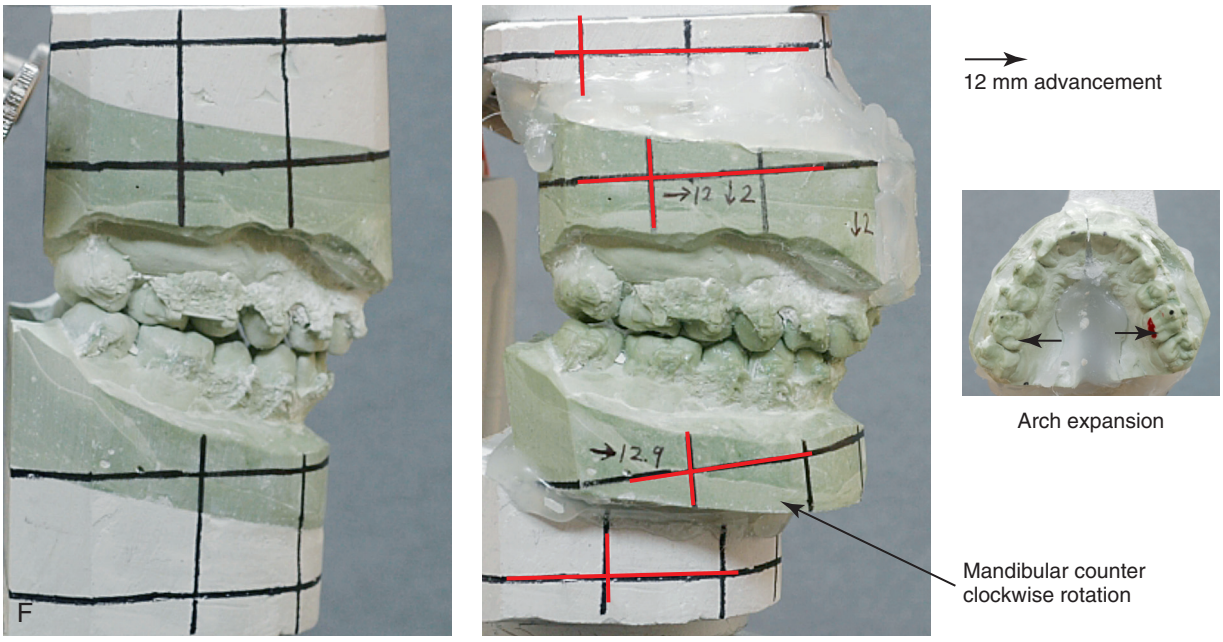
Continued

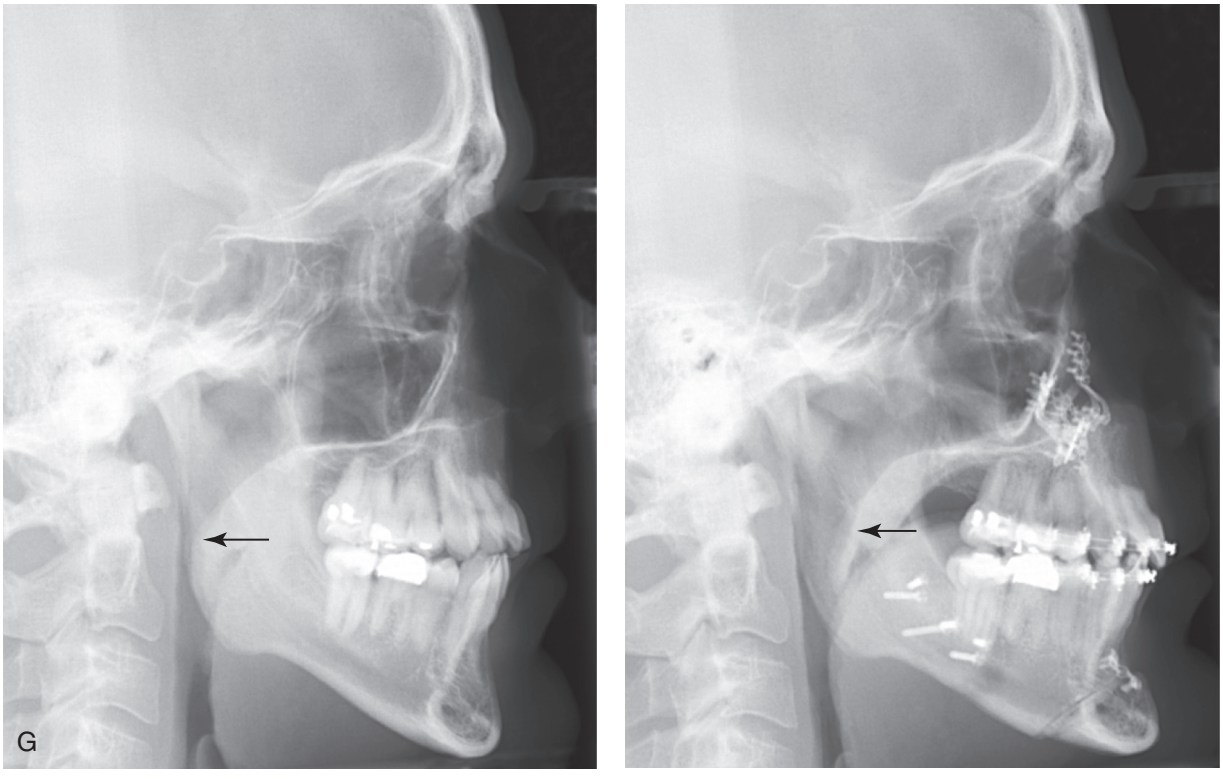
Prior to retreatment after 4 bicuspid extractions as a teenager

Pre surgery

After treatment

• **Figure 26-13, cont'd E,** Occlusal views before retreatment, with orthodontic appliances in place, and after treatment.





Respiratory disturbance index = 37/hr

Respiratory disturbance index = 10/hr

• **Figure 26-13, cont'd** **F,** Articulated dental casts that indicate analytic model planning. **G,** Lateral cephalometric radiographs before and after surgery.

• **Figure 26-14** A 34-year-old man was referred by his orthodontist for a surgical evaluation of a longstanding dentofacial deformity with malocclusion. Examination confirmed a long face asymmetric Class III growth pattern with an anterior open-bite, negative overjet, posterior crossbite malocclusion. During the mixed dentition, growth modification maneuvers were attempted. Compensatory orthodontic mechanics were then used but did not result in correction. A residual gummy smile with lip incompetence and asymmetric Class III negative overjet malocclusion remained. The patient had undergone septoplasty and tonsillectomy when he was 20 years old in an attempt to improve the airway but without success. At referral to this surgeon, the patient's history confirmed ongoing heavy snoring, difficulty sleeping at night, an inability to breathe through the nose, and excessive daytime fatigue. The patient underwent an attended polysomnogram that confirmed a respiratory disturbance index of 19 events/hour, with desaturations reaching as low as 84%. A continuous positive airway pressure titration study was carried out, and the use of continuous positive airway pressure was attempted; however, as a result of side effects, it was not practical for this patient. A head and neck examination documented a severely deviated septum, enlarged inferior turbinates, an elevated nasal floor, narrow pyriform rims, a soft palate that was close to the pharyngeal wall, and a retropositioned tongue. The tonsils had previously been removed, and the tongue and soft palate were of normal size. **A,** Frontal views in repose before and after surgery. **B,** Frontal views with smile before and after surgery.

• **Figure 26-14, cont'd** The patient hoped to achieve improved breathing during the day, better sleeping at night, and the relief of excessive daytime fatigue. He also desired the correction of his malocclusion to improve swallowing, speech, chewing, and lip closure as well as to enhance his profile and smile aesthetics. He agreed to a comprehensive orthodontic and surgical approach. No extractions were required, and the orthodontic relief of dental compensation was accomplished. The patient's jaw and intranasal procedures included maxillary Le Fort I osteotomy in three segments (horizontal advancement +8 mm, vertical shortening –1.0 mm, dental midline correction, clockwise rotation of 2 mm, and arch expansion) with interpositional graft; bilateral sagittal split osteotomies of the mandible (horizontal advancement +2 mm); osseous genioplasty (vertical shortening and minimal horizontal advancement); and inferior turbinate reduction, septoplasty, and recontouring of the nasal floor. The patient experienced immediate relief of his snoring as well as improvements in quality of sleep, ability to breathe through the nose during the day, and daytime fatigue. An attended polysomnogram was carried out 4 months after surgery and demonstrated the relief of apnea in all sleep positions other than supine. When sleeping supine, mild obstructive sleep apnea remained, but there were no longer any significant desaturations. **C,** Oblique facial views before and after surgery. **D,** Profile views before and after surgery.

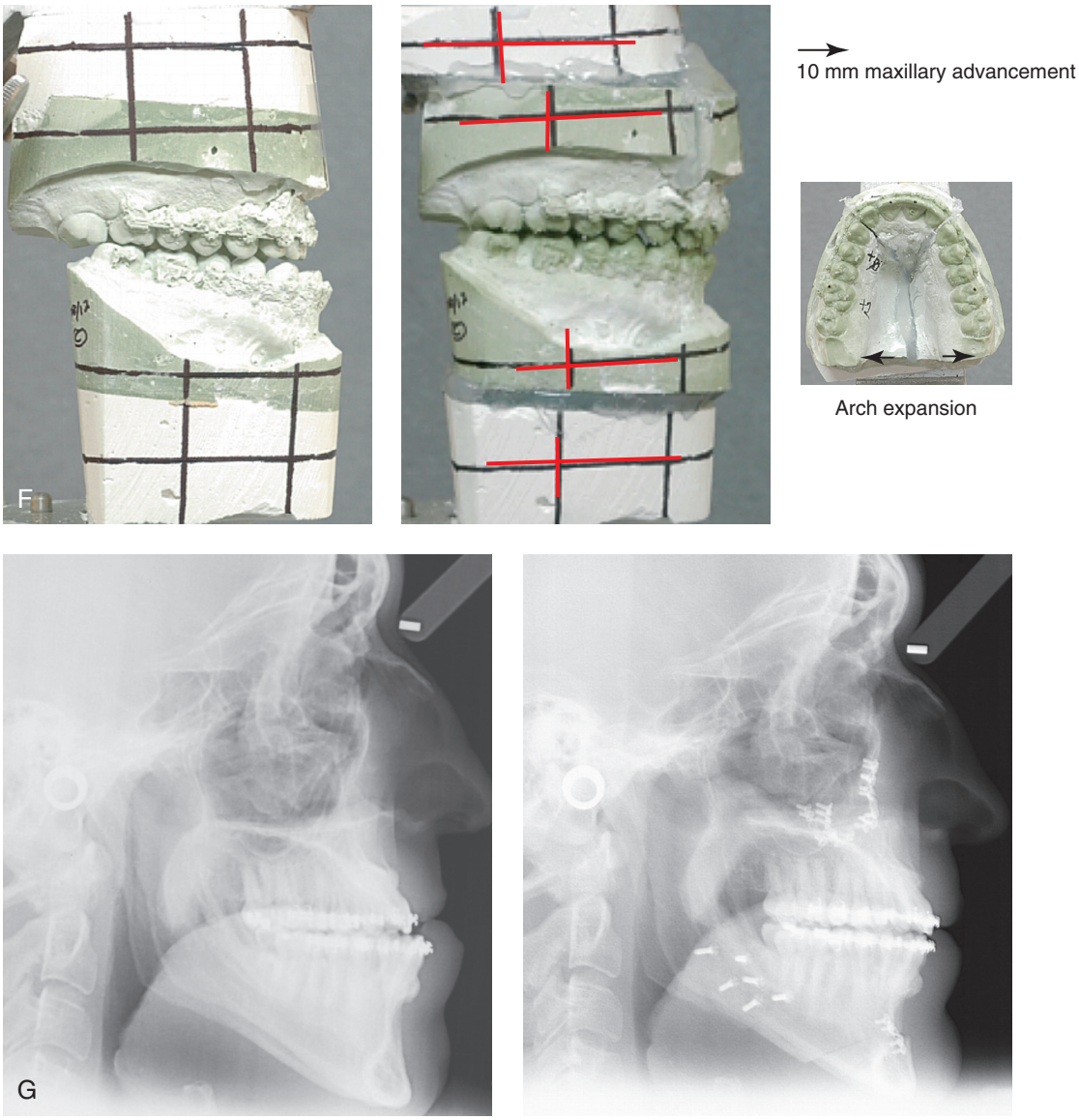
Continued

Prior to retreatment

Pre surgery

After treatment

• **Figure 26-14, cont'd E,** Occlusal views before retreatment, with orthodontic appliances in place, and after treatment.



• **Figure 26-14, cont'd F,** Articulated dental casts that indicate analytic model planning. **G,** Lateral cephalometric radiographs before and after surgery.

with general anesthesia in conjunction with orthognathic and intranasal procedures are expected to be greater among adults with OSA than among teenagers. These risks may include the potential for stress to the cardiovascular, renal, and respiratory systems (see Chapters 11 and 16).

The advantage of a combined intranasal and jaw operation for the individual with OSA is that, like CPAP, such a procedure effectively addresses all of the obstructing upper airway sites simultaneously (i.e., intranasal, retropalatal, and retroglossal). Straightening the septum; reducing the inferior turbinates; widening the maxilla and the piriform rim (i.e., segmental Le Fort I osteotomy), if indicated; and recontouring (deepening) the nasal floor, if indicated—in combination with MMCA—dramatically opens the whole upper airway. Posnick and colleagues have confirmed that jaw and intranasal procedures can be carried out simultaneously without increasing complications.[278]

If the individual requires tonsillectomy, UPPP, or both, then the procedures are carried out separately from the jaw and intranasal operations. This is done because the national reported incidence of significant postsurgical bleeding after UPPP or tonsillectomy is 2% to 4%. In this author's practice, amputation of an elongated uvula (i.e. without the resection of the soft palate) when indicated to further improve the retropalatal airway site is frequently carried out simultaneously with the jaw and intranasal surgery; this is relatively free of risk, and it will avoid the need for a second round of surgery, anesthesia, and recovery.

The Maxillary–Mandibular–Chin Advancement Option

Background

Clinicians now recognize that maxillomandibular deficiency results in reduced pharyngeal airway dimensions and that it can, on its own, lead to or be an additive factor for OSA in both children and adults (see Figs. 26-4 through 26-14).[11,17,154,158,162,184,239,271,285,341,354,363,380,383,390] Mandibular advancement was first suggested in 1979 by West and colleagues as an alternative to tracheostomy for the treatment of OSA.[181] By the early 1980s, Riley and colleagues further documented the value of mandibular advancement surgery that is specifically carried out for the treatment of OSA.[297,306] They advocated simultaneous maxillary and mandibular advancement (MMA) to open the upper airway and to diminish apnea episodes among appropriate individuals with OSA. This surgical approach has consistently been shown to be a highly effective modality for opening the whole upper airway passage for individuals with OSA* (see Figs. 26-4 through 26-14).

Prinsell and colleagues stated the following: "MMA surgery for obstructive sleep apnea syndrome is a highly successful and potentially definitive primary single-stage surgery that may result in a significant reduction in OSA-related health risks, as well as financial savings for the health care system."[284,285] **Li and colleagues** reached the same conclusion and stated the following: "Maxillomandibular advancement surgery is the most effective procedure for OSA."[197] In a surgical update published by the American Association of Oral and Maxillofacial Surgeons, Moore affirmed these conclusions: "Jaw advancement surgery is better tolerated and has a far more significant and beneficial effect than most airway soft tissue surgical procedures [for OSA]. Excellent long-term [5-year] results have been documented with favorable effects on quality of life."[238]

The abundance of published data now confirms that those individuals with documented OSA; anatomic findings of minimal tonsillar tissue and minimal redundant pharyngeal soft tissues; and physical findings of recessive jaws will not sufficiently respond to surgical procedures that are less thorough than MMCA. With today's state of knowledge, a treatment philosophy that suggests that an individual with documented OSA must "fail" Phase I soft-tissue reduction surgery before proceeding to Phase II skeletal expansion surgery" is like saying that a patient with a complex tumor defect must first fail a skin graft before undergoing an effective vascularized flap reconstruction.[209] Although an automatic "soft-tissue first" and "step-wise" surgical approach to OSA made sense during the 1980s, it does not make sense for the 21st century. The soft palate is a dynamic tissue organ with the primary function of preventing the reflux of air and liquids into the nasopharynx during speech and swallowing. Unfortunately, partial surgical ablation or resection of the soft palate (i.e., UPPP) may produce dysfunction, such as velopharyngeal insufficiency, stenosis, voice changes, dysphagia, or silent apnea. In addition, immediate postoperative concerns of pain, hemorrhage, and upper airway obstruction must also be considered. For these reasons, a case can be made for treating the most severe and critical site or area of obstruction first. This will frequently mean performing MMCA initially and then following up later with soft-tissue procedures, when indicated. This skeletal approach will enlarge and stabilize the entire velo-orohypopharyngeal upper airway to definitively treat the OSA and frequently to obviate the need for other soft-tissue procedures (e.g., UPPP, tongue reduction, tonsillectomy).

To review, treatment recommendations for OSA should be made in accordance with the patient's specific anatomic, physiologic, and psychosocial factors. OSA is commonly the result of multiple anatomic deformities that are correctable. The concept of a Phase I soft-tissue approach that is followed by Phase II skeletal procedures as a last resort should be discarded in favor of treating the specific anatomic abnormalities that are contributing to the individual's OSA, preferably during a single operation.[104]

Review of Published Studies concerning Maxillomandibular Advancement Surgery

Critical commentary in the earlier medical literature regarding the spectrum of surgical options may leave sleep physicians and others skeptical of the efficacy of any operative procedure for their patients with OSA. With the use of nasal CPAP as the reference for OSA therapy, a standard for a successful outcome has been established; this standard defines an acceptable compliance rate as 4 hours of CPAP per night for 70% of nights. However, this represents only approximately 50% of sleep time involving a normal airway. Li suggested that an equivalent standard for rating a successful surgical outcome is a 50% reduction in the RDI and fewer than 20 events/hour.[200] If we keep this proposed standard for treatment success in mind, then the literature clearly supports an MMA approach to OSA. Clinical studies that were published between 1989 and 2012 and that were carried out to evaluate the effectiveness of MMA as a treatment approach for OSA are reviewed in the remainder of this section.

Waite and colleagues described 23 consecutive patients with OSA documented by PSG (RDI range, 11 events/hour to 104 events/hour) who underwent MMA in addition to septoplasty and inferior turbinate reduction.[381] Twenty-two of 23 (96%) of the study patients were successfully treated. Successful treatment was based on achieving an RDI of less than 20 events/hour or that represented a more than 50% reduction in RDI. Sixty-five percent of the patients (15/23) achieved an RDI of less than 10 events/hour.

*References 3, 17, 24, 29, 55, 56, 59, 62, 82, 83, 109, 123, 131, 141-145, 149, 177, 179, 196, 197, 199, 200, 203, 208, 214, 222, 225, 227, 251, 274, 279, 283, 284, 287, 299, 300-303, 306, 311, 321, 347, 371, 372, 379, 381, 382, 397

Riley and colleagues reported on 40 patients with OSA documented by PSG who underwent maxillomandibular advancement.[302] Thirty-nine of the 40 study patients (97%) were successfully treated. Successful treatment was based on achieving a postoperative RDI of less than 20 events/hour or a more than 50% reduction in RDI and normal or near normal oxygen saturation. A mean presurgical RDI of 67 events/hour was reduced to 9 events/hour.

Riley and colleagues described 306 consecutively treated surgical patients with PSG-documented OSA.[305] A two-phase surgical protocol was used to reconstruct the upper airway. Phase I surgery consisted of UPPP and genioglossus advancement with hyoid myotomy suspension. Patients for whom Phase I surgery was unsuccessful were offered Phase II reconstruction in the form of maxillomandibular advancement. Surgery was considered successful if the postoperative RDI was less than 20 events/hour with normal oxygenation. Ninety-one patients who failed Phase I treatment entered the Phase II protocol. All patients who underwent Phase II treatment (i.e., maxillomandibular advancement) also had perioperative orthodontics. The success of Phase II therapy was 97% (i.e., RDI <20 events/hour with normal oxygenation).

Hochban and colleagues discussed 38 consecutive patients with OSA documented by PSG (RDI >20 events/hour) who underwent MMA (10-mm maxillary advancement); 37 of 38 of the study patients (95%) were successfully treated.[144] Successful treatment was based on achieving a postoperative AHI of less than 10 events/hour. This clinical series included only healthy, non-obese patients with a documented dentofacial skeletal deformity.

Prinsell reported on 50 consecutive patients with PSG-documented OSA (mean AHI, 59 events/hour) who underwent MMA, with or without adjunctive procedures.[283] All 50 patients were successfully treated. Successful treatment was based on the achievement of a postoperative AHI of 5 events/hour or less or a more than 60% reduction in RDI.

Li and colleagues described 175 patients with PSG-documented OSA (mean RDI, 72 events/hour) who underwent MMA.[200] One hundred sixty-six of the 175 patients (95%) were successfully treated. Successful treatment was based on achieving a postoperative RDI of less than 20 events/hour or a more than 50% reduction in RDI. The mean preoperative RDI of 72 events/hour was reduced to a mean postoperative RDI of 7.2 events/hour. Eighty-six of the 175 patients (49%) had undergone ineffective Phase I surgery before MMA. The cure rate for these patients in this study was 97% (83 out of 86). The mean age of the patients in this study who underwent Phase II treatment (i.e., MMA) was 44 years.

Bettega and colleagues completed a prospective study of a consecutive series of individuals (n = 51) with severe OSA (mean AHI, 59 events/hour).[29] All of these patients underwent surgical therapy. The surgery was considered successful if the postoperative AHI was less than 15 events/hour with at least a 50% reduction from presurgical rates. Forty-four of the patients initially underwent Phase I surgery, and the success rate of the Phase I procedures was only 22.7% (10 of 44 patients). Twenty of the 51 patients underwent MMA, including 13 patients for whom Phase I surgery had failed and 7 who underwent MMA as the primary treatment. On average, the AHI decreased from 59 events/hour to 11 events/hour after the MMA procedure. Overall, 15 of the 20 individuals undergoing MMA (75%) were considered to have successful outcomes.

Li and colleagues studied a consecutive series of 12 individuals with OSA that had been documented by PSG (mean RDI, 75 events/hour) and who had undergone MMA.[197] The authors were able to demonstrate a decrease in airway obstruction and collapse after MMA as documented by lateral cephalometric radiographs and by fiber-optic nasopharyngoscopy during passive respiration. Postoperative fiber-optic nasopharyngoscopy with the Müller maneuver specifically documented a decrease in airway collapsibility; an improvement in the retro displacement of the tongue base; and an improvement in lateral wall stability. The mean preoperative RDI was 75 events/hour, which improved by 6 months after surgery to 10.8 events/hour. Ten of the 12 patients who had been treated with MMA (83%) were felt to be cured (RDI <20 events/hour).

Fairburn and colleagues described 20 consecutive patients with PSG-documented OSA (AHI range, 26 events/hour to 134 events/hour) who had undergone MMA with at least 10 mm of mandibular advancement.[82] Eighteen of the 20 study patients (90%) were successfully treated. Successful treatment was based on the achievement of a significant reduction in the AHI. Ten of the 20 study patients (50%) achieved an AHI of less than 10 events/hour. There was a mean improvement of 60 events/hour. In addition, there was a measured enlargement of the upper airway space (in both the lateral and the anteroposterior dimensions) for all patients at all levels.

Vicini and colleagues completed a prospective, randomized, controlled trial to assess the effectiveness of surgery versus ventilation in adults with documented severe OSA (AHI average of >30 events/hour; range, 31 events/hour to 87 events/hour).[376] Fifty consecutive subjects who had severe OSA were enrolled and randomized into a conservative treatment group (i.e., auto-titrating positive air pressure) or a surgical treatment group (i.e., MMA). One year after MMA surgery or continuous auto-titrating positive air pressure treatment, both groups showed a remarkable improvement of mean AHI and ESS levels. The degree of improvement was not statistically different between the two groups. The MMA group (n = 25) had a mean age of 49 years, with a mean BMI of 32.7 kg/m^2 and a mean preoperative AHI of 56.8 events/hour. After MMA surgery, the mean postoperative AHI dropped to 8.1 events/hour ± 7.0. There were no fatal events, no significant bleeding events, and no complications that required a surgical revision procedure. According to a survey questionnaire, the subject's personal perception of the overall surgical experience was highly satisfactory. Twenty-four of the 25 patients stated they could "go through the operation all over again." MMA

proved to be a valuable alternative therapeutic tool in adults with severe OSA, with a success rate equivalent to that of therapeutic ventilation treatment.

Ronchi and colleagues completed a study to evaluate the effectiveness of MMA in patients with PSG-documented OSA.[311] Two groups with different skeletal patterns were studied. Group I (n = 11) had moderate or severe OSA and cephalometrically documented mandibular deficiency (i.e., a sella–nasion–A-point angle of ≤78 degrees or a sella–nasion–B-point angle of <65 degrees and a Class II malocclusion). Group II (n = 11) had moderate or severe OSA without cephalometrically documented maxillomandibular hypoplasia (i.e., a sella–nasion–A-point angle of >80 degrees and a Class I malocclusion). Preoperative and postoperative data collected included AHI; posterior airway space measurement; sella–nasion–A-point and sella–nasion–B-point angles; ESS scores; body mass index; and a subjective standardized questionnaire about facial aesthetics. Results documented that all patients (Groups I and II) achieved increased posterior airway space and the remission of OSA and that both groups were comparable. All patients were satisfied with the functional and aesthetic results achieved. In Group I, AHI decreased from 57.9 events/hour to 7.0 events/hour after surgery. Five patients reported slight improvement and six reported great improvement in their aesthetic results. In Group II, AHI decreased from 50.7 events/hour before surgery to 7.6 events/hour after surgery. Five patients reported slight improvement, five reported no changed, and one reported great improvement in facial appearance. No patients (Group I or II) reported a worsening of the aesthetic appearance. The study confirms that MMA is effective for patients with moderate to severe OSA and even in those who are considered to have cephalometrically normal skeletal and Angle-classified occlusions.

Holty and Guilleminault completed an extensive meta-analysis of the outcomes of MMA surgery. A total of 627 patients from 42 study populations were identified from a review of 914 reports.[149] The mean age was 44.4 years, and 88% of the study subjects were men. The mean postsurgical follow-up period was 5.3 months, and 67% of patients had previously undergone less invasive OSA treatment procedures without success. The MMA surgical success rate was 86%. As a result of the MMA procedures, the following occurred:

1. BMI decreased from 30.4 kg/m^2 to 29.4 kg/m^2.
2. AHI improved from 63.9 events/hour to 9.5 events/hour.
3. Oxygen nadir improved from 71.9% to 87.7%.
4. AI improved from 34.7 events/hour to an average of 1.6 events/hour.
5. Quality of life assessments documented a 72% absolute reduction in depression and irritability.
6. Systolic blood pressure improved from 139 mm Hg to 124 mm Hg.
7. Improvement in rapid eye movement sleep and Stage III or IV sleep was significant.

Fifty-six patients from three specific studies reported the long-term success rates after MMA procedures. With an average follow-up period of 43.7 months, an 89% success rate was documented. Univariant and multivariant predictors of MMA surgical success were younger age; lower preoperative AHI; lower BMI; and greater degree of maxillary advancement accomplished. Older age (>60 years) and higher BMI (>33 kg/m^2) were negative predictors of a successful outcome. However, according to the study results, this does not mean that even those patients with negative predictors cannot experience significant improvement.

Prinsell completed a literature survey and made treatment recommendations regarding both primary and secondary MMA with or without adjunctive procedures for adults with OSA.[44,285] A critical review of all published studies that included six or more cases per series of MMA and other multilevel surgery in adults that also documented preoperative and postoperative AHI was conducted. The AHI data from the case series were tabulated into categories of surgery from which the mean percent reduction of AHI was calculated and used as the primary measure of PSG therapeutic efficacy. The review of published studies indicates a mean percent reduction in AHI as follows:

- An 88.4% reduction of AHI for primary MMA
- An 86.6% reduction of AHI for secondary MMA
- A 79.4% reduction of AHI for primary MMA with combined intrapharyngeal procedures
- A 92.1% reduction of AHI for primary MMA with combined extrapharyngeal procedures
- A 53% reduction of AHI for non-MMA multilevel surgery
- A 31.3% reduction of AHI for UPPP
- An 89.8% reduction of AHI for nasal CPAP

The authors concluded that both primary and secondary MMA are highly therapeutic because they are extrapharyngeal procedures that open the entire velo-orohypopharyngeal airway via skeletal advancement with minimal risk of postoperative airway compromise.

Goodday and Bourque completed a prospective study to evaluate subjective outcomes and the use of CPAP after MMA surgery for the treatment of OSA. A self-administered questionnaire was completed preoperatively and postoperatively by the study patients (n = 16) who underwent MMA for OSA during a 10-year period from 2000 to 2010. The questionnaire included the ESS as well as questions about snoring, witnessed apneas, CPAP use, and overall individual personal satisfaction. Preoperatively, 40% of patients were very sleepy (ESS scores of ≥16), 32% were sleepy (ESS scores of 10 to 16), and 28% were not sleepy (ESS scores of ≤10). After surgery, only one patient (<1%) was very sleepy, 9% were sleepy, and 90% were not sleepy. Surgery was also found to decrease snoring by 83%, witnessed apneas by 94%, and CPAP use by 96% ($P < .001$). The surgery was judged to be worthwhile by 89% of patients,

and 95% of patients said that they would recommend the treatment to others with OSA.

Conclusions

The public health burden that is attributable to sleep disorders is undisputed, and the derangements that result from these disorders are both behavioral and pathophysiologic. The fundamental problem associated with an OSA event is the inability to maintain upper airway patency during sleep, which then results in a reduction (i.e., hypopnea) or a cessation (i.e., apnea) of breathing. The obstruction can occur at any level of the upper airway. It is critical to diagnose the sites of obstruction so that effective treatment can proceed. For the majority of individuals with moderate to severe OSA, all of the sites are at least partially obstructed. All areas of obstruction will likely require improvement to significantly relieve the individual's apnea events. Achieving and maintaining a normal BMI and avoiding medications that negatively affect oropharyngeal muscle tone are also clear objectives. The concept of soft-tissue (intra pharyngeal) surgery being performed first and only later followed by skeletal (jaw advancement) surgery is antiquated. Contemporary thinking aims to tailor treatment to match each patient's set of anatomic deformities.

Clinical studies confirm that MMCA—often in combination with intranasal procedures (septoplasty, inferior turbinate reduction, and nasal floor recontouring) and limited uvulectomy (if indicated)—is relatively safe and highly effective for the treatment of OSA when multiple sites of upper airway narrowing are present. Published studies consistently document that MMCA is the most effective surgical alternative to tracheostomy, with results that are comparative to the therapeutic efficacy of nasal CPAP.

References

1. Abbott MB, Donnelly LF, Dardzinski BJ, et al: Obstructive sleep apnea: MR imaging volume segmentation analysis. *Radiology* 232(3):889–895, 2004.
2. Aboussouan LS, Golish JA, Wood BG, et al: Dynamic pharyngoscopy in predicting outcome of uvulopalatopharyngoplasty for moderate and severe obstructive sleep apnea. *Chest* 107:946, 1995.
3. Abramson Z, Susarla AM, Lawler M, et al: Three-dimensional computed tomographic airway analysis of patients with obstructive sleep apnea treated by maxillomandibular advancement. *J Oral Maxillofac Surg* 69:677–686, 2011.
4. Abyholm F, D'Antonio L, Davidson Ward SL, et al: Pharyngeal flap and sphincteroplasty for velopharyngeal insufficiency have equal outcome at 1 year postoperatively: Results of a randomized trial. *Cleft Palate Craniofac J* 42(5):501–511, 2005.
5. Accardo JA, Shults J, Leonard MB, et al: Differences in overnight polysomnography scores using the adult and pediatric criteria for respiratory events in adolescents. *Sleep* 33(10):1333–1339, 2010.
6. Achilleos S, Krogstad O, Lyberg T: Surgical mandibular setback and changes in uvuloglossopharyngeal morphology and head posture: A short- and long-term cephalometric study in males. *Eur J Orthod* 22:383–394, 2000.
7. Adams GL, Gansky SA, Miller AJ, et al: Comparison between traditional 2-dimensonal cephalometry and a 3-dimensional approach on human dry skulls. *Am J Orthod Dentofacial Orthop* 126(4):397–409, 2004.
8. Agren K, Nordlander B, Linder-Aronsson S, et al: Children with nocturnal upper airway obstruction: Postoperative orthodontic and respiratory improvement. *Acta Otolaryngol* 118:581–587, 1998.
9. Akashiba T, Kawahara S, Kosaka N, et al: Determinants of chronic hypercapnia in Japanese men with obstructive sleep apnea syndrome. *Chest* 121:415–421, 2002.
10. Alajmi M, Mulgrew AT, Fox J, et al: Impact of continuous positive airway pressure therapy on blood pressure in patients with obstructive sleep apnea. Hypopnea: A meta-analysis of randomized controlled trials. *Lung* 185:67–72, 2007.
11. Al-Saleh A, Riekstins A, Forrest CR, et al: Sleep-related disordered breathing in children with syndromic craniosynostosis. *J Craniomaxillofac Surg* 39:153–157, 2011.
12. American Academy of Pediatrics: Clinical practice guideline: Diagnosis and management of childhood obstructive sleep apnea syndrome. *Pediatrics* 109:704–712, 2002.
13. American Academy of Sleep Medicine (AASM) Task Force: Sleep-related breathing disorders in adults: Recommendations for syndrome definition and measurement techniques in clinical research. *Sleep* 22:667–689, 1999.
14. American Academy of Sleep Medicine: *International classification of sleep disorders, ed 2, Diagnostic and coding manual.* Westchester, Ill, 2005, American Academy of Sleep Medicine, pp 51–55.
15. American Thoracic Society: Standards and indications for cardiopulmonary sleep studies in children. *Am J Respir Crit Care Med* 153:866–878, 1996.
16. American Thoracic Society Workshop Summary: Cardiorespiratory sleep studies in children. Establishment of normative data and polysomnographic predictors of morbidity. *Am J Respir Crit Care Med* 160:1381–1387, 1999.
17. Anderson P, Netherway D, Abbott A, et al: Mandibular lengthening by distraction for airway obstruction in Treacher-Collins syndrome: The long-term results. *J Craniofac Surg* 15:47, 2004.
18. Athanasiou AE, Toutountzakis N, Mavreas D, et al: Alterations of hyoid bone position and pharyngeal depth and their relationship after surgical correction of mandibular prognathism. *Am J Orthod Dentofacial Orthop* 100:259–265, 1991.
19. Avrahami E, Englender M: Relation between CT axial cross-sectional area of the oropharynx and obstructive sleep apnea syndrome in adults. *AJNR Am J Neuroradiol* 16:135, 1995.
20. Bacon WH, Turiot JC, Krieger J, Stierie JL: Cephalometric evaluation of pharyngeal obstructive factors in patients with sleep apnea syndrome. *Angle Orthod* 60:115–122, 1989.
21. Banerjee D, Yee BJ, Piper AJ, et al: Obesity hypoventilation syndrome: Hypoxemia during continuous positive airway pressure. *Chest* 131:1678–1684, 2007.
22. Battagel JM, Lestange PR: The cephalometric morphology of patients with obstructive sleep apnea (OSA). *Eur J Orthod* 18:557–569, 1996.
23. Bazzano LA, Khan Z, Reynolds K, He J: Effect of nocturnal nasal continuous positive airway pressure on blood pressure in obstructive sleep apnea. *Hypertension* 50:417–423, 2007
24. Bear SE, Priest JH: Sleep apnea syndrome: Correction with surgical advancement of the mandible. *J Oral Surg* 38:543–549, 1980.
25. Becker HF, Jerrentrup A, Ploch T, et al: Effect of nasal continuous positive airway pressure treatment on blood pressure in patients with obstructive sleep apnea. *Circulation* 107:68–73, 2003.
26. Beebe DW, Groesz L, Wells C, et al: The neuropsychological effects of obstructive sleep apnea: A meta-analysis of norm-referenced and case-controlled data. *Sleep* 26(3):298–307, 2003.

27. Berretin-Felix G, Yamashita RP, Filho HN, et al: Short- and long-term effect of surgically assisted maxillary expansion on nasal airway size. *J Craniofac Surg* 17:1045–1049, 2006.
28. Borowiecki B, Kukwa A, Blanks RHI, Irvine CA: Cephalometric analysis for diagnosis and treatment of obstructive sleep apnea. *Laryngoscope* 98:226–234, 1988.
29. Bettega G, Pepin JL, Veale D, et al: Obstructive sleep apnea syndrome: Fifty-one consecutive patients treated by maxillofacial surgery. *Am J Respir Crit Care Med* 162:641–649, 2000.
30. Blunden S, Lushington K, Kennedy D, et al: Behavior and neurocognitive performance in children aged 5-10 years who snore compared to controls. *J Clin Exp Neuropsychol* 22:554–568, 2000.
31. Bondenmark L: Does 2 years' nocturnal treatment with mandibular advancement splint in adult patients with snoring and OSAS cause a change in the posture of the mandible? *Am J Orthod Dentofacial Orthop* 116(6):621–628, 1999.
32. Bondenmark L, Lindman R: Craniomandibular status and function in patients with habitual snoring and obstructive sleep apnea after nocturnal treatment with a mandibular advancement splint: A 2-year follow-up. *Eur J Orthod* 22:53–60, 2000.
33. Borowiecki B, Pollak CP, Weitzman ED, et al: Fiberoptic study of pharyngeal airway during sleep in patients with hypersomnia obstructive sleep apnea syndrome. *Laryngoscope* 88:1310–1313, 1978.
34. Brietzke SE, Gallagher D: The effectiveness of tonsillectomy and adenoidectomy in the treatment of pediatric obstructive sleep apnea/hypopnea syndrome: A meta-analysis. *Otolaryngol Head Neck Surg* 134:979–984, 2006.
35. Brietzke SE, Katz ES, Roberson DW: Can history and physical examination reliably diagnose pediatric obstructive sleep apnea/hypopnea syndrome? A systematic review of the literature. *Otolaryngol Head Neck Surg* 131:827–832, 2004.
36. Buccheri A, Dilella G, Stella R: Rapid palatal expansion and pharyngeal space: Cephalometric evaluation. *Prog Orthod* 5(2):160–171, 2004.
37. Buchwald J, Avidor Y, Braunwald E, et al: Bariatric surgery: A systematic review and meta-analysis. *JAMA* 292:1724, 2004.
38. Burstein FD, Cohen SR, Scott PH, et al: Surgical therapy for severe refractory sleep apnea in infants and children: Application of the airway zone concept. *Plast Reconstr Surg* 96:34, 1995.
39. Cadieux RJ, Kales A, McGlynn T, et al: Sleep apnea precipitated by pharyngeal surgery in a patient with myotonic dystrophy. *Arch Otolaryngol* 110(9):611–613, 1984.
40. Cahali MB, Formigoni GG, Gebrim EM, et al: Lateral pharyngoplasty versus uvulopalatopharyngoplasty: A clinical, polysomnographic and computed tomography measurement comparison. *Sleep* 27(5):942–950, 2004.
41. Cakarne D, Urtane I, Skagers A: Pharyngeal airway sagittal dimension in patients with Class III skeletal dentofacial deformity before and after bimaxillary surgery. *Stomatologija* 5:13, 2003.
42. Campo A, Fuhbeck G, Zulueta JJ, et al: Hyperleptinaemia, respiratory drive and hypercapnic response in obese patients. *Eur Respir J* 30:223–231, 2007.
43. Campos-Rodriguez F, Pena-Grinon N, Reyes-Nunez N, et al: Mortality in obstructive sleep apnea-hypopnea patients treated with positive airway pressure. *Chest* 128:624–633, 2005.
44. Caples SM, Rowley JA, Prinsell JR, et al: Surgical modifications of the upper airway for obstructive sleep apnea in adults: A systemic review and meta-analysis. *Sleep* 33:1396, 2010.
45. Carlson D, Onal ME, Carley DW, et al: Palatal muscle electromyogram activity in obstructive sleep apnea. *Am J Respir Crit Care Med* 152:1022–1027, 1995.
46. Carroll J, Loughlin G: Diagnostic criteria for obstructive sleep apnea syndrome in children. *Pediatr Pulmonol* 14:71–74, 1992.
47. Chabolle F, Wagner I, Blumen MB, et al: Tongue base reduction with hyoepiglottoplasty: A treatment for severe obstructive sleep apnea. *Laryngoscope* 109:1273–1280, 1999.
48. Chen F, Terada K, Hua Y, Saito I: Effects of bimaxillary surgery and mandibular setback surgery on pharyngeal airway measurements in patients with Class III skeletal deformities. *Am J Orthod Dentofacial Orthop* 131:372–377, 2007.
49. Chervin RD, Ruzicka DL, Giodani BJ, et al: Sleep-disordered breathing, behavior, and cognition in children before and after adenotonsillectomy. *Pediatrics* 117:769–778, 2006.
50. Chervin RD, Weatherly RA, Garetz SL, et al: Pediatric sleep questionnaire: Prediction of sleep apnea and outcomes. *Arch Otolaryngol Head Neck Surg* 133:216–222, 2007.
51. Cillo JE, Jr, Thayer S, Dasheiff RM, Finn R: Relations between obstructive sleep apnea syndrome and specific cephalometric measurements, body mass index, and apnea-hypopnea index. *J Oral Maxillofac Surg* 70:e278–e283, 2012.
52. Cleator IGM, Birmingham CL, Kovacevic S, et al: Long-term effect of ileogastrostomy surgery for morbid obesity on diabetes mellitus and sleep apnea. *Obes Surg* 16:1337–1341, 2006.
53. Coccagna G, diDonato G, Verucchi R, et al: Hypersomnia with periodic apneas in acquired micrognathia: A bird-like face syndrome. *Arch Neurol* 33:769–776, 1976.
54. Compadretti GC, Tasca I, Bonetti GA: Nasal airway measurements in children treated by rapid maxillary expansion. *Am J Rhinol* 20(4):385–393, 2006.
55. Conley RS, Legan HL: Correction of severe obstructive sleep apnea with bimaxillary transverse distraction osteogenesis and maxillomandibular advancement. *Am J Orthod Dentofacial Orthop* 129(2):283–292, 2006.
56. Conradt R, Hochban W, Brandenburg U, et al: Long-term follow-up after surgical treatment of obstructive sleep apnea by maxillomandibular advancement. *Eur Respir J* 10:123–128, 1997.
57. Contencin P, Guilleminault C, Manach Y: Long-term follow-up and mechanisms of obstructive sleep apnea (OSA) and related syndromes through infancy and childhood. *Int J Pediatr Otorhinolaryngol* 67(Suppl 1):S119–S123, 2003.
58. Conway W, Fujita S, Zohck F, et al: Uvulopalatopharyngoplasty: One year follow-up. *Chest* 88:385, 1985.
59. Costello BJ, Posnick JC: The role of maxillofacial osteotomies in the treatment of obstructive sleep apnea. *Curr Opin Otolaryngol Head Neck Surg* 11(4):267–274, 2003.
60. Crabtree VM, Varni JW, Gozal D: Health-related quality of life and depressive symptoms in children with suspected sleep-disordered breathing. *Sleep* 15;1131–1138, 2004.
61. Crumley RL, Stein S, Ganser G, et al: Determination of obstructive sites in obstructive sleep apnea. *Laryngoscope* 97:301, 1987.
62. Dattilo DJ, Drooger SA: Outcome assessment of patients undergoing maxillofacial procedures for the treatment of sleep apnea: Comparison of subjective and objective results. *J Oral Maxillofac Surg* 62(2):164–168, 2004.
63. Davies RJ, Stradling JR: The relationship between neck circumference, radiographic pharyngeal anatomy, and the obstructive sleep apnea syndrome. *Eur Respir J* 3:509–514, 1990.
64. De Berry-Borowieki B, Kukwa A, Banks R: Cephalometric analysis for diagnosis and treatment of obstructive sleep apnea. *Laryngoscope* 98:226, 1988.
65. De Lucas-Ramos P, de Miguel-Diez J, Santacruz-Siminiani A, et al: Benefits at 1 year of nocturnal intermittent positive pressure ventilation in patients with obesity hypoventilation syndrome. *Respir Med* 98:961–967, 2004.
66. De Miguel Diez J, de Lucas Ramos P, Perez Parra JJ, et al: Analysis of withdrawal from noninvasive mechanical ventilation in patients with obesity-hypoventilation syndrome. Medium term results [Spanish]. *Arch Bronconeumol* 39:292–297, 2003.
67. Degerliyurt K, Ueki K, Hashiba Y, et al: A comparative CT evaluation of pharyngeal airway changes in class III patients receiving bimaxillary surgery or mandibular setback

surgery. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 105:495–502, 2008.
68. Degerliyurt K, Ueki K, Hashiba Y, et al: The effect of mandibular setback or two-jaws surgery on pharyngeal airway among different genders. *Int J Oral Maxillofac Surg* 38:647–652, 2009.
69. Demetriades N, Chang DJ, Laskarides C, Papageorge M: Effects of mandibular retropositioning, with or without maxillary advancement, on the oro-naso-pharyngeal airway and development of sleep-related breathing disorders. *J Oral Maxillofac Surg* 68:2431–2436, 2010.
70. Dempsey JA, Skatrud JB, Jacques AJ, et al: Anatomic determinants of sleep disordered breathing across the spectrum of clinical and nonclinical male subjects. *Chest* 122:840–851, 2002.
71. Douglas NJ, Engleman HM: Effects of CPAP on vigilance and related functions in patients with the sleep apnea/hypopnea syndrome. *Sleep* 23(Suppl 4):S147–S149, 2000.
72. Eckert DJ, Malhotra A: Pathophysiology of adult obstructive sleep apnea. *Proc Am Thorac Soc* 5:144–153, 2008.
73. Edström L, Larsson H, Larsson L: Neurogenic effects on the palatopharyngeal muscle in patients with obstructive sleep apnea: A muscle biopsy study. *J Neurol Neurosurg Psychiatry* 55:916–920, 1992.
74. Eggensperger N, Smolka W, Iizuka T: Long-term changes of hyoid bone position and pharyngeal airway size following mandibular setback by sagittal split ramus osteotomy. *J Craniomaxillofac Surg* 33:111–117, 2005.
75. El AS, El H, Palomo JM, Baur DA: A 3-dimensional airway analysis of an obstructive sleep apnea surgical correction with cone beam computed tomography. *J Oral Maxillofac Surg* 69:2424–2436, 2011.
76. Elshaug AG, Moss JR, Southcott AM, et al: An analysis of the evidence-practice continuum: Is surgery for obstructive sleep apnea contraindicated? *J Eval Clin Pract* 13:3, 2007.
77. Elshaug AG, Moss JR, Southcott AM, et al: Redefining success in airway surgery for obstructive sleep apnea: A meta analysis and synthesis of the evidence. *Sleep* 30:461, 2007.
78. Enacar A, Aksoy AU, Sençift Y, et al: Changes in hypopharyngeal airway space and in tongue and hyoid bone positions following the surgical correction of mandibular prognathism. *Int J Adult Orthodon Orthognath Surg* 9:285–290, 1994.
79. Enoki C, Valera FC, Lessa FC, et al: Effect of rapid maxillary expansion on the dimension of the nasal cavity and on nasal air resistance. *Int J Pediatr Otorhinolaryngol* 70:1225–1230, 2006.
80. Epstein LJ, Kristo D, Strollo PJ, et al: Clinical guidelines for the evaluation, management, and long-term care of obstructive sleep apnea in adults. *J Clin Sleep Med* 5:263, 2009.
81. Esclamado RM, Glenn MG, McCulloch TM, Cummings CW: Perioperative complications and risk factors in the surgical treatment of obstructive sleep apnea syndrome. *Laryngoscope* 99:1125–1129, 1989.
82. Fairburn SC, Waite PD, Vilos G, et al: Three-dimensional changes in upper airways of patients with obstructive sleep apnea following maxillomandibular advancement. *J Oral Maxillofac Surg* 65:6–12, 2007.
83. Farole A, Mundenar MJ, Braitman LE: Posterior airway changes associated with mandibular advancement surgery: Implications for patients with obstructive sleep apnea. *Int J Adult Orthodon Orthognath Surg* 5:255–258, 1990.
84. Fengshan C, Terada K, Hua Y, et al: Effects of bimaxillary surgery and mandibular setback surgery on pharyngeal airway measurements in patients with Class III skeletal deformities. *Am J Orthod Dentofacial Orthop* 131:372, 2007.
85. Findley LJ, Unverzagt ME, Suratt PM: Automobile accidents involving patients with obstructive sleep apnea. *Am Rev Respir Dis* 138:337–340, 1988.
86. Flemons WW, Reimer MA: Development of a disease-specific health-related quality-of-life questionnaire for sleep apnea. *Am J Respir Crit Care Med* 158:494–503, 1998.
87. Fogel R, Mallhotra A, Pillar G, et al: Genioglossal activation in patients with obstructive sleep apnea versus control subjects. Mechanisms of muscle control. *Am J Respir Crit Care Med* 164:2025–2030, 2001.
88. Foster GD, Wadden TA, Mullen JL, et al: Resting energy expenditure, body composition, and excess weight in the obese. *Metabolism* 37:467–472, 1988.
89. Friberg D, Ansved T, Borg K, et al: Histological indications of a progressive snorers disease in an upper-airway muscle. *Am J Respir Crit Care Med* 157:586–593, 1998.
90. Friberg D, Gazelius B, Holfelt T, et al: Abnormal afferent nerve endings in the soft palatal mucosa of sleep apneics and habitual snorers. *Regul Pept* 71:29–36, 1997.
91. Friberg D, Gazelius B, Linblad LE, et al: Habitual snorers and sleep apneics have abnormal vascular reaction of the soft palatal mucosa or afferent nerve stimulation. *Laryngoscope* 108:431–436, 1998.
92. Friedman M, Ibrahim H, Bass I: Clinical staging for sleep-disordered breathing. *Otolaryngol Head Neck Surg* 127(1):13–21, 2002.
93. Friedman M, Ibrahim H, Lee G, Joseph NJ: Combined uvulopalatopharyngoplasty and radiofrequency tongue base reduction for treatment of obstructive sleep apnea/hypopnea syndrome. *Otolaryngol Head Neck Surg* 129:611–621, 2003.
94. Friedman M, Tanyeri H, Lim JW, et al: Effect of improved nasal breathing on obstructive sleep apnea. *Otolaryngol Head Neck Surg* 122(1):71–74, 2000.
95. Friedman M, Vidyasagar R, Bliznikas D, et al: Does severity of obstructive sleep apnea/hypopnea syndrome predict uvulopalatopharyngoplasty outcome? *Laryngoscope* 115(12):2109–2113, 2005.
96. Fritsch KM, Iseli A, Russi EW, Bloch KE: Side effects of mandibular advancement devices for sleep apnea treatment. *Am J Respir Crit Care Med* 164:813–818, 2001.
97. Fritscher LG, Mottin CC, Canani S, Chatkin JM: Obesity and obstructive sleep apnea-hypopnea syndrome: the impact of bariatric surgery. *Obes Surg* 17:95–99, 2007.
98. Fujita S: Pharyngeal surgery for obstructive sleep apnea. In Fairbanks DNF, Fujita S, Ikermatsu T, et al, editors: *Snoring and obstructive sleep apnea*, New York, 1987, Raven Press, pp 101–128.
99. Fujita S, Conway W, Zorich F, Roth T: Surgical correction of anatomic abnormalities in obstructive sleep apnea syndrome: Uvulopalatopharyngoplasty. *Otolaryngol Head Neck Surg* 89:923–934, 1981.
100. Gale SD, Hopkins RO: Effects of hypoxia on the brain: Neuroimaging and neuropsychological findings following carbon monoxide poisoning and obstructive sleep apnea. *J Int Neuropsychol Soc* 10:60–71, 2004.
101. Galvin JR, Rooholamini SA, Stanford W: Obstructive sleep apnea: Diagnosis with ultrafast CT. *Radiology* 171:775–778, 1989.
102. Gastaut H, Tassinari CA, Duron B: Polygraphic study of the episodic diurnal and nocturnal (hypnic and respiratory) manifestations of the Pickwick syndrome. *Brain Res* 1:167–186, 1967.
103. George CFP, Smiley A: Sleep apnea and automobile crashes. *Sleep* 22:790–795, 1999.
104. Giarda M, Brucoli M, Arcuri F, et al: Proposal of presurgical algorithm for patients affected by obstructive sleep apnea syndrome. *J Oral Maxillofac Surg* 70:2433–2439, 2012
105. Giles TL, Lasserson TJ, Smith BH, et al: Continuous positive airways pressure for obstructive sleep apnea in adults. *Cochrane Database Syst Rev* (3):CD001106, 2006.
106. Gislason T, Almqvist M, Eriksson G, et al: Prevalence of sleep apnea syndrome among Swedish men—an epidemiological study. *J Clin Epidemiol* 41:571–576, 1988.
107. Gold AR, Schwartz AR: The pharyngeal critical pressure: The whys and hows of using nasal continuous positive airway pressure diagnostically. *Chest* 110:1077, 1996.
108. Goldstein NA, Pugazhendhi V, Rao SM, et al: Clinical assessment of pediatric obstructive sleep apnea. *Pediatrics* 114:33–43, 2004.
109. Goncalves JR, Buschang PH, Goncalves DG, et al: Postsurgical stability of oropharyngeal airway changes following counterclockwise

maxillomandibular advancement surgery. *J Oral Maxillofac Surg* 64:755–762, 2006.
110. Goodwin JL, Kaemingk KL, Fregosi RF, et al: Clinical outcomes associated with sleep-disordered breathing in Caucasian and Hispanic children—the Tucson Children's Assessment of Sleep Apnea study (TuCASA). *Sleep* 26:587–591, 2003.
111. Gottlieb DJ, Whitney CW, Bonekat WH, et al: Relation of sleepiness to respiratory disturbance index: The Sleep Heart Health study. *Am J Respir Crit Care Med* 159:502, 1999.
112. Gozal D: Sleep-disordered breathing and school performance in children. *Pediatrics* 102(3 Pt 1):616–620, 1998.
113. Greco JM, Frohberg U, Van Sickels JE: Long-term airway space changes after mandibular setback using bilateral sagittal split osteotomy. *Int J Oral Maxillofac Surg* 19:103–105, 1990.
114. Grote L, Hedner J, Grunstein R, et al: Therapy with nCPAP: incomplete elimination of sleep related breathing disorder. *Eur Respir J* 16:921, 2000.
115. Gu G, Gu G, Nagata J, et al: Hyoid position, pharyngeal airway and head posture in relation to relapse after the mandibular setback in skeletal Class III. *Clin Orthod Res* 3:67–77, 2000.
116. Guardiano SA, Scott JA, Ware JC, Schechner SA: The long-term results of gastric bypass on indexes of sleep apnea. *Chest* 124:1615–1619, 2003
117. Guilleminault C, Chowdhury S: Upper airway resistance syndrome is a distinct syndrome. *Am J Respir Crit Care Med* 161:1412–1413, 2000.
118. Guilleminault C, Connolly S, Winkle R, et al: Cyclical variation of the heart rate in sleep apnea syndrome: Mechanisms and usefulness of 24 h electrocardiography as a screening technique. *Lancet* 1:126–131, 1984.
119. Guilleminault C, Hayes B, Smith FB: Palatopharyngoplasty and obstructive sleep apnea: A review of 35 cases. *Bull Eur Physiopathol Respir* 19:595, 1983.
120. Guilleminault C, Hill MW, Simmons FB: Obstructive sleep apnea: Electromyelographic and fiberoptic studies. *Exp Neurol* 62:48, 1978.
121. Guilleminault C, Huang YS, Glamann C, Chan A: Adenotonsillectomy and obstructive sleep apnea in children: A prospective survey. *Otolaryngol Head Neck Surg* 136:169–175, 2007.
122. Guilleminault C, Li K, Chen NH, Poyares D: Two-point palatal discrimination in patients with upper airway resistance syndrome, obstructive sleep apnea syndrome, and normal control subjects. *Chest* 122:866–870, 2002.
123. Guilleminault C, Li KK: Maxillomandibular expansion for the treatment of sleep-disordered breathing: Preliminary result. *Laryngoscope* 114(5):893–896, 2004.
124. Guilleminault C, Li KK, Quo S, et al: A prospective study on the surgical outcome of children with sleep-disordered breathing. *Sleep* 27(1):95–100, 2004.
125. Guilleminault C, Motta J, Mihm F, Melvin K: Obstructive sleep apnea and cardiac index. *Chest* 89:331–334, 1986.
126. Guilleminault C, Partinen M, Praud JP, et al: Morphometric facial changes and obstructive sleep apnea in adolescents. *J Pediatr* 114:997–999, 1989.
127. Guilleminault C, Partinen M, Praud JP, et al: Can history and physical examination reliably diagnose pediatric obstructive sleep apnea/hypopnea syndrome? A systematic review of the literature. *Otolaryngol Head Neck Surg* 131:827–832, 2004.
128. Guilleminault C, Poyares D, Palombini L, et al: Variability of respiratory effort in relation to sleep stages in normal controls and upper airway resistance syndrome patients. *Sleep Med* 2:397–406, 2001.
129. Guilleminault C, Tilkian AG, Dement WC: Sleep apnea syndromes. *Annu Rev Med* 27:465–484, 1976.
130. Guilleminault C, Tilkian AC, Eldridge FL: Sleep apnea syndrome upper airway obstruction: A review of 25 cases. *Arch Intern Med* 137:296, 1977.
131. Guven O, Saracoglu U: Changes in pharyngeal airway space and hyoid bone positions after body ostectomies and sagittal split ramus osteotomies. *J Craniofac Surg* 16:23–30, 2005.
132. Hack M, Davies RJ, Mullins R, et al: Randomized prospective parallel trial of therapeutic versus subtherapeutic nasal continuous positive airway pressure on simulated steering performance in patients with obstructive sleep apnoea. *Thorax* 55:224, 2000.
133. Haentjens P, Meerhaeghe AV, Moscariello A, et al: The impact of continuous positive airway pressure on blood pressure in patients with obstructive sleep apnea syndrome. Evidence from a meta-analysis of placebo-controlled randomized trials. *Arch Intern Med* 167(8):757–765, 2007
134. Haines KL, Nelson LG, Gonzalez R, et al: Objective evidence that bariatric surgery improves obesity-related obstructive sleep apnea. *Surgery* 141:354–358, 2007
135. Hans MG, Goldberg J: Cephalometric exam in obstructive sleep apnea. In Waite PD, editor: Oral and maxillofacial treatment of obstructive sleep apnea. *Oral Maxillofac Surg Clin North Am* 7:209–281, 1995.
136. Haponik EF, Smith PL, Bohlman ME, et al: Computerized tomography in obstructive sleep apnea. *Am Rev Respir Dis* 127:221, 1983.
137. Hatcher DC: Cone beam computed tomography: Craniofacial and airway analysis. *Sleep Med Clin* 5:59–70, 2010.
138. He J, Kryger MH, Zorick FJ, et al: Mortality and apnea index in obstructive sleep apnea: Experience in 385 male patients. *Chest* 94:9–14, 1988.
139. Hendler BH, Costello BJ, Silverstein K, et al: A protocol for uvulopalatopharyngoplasty, mortised genioplasty, and maxillomandibular advancement in patients with obstructive sleep apnea: An analysis of 40 cases. *J Oral Maxillofac Surg* 59:892, 2001.
140. Hla KM, Young TB, Bidwell T, et al: Sleep apnea and hypertension: A population-based study. *Ann Intern Med* 120:382–388, 1994.
141. Hochban W: Surgical treatment of obstructive sleep apnea. *Otorhinolaryngol Nova* 10:149–156, 2000.
142. Hochban W, Brandenburg U: Morphology of the viscerocranium in obstructive sleep apnea syndrome—cephalometric evaluation of 400 patients. *J Craniomaxillofac Surg* 22:205, 1994.
143. Hochban W, Brandenburg U, Peter JH: Surgical treatment of obstructive sleep apnea by maxillomandibular advancement. *Sleep* 17(7):624–629, 1994.
144. Hochban W, Conradt R, Brandenburg U, et al: Surgical maxillofacial treatment of obstructive sleep apnea. *Plast Reconstr Surg* 99:619–626, 1997.
145. Hochban W, Schurmann R, Brandenburg U, Conradt R: Mandibular setback for surgical correction of mandibular hyperplasia: Does it provoke sleep-related breathing disorders? *Int J Oral Maxillofac Surg* 25:333–338, 1996.
146. Hoffman EA, Gefter WB: Multimodality imaging of the upper airway: MRI, MR spectroscopy, and ultrafast X-ray CT. *Prog Clin Biol Res* 345:291–301, 1990.
147. Hoffstein V, Mateika S: Differences in abdominal and neck circumferences in patients with and without obstructive sleep apnea. *Eur Respir J* 5:377–381, 1992
148. Hoffstein V, Viner S, Mateika S, Conway J: Treatment of obstructive sleep apnea with nasal continuous positive airway pressure: Patient compliance, perception of benefits and side effects. *Am Rev Respir Dis* 145:841–845, 1992.
149. Holty JEC, Guilleminault C: Maxillomandibular advancement for the treatment of obstructive sleep apnea: A systematic review and meta-analysis. *Sleep Med Rev* 14:287, 2010.
150. Horne J: *Why we sleep: The functions of sleep in humans and other mammals*. Oxford, UK, 1988, Oxford University Press, p 216.
151. Hsu PP, Tan BY, Chan YH, et al: Clinical predictors in obstructive sleep apnea patients with computer-assisted quantitative videoendoscopic upper airway analysis. *Laryngoscope* 114(5):791–799, 2004.
152. Huang YS, Guilleminault C, Li HY, et al: Attention-deficit/hyperactivity disorder with obstructive sleep apnea: A treatment outcome study. *Sleep Med* 8:18–30, 2007.
153. Hung J, Whitford EG, Parsons RW, Hillman DR: Association of sleep apnoea with myocardial infarction in men. *Lancet* 336:261–264, 1990.
154. Iatrou I, Theologie-Lygidakis N, Schoinohoriti O: Mandibular distraction

osteogenesis for severe airway obstruction in Robin sequence: Case report. *J Craniomaxillofac Surg* 38:431–435, 2010.
155. Iber C, Ancoli-Israel S, Chesson A, et al: *The AASM manual for the scoring of sleep and associated events: Rules, terminology and technical specifications*, Westchester, Ill, 2007, American Academy of Sleep Medicine.
156. Ikeda K, Ogura M, Oshima T, et al: Quantitative assessment of the pharyngeal airway by dynamic magnetic resonance imaging in obstructive sleep apnea syndrome. *Ann Otol Rhinol Laryngol* 110:183–189, 2001.
157. Jackson IT, Silverton J: The sphincter pharyngoplasty as a secondary procedure in cleft palates. *Plast Reconstr Surg* 59(4):518–524, 1977.
158. Jamieson A, Guilleminault C, Partinen M, et al: Obstructive sleep apneic patients have craniomandibular abnormalities. *Sleep* 9:469, 1986.
159. Jenkinson C, Davies RJ, Mullins R, et al: Comparison of therapeutic and subtherapeutic nasal continuous positive airway pressure for obstructive sleep apnoea: A randomized prospective parallel trial. *Lancet* 353:2100, 1999.
160. Jenkinson C, Stradling J, Petersen S: Comparison of three measures of quality of life outcome in the evaluation of continuous positive airway pressure therapy for sleep apnea. *J Sleep Res* 6:199–204, 1997.
161. Jennum P, Hein HO, Suadicani P, Gyntelberg F: Cognitive functioning and snoring. *Sleep* 16:562–564, 1993.
162. Johns FR, Strollo PJ, Jr, Buckley M, et al: The influence of craniofacial structure on obstructive sleep apnea in young adults. *J Oral Maxillofac Surg* 56:596, 1998.
163. Johns MW: A new method for measuring daytime sleepiness: The Epworth sleepiness scale. *Sleep* 14:50–55, 1991.
164. Johns MW: Reliability and factor analysis of the Epworth sleepiness scale. *Sleep* 15:376–381, 1992.
165. Jung R, Kuhlo W: Neurophysiological studies of abnormal night sleep and the Pickwickian syndrome. *Prog Brain Res* 18:140–159, 1965.
166. Kaemingk KL, Pasvogel AE, Goodwin JL, et al: Learning in children and sleep disordered breathing: Findings of the Tucson Children's Assessment of Sleep Apnea (TuCASA) prospective cohort study. *J Int Neuropsychol Soc* 1016–1026, 2003.
167. Kajaste S, Telakivi T, Mustajoki P, et al: Effects of a cognitive-behavioural weight loss programme on overweight obstructive sleep apnoea patients. *J Sleep Res* 3:245–249, 1994.
168. Katsantonis GR Maas CS, Walsh JK: The predictive efficacy of the Mueller maneuver in uvulopalatopharyngoplasty. *Laryngoscope* 99:677–680, 1989.
169. Katz I, Stradling J, Slutsky AS, et al: Do patients with obstructive sleep apnea have thick necks? *Am Rev Respir Dis* 141:1228–1231, 1990.
170. Katz ES, D'Ambrosio CM: Pathophysiology of pediatric obstructive sleep apnea. *Proc Am Thorac Soc* 5:253–262, 2008.
171. Kawakami M, Yamamoto K, Fujimoto M, et al: Changes in tongue and hyoid positions, and posterior airway space following mandibular setback surgery. *J Craniomaxillofac Surg* 33:107–110, 2005.
172. Kawamata A, Fujishita M, Ariji Y, Ariji E: Three-dimensional computed tomographic evaluation of morphologic airway changes after mandibular setback osteotomy for prognathism. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 89:278–287, 2000.
173. Kezirian EJ, Weaver EM, Yueh B, et al: Incidence of serious complications after uvulopalatopharyngoplasty. *Laryngoscope* 114(3):450–453, 2004.
174. Kitagawara K, Kobayashi T, Goto H, et al: Effects of mandibular setback surgery on oropharyngeal airway and arterial oxygen saturation. *Int J Oral Maxillofac Surg* 37:328–333, 2008.
175. Koskenvuo M, Kaprio J, Telakivi T, et al: Snoring as a risk factor for ischaemic heart disease and stroke in men. *BMJ* 294:16–19, 1987.
176. Kravath RE, Pollak C, Borowiecki B, Weitzman E: Obstructive sleep apnea and death associated with surgical correction of velopharyngeal incompetence. *J Pediatr* 96(4):645–648, 1980.
177. Krekmanov L, Andersson L, Ringqvist M, et al: Anterior-inferior mandibular osteotomy in treatment of obstructive sleep apnea syndrome. *Int J Adult Orthodon Orthognath Surg* 13:289–297, 1998.
178. Kribbs NB, Redline S, Smith PL, et al: Objective monitoring of nasal CPAP usage in OSAS patients. *J Sleep Res* 20:270, 1991.
179. Kuo PC, West RA, Bloomquist DS, McNeil RW: The effect of mandibular osteotomy in three patients with hypersomnia sleep apnea. *Oral Surg Oral Med Oral Pathol* 48:385–392, 1979.
180. Kushida CA, Littner MR, Morgenthaler T, et al: Practice parameters for the indications for polysomnography and related procedures: An update for 2005. *Sleep* 28(4):499–521, 2005.
181. Kushida CA, Morgenthaler T, Littner MR, et al: Practice parameters for the treatment of snoring and obstructive sleep apnea with oral appliances: An update for 2005. *Sleep* 29:240, 2006.
182. Laks L, Krieger J, Podszus T: Pulmonary hypertension in obstructive sleep apnea: Retrospective multicentre study. *Am Rev Respir Dis* 145:A865, 1992.
183. Lalakea ML, Marquez-Biggs I, Messner AH: Safety of pediatric short-stay tonsillectomy. *Arch Otolaryngol Head Neck Surg* 125:749–752, 1999.
184. Lam B, Ip MSM, Tench E, et al: Craniofacial profile in Asian and white subjects with obstructive sleep apnea. *Thorax* 60:504–510, 2005.
185. Lavie P: Incidence of sleep apnea in a presumably healthy working population: A significant relationship with excessive daytime sleepiness. *Sleep* 6:312–318, 1983.
186. Lee NR, Givens CD, Jr, Wilson J, Robins RB: Staged surgical treatment of obstructive sleep apnea syndrome. *J Oral Maxillofac Surg* 57:382–385, 1999.
187. Lesavoy MA, Borud L, Thorson T, et al: Upper airway obstruction after pharyngeal flap surgery. *Ann Plast Surg* 36(1):26–30, 1996.
188. Li AM, Hui S, Wong E, et al: Obstructive sleep apnea in children with adenotonsillar hypertrophy: Prospective study. *Hong Kong Med J* 7:236–240, 2001.
189. Li HY, Chen NH, Shu YH, et al: Changes in quality of life and respiratory disturbance after extended uvulopalatal flap surgery in patients with obstructive sleep apnea. *Arch Otolaryngol Head Neck Surg* 130(2):195–200, 2004.
190. Li HY, Chen NH, Wang CR, et al: Use of 3-dimensional computed tomography scan to evaluate upper airway patency for patients undergoing sleep-disordered breathing surgery. *Otolaryngol Head Neck Surg* 129(4):336–342, 2003.
191. Li HY, Lin Y, Chen NH, et al: Improvement in quality of life after nasal surgery alone for patients with obstructive sleep apnea and nasal obstruction. *Arch Otolaryngol Head Neck Surg* 134(4):429–433, 2008.
192. Li HY, Wang PC, Lee LA, et al: Prediction of uvulopalatopharyngoplasty outcome: Anatomy-based staging system versus severity-based staging system. *Sleep* 29(12):1534–1541, 2006.
193. Li KK: Surgical management of obstructive sleep apnea. *Clin Chest Med* 24:365, 2003.
194. Li KK: Surgical therapy for adult obstructive sleep apnea. *Sleep Med Rev* 9:201–209, 2005.
195. Li KK: Surgical therapy for obstructive sleep apnea syndrome. *Semin Respir Crit Care Med* 26:80, 2005.
196. Li KK: Maxillomandibular advancement for obstructive sleep apnea. *J Oral Maxillofac Surg* 69:687–694, 2011.
197. Li KK, Guilleminault C, Riley RW, Powell NB: Obstructive sleep apnea and maxillomandibular advancement: An assessment of airway changes using radiographic and nasopharyngoscopic examinations. *J Oral Maxillofac Surg* 60:526–530, 2002.
198. Li KK, Powell NB, Riley RW, et al: Morbidly obese patients with severe obstructive sleep apnea: Is airway reconstructive surgery a viable treatment option? *Laryngoscope* 110:982, 2000.
199. Li KK, Powell NB, Riley RW, et al: Long-term results of maxillomandibular advancement surgery. *Sleep Breath* 4:137–140, 2000.
200. Li KK, Riley RW, Powell NB, et al: Overview of phase II surgery for obstructive

sleep apnea syndrome. *Ear Nose Throat J* 78:854, 1999.
201. Li KK, Riley RW, Powell NB, et al: Maxillomandibular advancement for persistent OSA after phase I surgery in patients without maxillomandibular deficiency. *Laryngoscope* 110:1684, 2000.
202. Li KK, Riley RW, Powell NB, et al: Obstructive sleep apnea surgery: Patients' perspective and polysomnographic results. *Otolaryngol Head Neck Surg* 123:572, 2000.
203. Li KK, Riley RW, Powell NB, Guilleminault C: Patient's perception of the facial appearance after maxillomandibular advancement for obstructive sleep apnea syndrome. *J Oral Maxillofac Surg* 59:377–380, 2001.
204. Li KK, Troell RJ, Riley RW, et al: Uvulopalatopharyngoplasty, maxillomandibular advancement, and the velopharynx. *Laryngoscope* 111:1075, 2001.
205. Liao YF, Chuang M, Chen P, et al: Incidence and severity of obstructive sleep apnea following pharyngeal flap surgery in patients with cleft palate. *Cleft Palate Craniofac J* 39(3):312–316, 2002.
206. Liu Y, Lowe AA, Zeng X, et al: Cephalometric comparisons between Chinese and Caucasian patients with obstructive sleep apnea. *Am J Orthod Dentofacial Orthop* 117:479–485, 2000.
207. Lojander J, Maasilta P, Partinen M, et al: Nasal CPAP, surgery, and conservative management for treatment of obstructive sleep apnea syndrome: A randomized study. *Chest* 110:114–119, 1996.
208. Louis PJ, Austin RB, Waite PD, et al: Soft tissue changes of the upper lip associated with maxillary advancement in obstructive sleep apnea patients. *J Oral Maxillofac Surg* 59:151–156, 2001.
209. Lowe AA, Gionhaku N, Takeuchi K, Fleetham JA: Three-dimensional CT reconstructions of tongue and airway in adult subjects with obstructive sleep apnea. *Am J Orthod* 90:364–374, 1986.
210. Lowe AA, Fleetham JA, Adachi S, Ryan F: Cephalometric and computed tomographic predictors of obstructive sleep apnea severity. *Am J Orthod Dentofacial Orthop* 107:589–595, 1995.
211. Lowe AA, Ozbek MM, Miyamoto K, et al: Cephalometric and demographic characteristics of obstructive sleep apnea: An evaluation with partial least square analysis. *Angle Orthod* 67:143–154, 1997.
212. Lowe AA, Santamaria JD, Fleetham JA, Price C: Facial morphology and obstructive sleep apnea. *Am J Orthod Dentofacial Orthop* 90:484–491, 1986.
213. Lowe AA, Sjoholm T, Ryan C, et al: Treatment, airway and compliance effects of a titratbale oral appliance. *Sleep* 23:S172, 2000.
214. Lye KW, Waite PD, Meara D, Wang D: Quality of life evaluation of maxillomandibular advancement surgery for treatment of obstructive sleep apnea. *J Oral Maxillofac Surg* 66:968–972, 2008.
215. Malkoç S, Usümez S, Iseri H: Long-term effects of symphyseal distraction and rapid maxillary expansion on pharyngeal airway dimensions, tongue, and hyoid position. *Am J Orthod Dentofacial Orthop* 132:769–775, 2007.
216. Marcus CL, Omlin KJ, Basinski DJ, et al: Normal polysomnographic values for children and adolescents. *Am Rev Respir Dis* 146:1235–1239, 1992.
217. Marin JM, Carrizo SJ, Vicente E, et al: Long-term cardiovascular outcomes in men with obstructive sleep apnea-hypopnea with or without treatment with continuous positive airway pressure: An observational study. *Lancet* 365:1046–1053, 2005.
218. Marklund M, Stenlund H, Franklin KA: Mandibular advancement devices in 630 men and women with obstructive sleep apnea and snoring-tolerability and predictors of treatment success. *Chest* 125:1270, 2004.
219. Mather R, Douglas NJ: Family studies in patients with the sleep apnea-hypopnea syndrome. *Ann Intern Med* 122:174–178, 1995.
220. McColley SA, April MM, Carroll JL, et al: Respiratory compromise after adenotonsillectomy in children with obstructive sleep apnea. *Arch Otolaryngol Head Neck Surg* 118:940–943, 1992.
221. McNicholas WT, Bonsignore MR: Sleep apnoea as an independent risk factor for cardiovascular disease: Current evidence, basic mechanisms and research priorities. *Eur Respir J* 29:156–178, 2007.
222. Mehra P, Downie M, Pita MC, et al: Pharyngeal airway space changes after counterclockwise rotation of the maxillomandibular complex. *Am J Orthod Dentofacial Orthop* 120:154–159, 2001.
223. Meltzer LJ, Johnson C, Crosette J, et al: Prevalence of diagnosed sleep disorders in pediatric primary care practices. *Pediatrics* 125:1410–1418, 2010.
224. Messner AH: Evaluation of obstructive sleep apnea by polysomnography prior to pediatric adenotonsillectomy. *Arch Otolaryngol Head Neck Surg* 125:353–355, 1999.
225. Metes A, Hoffstein V, Direnfeld V, et al: Three-dimensional CT reconstruction and volume measurements of the pharyngeal airway before and after maxillofacial surgery in obstructive sleep apnea. *J Otol* 22:261, 1993.
226. Mickelson SA, Hakim I: Is postoperative intensive care monitoring necessary after uvulopalatopharyngoplasty? *Otolaryngol Head Neck Surg* 119(4):352–356, 1998.
227. Miles PJ, Nimkarn Y: Maxillomandibular advancement surgery in patients with obstructive sleep apnea: Mandibular morphology and stability. *Int J Adult Orthodon Orthognath Surg* 10:193–200, 1995.
228. Miller WE: Cardiac arrhythmias and conduction disturbances in the sleep apnea syndrome: Prevalence and significance. *Am J Med* 73:317–321, 1982.
229. Mitchell RB: Adenotonsillectomy for obstructive sleep apnea in children: Outcome evaluated by pre- and postoperative polysomnography. *Laryngoscope* 117:1844–1854, 2007.
230. Mitchell RB, Kelly J: Quality of life after adenotonsillectomy for SDB in children. *Otolaryngol Head Neck Surg* 133:569–572, 2005.
231. Mitchell RB, Kelly J: Behavior, neurocognition and quality-of-life in children with sleep-disordered breathing. *Int J Pediatr Otorhinolaryngol* 70(3):395–406, 2006.
232. Mitchell RB, Kelly J: Long-term changes in behavior after adenotonsillectomy for obstructive sleep apnea syndrome in children. *Otolaryngol Head Neck Surg* 134(3):374–378, 2006.
233. Mitchell RB, Kelly J: Changes in behavior for children with mild sleep-disordered breathing (SDB) or obstructive sleep apnea (OSA) after adenotonsillectomy. *Laryngoscope* 117:1685–1688, 2007.
234. Mitchell RB, Kelly J: Outcomes and quality of life following adenotonsillectomy for sleep-disordered breathing in children. *ORL J Otorhinolaryngol Relat Spec* 69(6):345–348, 2007.
235. Mitchell RB, Pereira KD, Friedman NR: Sleep-disordered breathing in children: Survey of current practice. *Laryngoscope* 116:956–958, 2006.
236. Mokhlesi B, Tulaimat A, Evans AT, et al: Impact of adherence with positive airway pressure therapy on hypercapnia in obstructive sleep apnea. *J Clin Sleep Med* 2:57–62, 2006.
237. Mokhlesi B, Tulaimat A, Faibussowitsch I, et al: Obesity hypoventilation syndrome: Prevalence and predictors in patients with obstructive sleep apnea. *Sleep Breath* 11:117–124, 2007.
238. Moore K: Obstructive Sleep Apnea. Surgical Update Vol 22, American Association of Oral and Maxillofacial Surgeons.
239. Moore MH, Guzman-Stein G, Prodman TW, et al: Mandibular lengthening by distraction for airway obstruction in Treacher Collins syndrome. *J Craniofac Surg* 5:22, 1994.
240. Morrison DL, Launois SH, Isono S, et al: Pharyngeal narrowing and closing pressures in patients with obstructive sleep apnea. *Am Rev Respir Dis* 148:606–611, 1993.
241. Mortimore IL, Marshall I, Wraith PK, et al: Neck and total body fat deposition in nonobese and obese patients with sleep apnea compared with that in control subjects. *Am J Respir Crit Care Med* 157:280–283, 1998.
242. Motta J, Guilleminault C, Schroeder JS, et al: Tracheostomy and hemodynamic changes in sleep-inducing apnea. *Ann Intern Med* 89(4):454–458, 1978.

243. Moyer CA, Sonnad SS, Garetz SL, et al: Quality of life in obstructive sleep apnea: A systematic review of the literature. *Sleep Med* 2:477–491, 2001.
244. Muto T, Takeda S, Kanazawa M, et al: The effect of head posture on the pharyngeal airway space (PAS). *Int J Oral Maxillofac Surg* 31:579–583, 2002.
245. National Commission on Sleep Disorders Research: *Report of the National Commission on Sleep Disorders Research, Publication 92*. Washington, DC, 1992, Superintendent of Documents, US Government Printing Office, Department of Health and Human Services.
246. National Institutes of Health Consensus Development Conference Statement: The treatment of sleep disorders of older people, March 26-28, 1990. *Sleep* 14:169–177, 1991.
247. Neeley WW 2nd, Edgin WA, Gonzales DA: A review of the effects of expansion of the nasal base on nasal airflow and resistance. *J Oral Maxillofac Surg* 65:1174–1179, 2007.
248. Neligan PJ, Porter S, Max B, et al: Obstructive sleep apnea is not a risk factor for difficult intubation in morbidly obese patients. *Anesth Analg* 109:1182, 2009.
249. Nieminen P, Tolonen U, Lopponen H: Snoring and obstructive sleep apnea in children: A 6-month follow-up study. *Arch Otolaryngol Head Neck Surg* 126:481–486, 2000.
250. Nieto FJ, Young TB, Lind BK, et al: Association of sleep-disordered breathing, sleep apnea, and hypertension in a large community-based *study. JAMA* 283:1829–1836, 2000.
251. Nimkarn Y, Miles PG, Waite PD: Maxillomandibular advancement surgery in obstructive sleep apnea syndrome patients: Long-term surgical stability. *J Oral Maxillofac Surg* 53:1414–1418, 1995.
252. Nino-Murcia G, McCann I, Bliwise DL, et al: Compliance and side effects in sleep apnea patients treated with nasal continuous positive airway pressure. *West J Med* 150:165–169, 1989.
253. O'Brien LM, Holbrook CR, Mervis CB, et al: Sleep and neurobehavioral characteristics of 5- to 7-year-old children with parentally reported symptoms of attention-deficit/hyperactivity disorder. *Pediatrics* 111:554–563, 2003.
254. O'Brien LM, Mervis CB, Holbrook CR, et al: Neurobehavioral implications of habitual snoring in children. *Pediatrics* 114:44–49, 2004.
255. Ogden CL, Carroll MD, Curtin LR, et al: Prevalence of overweight and obesity in the United States 1999-2004. *JAMA* 295:1549–1555, 2006.
256. Oksenberg A, Khaymaysi I, Silverberg DS, et al: Association of body position with severity of apneic events in patients with severe nonpositional obstructive sleep apnea. *Chest* 118:1018–1024, 2000.
257. Orr WC, Levine N, Buchanan R: Effect of cleft palate repair and pharyngeal flap surgery on upper airway obstruction during sleep. *Plast Reconstr Surg* 80(2):226–230, 1987.
258. Osman EZ, Abo-Khatwa MM, Hill PD, et al: Palatal surgery for snoring: Objective long-term evaluation. *Clin Otolaryngol Allied Sci* 28(3):257–261, 2003.
259. Otsuka R, Ribeiro de Almeida F, Lowe AA: The effects of oral appliance therapy on occlusal function in patients with obstructive sleep apnea: A short-term prospective study. *Am J Orthod Dentofacial Orthop* 131:176–183, 2007.
260. Pae EK, Ferguson KA: Cephalometric characteristics of non-obese patients with severe OSA. *Angle Orthod* 69:408–412, 1999.
261. Pancer J, Al-Faifi S, Al-Faifi M, et al: Evaluation of variable mandibular advancement appliance for treatment of snoring and sleep apnea. *Chest* 116:1511, 1999.
262. Pankow W, Hijjeh N, Schuttler F, et al: Influence of noninvasive positive pressure ventilation on inspiratory muscle activity in obese subjects. *Eur Respir J* 10:2847–2852, 1997.
263. Partinen M, Guilleminault C: Daytime sleepiness and vascular morbidity at seven-year follow-up in obstructive sleep apnea patients. *Chest* 97:27–32, 1990.
264. Partinen M, Guilleminanlt C, Quera-Salva MA, Jamieson A: Obstructive sleep apnea and cephalometric roentgenograms: The role of anatomic upper airway abnormalities in the definition of abnormal breathing during sleep. *Chest* 93:1199–1205, 1988.
265. Partinen M, Jamieson A, Guilleminault C: Long-term outcome for obstructive sleep apnea syndrome patients: Mortality. *Chest* 94:1200–1204, 1988.
266. Peker Y, Kraiczi H, Hedner J, et al: An independent association between obstructive sleep apnea and coronary artery disease. *Eur Respir J* 14:179, 1999.
267. Pepin JL, Krieger J, Rodenstein D, et al: Effective compliance during the first three months of continuous positive airways pressure: A European prospective study of 121 patients. *Am J Respir Crit Care Med* 160:1124–1129, 1999.
268. Peppard PE, Young T, Palta M, et al: Longitudinal study of moderate weight change and sleep-disordered breathing. *JAMA* 284:3015, 2000.
269. Peppard PE, Young T, Palta M, et al: Prospective study of the association between sleep-disordered breathing and hypertension. *N Engl J Med* 342:1378–1384, 2000.
270. Perkins JA, Sie KC, Milczuk H, et al: Airway management in children with craniofacial anomalies. *Cleft Palate Craniofac J* 34:135, 1997.
271. Perlyn CA, Schmelzer RE, Sutera SP, et al: Effect of distraction osteogenesis of the mandible on upper airway volume and resistance in children with micrognathia. *Plast Reconstr Surg* 109:1809–1818, 2002.
272. Petri N, Suadicani P, Wildschiodtz G, et al: Predictive value of Muller maneuver, cephalometry and clinical features for the outcome of uvulopalatopharyngoplasty. *Acta Otolaryngol* 114:565, 1994.
273. Pillar G, Peled R, Lavie P: Recurrence of sleep apnea without concomitant weight increase 7.5 years after weight reduction surgery. *Chest* 106:1702–1704, 1994.
274. Pirklbauer K, Russmueller G, Stiebellehner L, et al: Maxillomandibular advancement for treatment of obstructive sleep apnea syndrome: A systematic review. *J Oral Maxillofac Surg* 69:e165–e176, 2011.
275. Posnick JC, Agnihotri N: Consequences and management of nasal airway obstruction in the dentofacial deformity patient. *Curr Opin Otolaryngol Head Neck Surg* 18:323–331, 2010.
276. Posnick JC, Agnihotri N: Managing chronic nasal airway obstruction at the time of orthognathic surgery: A twofer. *J Oral Maxillofac Surg* 69:695–701, 2011.
277. Posnick JC, Fantuzzo J, Orchin J: Deliberate operative rotation of the maxillomandibular complex to alter the A-point to B-point relationship for enhanced facial esthetics. *J Oral Maxillofac Surg* 64:1687–1695, 2006.
278. Posnick JC, Fantuzzo JJ, Troost, T: Simultaneous intranasal procedures to improve chronic obstructive nasal breathing in patients undergoing maxillary (Le Fort I) osteotomy. *J Oral Maxillofac Surg* 65:2273–2281, 2007.
279. Powell NB, Guilleminault C, Riley RW, Smith L: Mandibular advancement and obstructive sleep apnea syndrome. *Bull Eur Physiopathol Respir* 19:607–610, 1983.
280. Powell NB, Riley RW: A surgical protocol for sleep disordered breathing. In Waite PD, editor. *Oral Maxillofac Surg Clin North Am* 7:345–354, 1995.
281. Pracharktam N, Nelson S, Hans MG, et al: Cephalometric assessment in obstructive sleep apnea. *Am J Orthod Dentofacial Orthop* 109:410–419, 1996.
282. Practice parameters for the treatment of obstructive sleep apnea in adults: The efficacy of surgical modifications of the upper airway. Report of the American Sleep Disorders Association. *Sleep* 19:152, 1996.
283. Prinsell JR: Maxillomandibular advancement surgery in a site-specific treatment approach for obstructive sleep apnea in 50 consecutive patients. *Chest* 116:1519–1529, 1999.
284. Prinsell JR: Maxillomandibular advancement surgery for obstructive sleep apnea syndrome. *J Am Dent Assoc* 133:1489–1497, 2002.
285. Prinsell JR: Primary and secondary telegnathic maxillomandibular advancement, with or without adjunctive procedures, for obstructive sleep apnea in adults: A literature review and treatment recommendations. *J Oral Maxillofac Surg* 70:1659–1677, 2012.

286. Punjabi NR: The epidemiology of adult sleep apnea. *Proc Am Thorac Soc* 5:136–143, 2008.
287. Rachmiel A, Aizenbud D, Pillar G, et al: Bilateral mandibular distraction for patients with compromised airway analyzed by three-dimensional CT. *Int J Oral Maxillofac Surg* 34:9–18, 2005.
288. Randall DK: *Dreamland: Adventures in the strange science of sleep*. New York, 2012, W.W. Norton & Co..
289. Rapoport DM, Garay SM, Epstein H, et al: Hypercapnia in the obstructive sleep apnea syndrome. A reevaluation of the "Pickwickian syndrome". *Chest* 89:627–635, 1986.
290. Rasheid S, Banasiak M, Gallagher SF, et al: Gastric bypass is an effective treatment for obstructive sleep apnea in patients with clinically significant obesity. *Obes Surg* 13:58–61, 2003
291. Reeves-Hoche MK, Meck R, Zwillich CW, et al: An objective evaluation of patient compliance. *Am J Respir Crit Care Med* 149:149, 1994.
292. Reimer MA, Flemons WW: Quality of life in sleep disorders. *Sleep Med Rev* 7:335–349, 2003.
293. Remmers JE, deGroot WJ, Sauerland EK, Anch AM: Pathogenesis of upper airway occlusion during sleep. *J Appl Physiol* 44:931–938, 1978.
294. Ribeiro de Almeida F, Bittencourt LR, de Almeida CI, et al: Effects of mandibular posture on obstructive sleep apnea severity and the temporomandibular joint in patients fitted with an oral appliance. *Sleep* 25(5):505–511, 2002.
295. Ricketts RM: Respiratory obstruction syndrome. *Am J Orthod* 54:495–507, 1968.
296. Rieder AA, Flanary V: The effect of polysomnography on pediatric adenotonsillectomy postoperative management. *Otolaryngol Head Neck Surg* 132:263–267, 2005.
297. Riley R, Guilleminault C, Powell N, Derman S: Mandibular osteotomy and hyoid bone advancement for obstructive sleep apnea: A case report. *Sleep* 7:79, 1984.
298. Riley RW, Guilleminault C, Powell N: Palatopharyngoplasty failure, cephalometric roentgenograms and obstructive sleep apnea. *Otolaryngol Head Neck Surg* 93:240, 1985.
299. Riley RW, Powell NB, Guilleminault C: Inferior sagittal osteotomy of the mandible with hyoid myotomy suspension: A new procedure for obstructive sleep apnea. *Otolaryngol Head Neck Surg* 94:589, 1986.
300. Riley RW, Powell NB, Guilleminault C: Inferior mandibular osteotomy and hyoid myotomy suspension for obstructive sleep apnea: A review of 55 patients. *J Oral Maxillofac Surg* 47:159, 1989.
301. Riley RW, Powell NB, Guilleminault C: Maxillofacial surgery and obstructive sleep apnea: A review of 80 patients. *Otolaryngol Head Neck Surg* 101:353, 1989.
302. Riley RW, Powell NB, Guilleminault C: Maxillary, mandibular, and hyoid advancement for treatment of obstructive sleep apnea: A review of 40 patients. *J Oral Maxillofac Surg* 48:20–26, 1990.
303. Riley RW, Powell NB, Guilleminault C: Maxillofacial surgery and nasal CPAP: A comparison of treatment for obstructive sleep apnea syndrome. *Chest* 98:1421–1425, 1990.
304. Riley RW, Powell NB, Guilleminault C: Obstructive sleep apnea syndrome: A surgical protocol for dynamic upper airway reconstruction. *J Oral Maxillofac Surg* 51:742–747, 1993.
305. Riley RW, Powell NB, Guilleminault C: Obstructive sleep apnea syndrome: A review of 306 consecutively treated surgical patients. *Otolaryngol Head Neck Surg* 108:117–125, 1993.
306. Riley RW, Powell NB, Guilleminault C, et al: Maxillary, mandibular and hyoid advancement: An alternative to tracheostomy in obstructive sleep apnea. *Otolaryngol Head Neck Surg* 94:584, 1986.
307. Riley RW, Powell NB, Li KK, et al: Surgery and obstructive sleep apnea: Long-term clinical outcomes. *Otolaryngol Head Neck Surg* 122:415–421, 2000.
308. Ringqvist M, Walker-Engstrom ML, Tegelberg A, Ringqvist I: Dental and skeletal changes after 4 years of obstructive sleep apnea treatment with a mandibular advancement device: A prospective, randomized study. *Am J Orthod Dentofacial Orthop* 124:54–60, 2003.
309. Ritter CT, Trudo FJ, Goldbert AN, et al: Quantitative evaluation of the upper airway during nasopharyngoscopy with the Muller maneuver. *Laryngoscope* 109:954, 1999.
310. Robertson C, Herbison P, Harkness M: Dental and occlusal changes during mandibular advancement splint therapy in sleep disordered patients. *Eur J Orthod* 25:371–376, 2003.
311. Ronchi G, Novelli G, Colombo L, et al: Effectiveness of maxillomandibular advancement in obstructive sleep apnea patients with and without skeletal anomalies. *Int J Oral Maxillofac Surg* 39:541–547, 2010.
312. Rosen CL, Storfer-Isser A, Taylor HG, et al: Increased behavioral morbidity in school-aged children with sleep-disordered breathing. *Pediatrics* 114:1640–1648, 2004.
313. Rosen GM, Muckle RP, Mahowald MW, et al: Postoperative respiratory compromise in children with obstructive sleep apnea syndrome: Can it be anticipated? *Pediatrics* 93:784–788, 1994.
314. Saitoh K: Long-term changes in pharyngeal airway morphology after mandibular setback surgery. *Am J Orthod Dentofacial Orthop* 125:556–561, 2004.
315. Sakakibara H, Tong M, Matsushita K, et al: Cephalometric abnormalities in non-obese and obese patients with obstructive sleep apnea. *Eur Respir J* 13:403– 410, 1999.
316. Samman N, Tang SS, Xia J: Cephalometric study of the upper airway in surgically corrected class III skeletal deformity. *Int J Adult Orthodon Orthognath Surg* 17:180–190, 2002.
317. Sanders JC, King MA, Mitchell RB, Kelly JP: Perioperative complications of adenotonsillectomy in children with obstructive sleep apnea syndrome. *Anesth Analg* 103:1115–1121, 2006.
318. Sanders MH, Gruendl CA, Rogers RM: Patient compliance with nasal CPAP therapy for sleep apnea. *Chest* 90:330–333, 1986.
319. Santos M, Laureano Filho J, Campello RI, et al: Improvement in respiration and craniofacial changes associated with weight loss after bariatric surgery. *J Oral Maxillofac Surg* 69:e177–e185, 2011.
320. Shepard JW, Jr, Thawley SE: Localization of upper airway collapse during sleep in patients with obstructive sleep apnea. *Am Rev Respir Dis* 141:1350–1355, 1990.
321. Schendel S, Powell N, Jacobson R: Maxillary, mandibular, and chin advancement: Treatment planning based on airway anatomy in obstructive sleep apnea. *J Oral Maxillofac Surg* 69:663–676, 2011.
322. Schendel SA, Jacobson R, Khalessi S: Airway growth and development: A computerized three dimensional analysis. *J Oral Maxillofac Surg* 70:2174–2183, 2012
323. Schwab RJ, Gefter WB, Hoffman EA, et al: Dynamic upper airway imaging during awake respiration in normal subjects and patients with sleep-disordered breathing. *Am Rev Respir Dis* 148(5):1385–1400, 1993.
324. Schwab RJ, Gupta KB, Gefter WB, et al: Upper airway and soft tissue anatomy in normal subjects and patients with sleep-disordered breathing: Significance of lateral pharyngeal walls. *Am J Respir Crit Care Med* 152:1673, 1995.
325. Schwab RJ, Gupta KB, Metzger LJ, et al: Upper airway soft tissue structural changes with a dental appliance in apneics. *Am J Respir Crit Care Med* 153:A719, 1996.
326. Schwab RJ, Pierson R, Pasirstein M, et al: Family aggregation of upper airway soft tissue structures in normals and patients with sleep apnea. *Am J Respir Crit Care Med* 173:453–463, 2006.
327. Schwartz AR, Smith PL, Wise RA, et al: Induction of upper airway occlusion in sleeping individuals with subatmospheric negative pressure. *J Appl Physiol* 64:535, 1988.
328. Section on Pediatric Pulmonology, Subcommittee on Obstructive Sleep Apnea Syndrome, American Academy of Pediatrics: Clinical practice guideline: Diagnosis and management of childhood obstructive sleep apnea syndrome. *Pediatrics* 109:704–712, 2002.
329. Series F, Simoneau JA, St Pierre S, et al: Characteristics of the genioglossus and musculus uvulae in sleep apnea/hypopnea syndrome and in snorers. *Am J Respir Crit Care Med* 153:1870–1874, 1996.

330. Shamsuzzaman AS, Somers VK: Fibrinogen, stroke, and obstructive sleep apnea: An evolving paradigm of cardiovascular risk [editorial]. *Am J Respir Crit Care Med* 162:2018, 2000.
331. Shamsuzzaman ASM, Gersch BJ, Somers VK: Obstructive sleep apnea: Implications for cardiac and vascular disease. *JAMA* 290:1906–1914, 2003.
332. Shelton KE, Gay SB, Woodson H, et al: Pharyngeal fat in obstructive sleep apnea. *Am Rev Respir Dis* 148:462, 1993.
333. Shepard JW, Jr: Hypertension, cardiac arrhythmias, myocardial infarction and stroke in relation to obstructive sleep apnea. *Clin Chest Med* 13:437–458, 1992.
334. Sher AE: Upper airway surgery for obstructive sleep apnea. *Sleep Med Rev* 6:195, 2002.
335. Sher AE, Schechtman KB, Piccirillo JE: The efficacy of surgical modifications of the upper airway in adults with obstructive sleep apnea syndrome. *Sleep* 19:156–177, 1996.
336. Sher AE, Thorpy MJ, Shprintzen RJ, et al: Predictive value of Muller's maneuver in selection of patients for uvulopalatopharyngoplasty. *Laryngoscope* 95:1483–1487, 1988.
337. Shimura R, Tatsumi K, Nakamura A, et al: Fat accumulation, leptin, and hypercapnia in obstructive sleep apnea-hypopnea syndrome. *Chest* 127:543–549, 2005.
338. Shintani T, Kozawa T, Himi T: Obstructive sleep apnea by analysis of MRI findings. *Int Congr Ser* 1257:99–102, 2003.
339. Shprintzen RJ: Pharyngeal flap surgery and the pediatric upper airway. *Int Anesthesiol Clin* 26(1):79–88, 1988.
340. Shprintzen RJ, Singer L, Sidoti EJ, Argamaso RV: Pharyngeal flap surgery: Postoperative complications. *Int Anesthesiol Clin* 30(4):115–124, 1992.
341. Sidman J, Sampson D, Templeton B: Distraction osteogenesis of the mandible for airway obstruction in children. *Laryngoscope* 111:1137, 2001.
342. Simmons FB, Guilleminanlt C, Miles LE: The palatopharyngoplasty operation for snoring and sleep apnea: An interim report. *Otolaryngol Head Neck Surg* 92:375–380, 1984.
343. Sirois M, Caouette-Laberge L, Spier S, et al: Sleep apnea following a pharyngeal flap: A feared complication. *Plast Reconstr Surg* 93(5):943–947, 1994.
344. Slovik Y, Tal A, Shapira Y, et al: Complications of adenotonsillectomy in children with OSAS younger than 2 years of age. *Int J Pediatr Otorhinolaryngol* 67:847–851, 2003.
345. Smatt Y, Ferri J: Retrospective study of 18 patients treated by maxillomandibular advancement with adjunctive procedures for obstructive sleep apnea syndrome. *J Craniofac Surg* 16:770–777, 2005.
346. Smirne S, Iannaccone S, Ferini-Strambi L, et al: Muscle fibre type and habitual snoring. *Lancet* 337:597–599, 1991.
347. Smith CB, Waite PD: Surgical management of obstructive sleep apnea in acromegaly with mandibular prognathism and macroglossia: A treatment dilemma. *J Oral Maxillofac Surg* 70:207–210, 2012
348. Solow B, Siersbaek-Nelsen S, Greve EE: Airway adequacy, head posture, and craniofacial morphology. *Am J Orthod* 86:214–223, 1984.
349. Somers VK, Dyken ME, Clary MP, Abboud FM: Sympathetic neural mechanisms in obstructive sleep apnea. *J Clin Invest* 96:897–904, 1995.
350. Somers VK, White DP, Amin R, et al; American Heart Association Council for High Blood Pressure Research Professional Education Committee, Council on Clinical Cardiology; American Heart Association Stroke Council; American Heart Association Council on Cardiovascular Nursing; American College of Cardiology Foundation: Sleep apnea and cardiovascular disease: an American Heart Association/American College of Cardiology Foundation Scientific Statement from the American Heart Association Council for High Blood Pressure Research Professional Education Committee, Council on Clinical Cardiology, Stroke Council, and Council on Cardiovascular Nursing. In collaboration with the National Heart, Lung, and Blood Institute National Center on Sleep Disorders Research (National Institutes of Health). *Circulation* 118:1080–1111, 2008.
351. Statham MM, Elluru RG, Buncher R, Kalra M: Adenotonsillectomy for obstructive sleep apnea syndrome in young children: Prevalence of pulmonary complications. *Arch Otolaryngol Head Neck Surg* 132:476–480, 2006.
352. Stradling JR, Crosby JH: Predictors and prevalence of obstructive sleep apnea and snoring in 1001 middle aged men. *Thorax* 46:85–90, 1991.
353. Straith RE, Ritter G: Partial resection of the tongue for the amelioration of obstructive sleep apnea: A report on 34 cases with long-term follow-up. *J Craniomaxillofac Surg* 25:305–309, 1997.
354. Strelzow VV, Blanks RH, Basile A, Strelzow AE: Cephalometric airway analysis in obstructive sleep apnea syndrome. *Laryngoscope* 98:1149–1158, 1988.
355. Suen JS, Arnold JE, Brooks LJ: Adenotonsillectomy for treatment of obstructive sleep apnea in children. *Arch Otolaryngol Head Neck Surg* 121:525–530, 1995.
356. Sugiura T, Noda A, Nakata S, et al: Influence of nasal resistance on initial acceptance of continuous positive airway pressure in treatment for obstructive sleep apnea syndrome. *Respiration* 74(1):56–60, 2007.
357. Sullivan CE, Berthon-Jones M, Issa FG, et al: Reversal of obstructive sleep apnea by continuous positive airway pressure applied through the nares. *Lancet* 1:862–655, 1981.
358. Sundaram S, Bridgman S, Lim J, Lasserson TJ: Surgery for obstructive sleep apnoea. *Cochrane Database Syst Rev* 19(4):CD001004, 2005.
359. Suratt PM, Dee P, Atkinson RL, et al: Fluoroscopic and computer tomographic features of the pharyngeal airway in obstructive sleep apnea. *Am Rev Respir Dis* 127:487, 1993.
360. Susarla SM, Abramson ZR, Dodson TB, Kaban LB: Cephalometric measurement of upper airway length correlates with the presence and severity of obstructive sleep apnea. *J Oral Maxillofac Surg* 68:2846–2855, 2010.
361. Susarla SM, Thomas RJ, Abramson ZR, Kaban LB: Biomechanics of the upper airway: Changing concepts in the pathogenesis of obstructive sleep apnea. *Int J Oral Maxillofac Surg* 39:1149–1159, 2010.
362. Suto Y, Matsuo T, Kato T, et al: Evaluation of pharyngeal airway in patients in sleep apnea: Value of ultrafast MR imaging. *AJR Am J Roentgenol* 160:311, 1993.
363. Tallgren A, Solow B: Hyoid bone position, facial morphology and head posture in adults. *Eur J Orthod* 9:1–8, 1987.
364. Tangugsorn V, Skatvedt O, Krogstad O, Lyberg T: Obstructive sleep apnea: A cephalometric study. Part I. Cervico-craniofacial skeletal morphology. *Eur J Orthod* 17:45–56, 1995.
365. Tassinari CA, Dalla Bernardina B, Cirignotta F, Ambrosetto G: Apneaic periods and the respiratory related arousal patterns during sleep in the Pickwickian syndrome. A polygraphic study. *Bull Physiopathol Respir (Nancy)* 8:1087–1102, 1972.
366. Tauman R, Gulliver TE, Krishna J, et al: Persistence of obstructive sleep apnea syndrome in children after adenotonsillectomy. *J Pediatr* 149:803–808, 2006.
367. Telakivi T, Kajaste S, Partinert M, et al: Cognitive function in obstructive sleep apnea. *Sleep* 74–75, 1993.
368. Thatcher GW, Maisel RH: The long-term evaluation of tracheostomy in the management of severe obstructive sleep apnea. *Laryngoscope* 113:201, 2003.
369. Tilkian AG, Guilleminault C, Schroeder JS: Sleep-induced apnea syndrome: Prevalence of cardiac arrhythmias and their reversal after tracheostomy. *Am J Med* 63:348, 1977.
370. Tselnik M, Pogrel MA: Assessment of the pharyngeal airway space after mandibular setback surgery. *J Oral Maxillofac Surg* 58:282–285, 2000.
371. Tsuiki S, Ono T, Kuroda T: Mandibular advancement modulates respiratory-related genioglossus electromyographic activity. *Sleep Breath* 4:53, 2000.
372. Turnbull NR, Battagel JM: The effects of orthognathic surgery on pharyngeal airway dimensions and quality of sleep. *J Orthod* 27:235–247, 2000.
373. Valnicek SM, Zuker R, Halpern L, Roy W: Perioperative complications of superior

pharyngeal flap surgery in children. *Plast Reconstr Surg* 93(5):954–958, 1994.
374. Vanderveken OM, Devolder A, Marklund M, et al: Comparison of a custom-made and a thermoplastic oral appliance for the treatment of mild sleep apnea. *Am J Respir Crit Care Med* 178:197, 2008.
375. Verse T, Baisch A, Maurer JT, et al: Multilevel surgery for obstructive sleep apnea: Short-term results. *Otolaryngol Head Neck Surg* 134:571–577, 2006.
376. Vicini C, Dallan I, Campanini A, et al: Surgery vs ventilation in adult severe obstructive sleep apnea syndrome. *Am J Otolaryngol* 31:14–20, 2010.
377. Villa MP, Bernkopf E, Pagani J, et al: Randomized controlled study of an oral jaw-positioning appliance for the treatment of obstructive sleep apnea in children with malocclusion. *Am J Respir Crit Care Med* 165:123, 2002.
378. Vos W, De Backer K, Devolder A, et al: Correlation between severity of sleep apnea and upper airways morphology based on advanced anatomical and functional imaging. *J Biomech* 40:2207–2213, 2007.
379. Waite PD, Shettar SM: Maxillomandibular advancement surgery: A cure for obstructive sleep apnea syndrome. In Waite PD, editor: Oral and maxillofacial treatment of obstructive sleep apnea. *Oral Maxillofac Surg Clin North Am* 7:327–336, 1995.
380. Waite PD, Tejera TJ, Anucul B: The stability of maxillary advancement using Le Fort I osteotomy with and without genial bone grafting. *Int J Oral Maxillofac Surg* 25:264–267, 1996.
381. Waite PD, Wooten V, Lachner J, Guyette RF: Maxillomandibular advancement surgery in 23 patients with obstructive sleep apnea. *J Oral Maxillofac Surg* 47:1256–1261, 1989.
382. Warwick JP, Mason DG: Obstructive sleep apnea in children. *Anaesthesia* 53:571–579, 1998.
383. Watanabe T, Isono S, Tanaka A, et al: Contribution of body habitus and craniofacial characteristics to segmental closing pressures of the passive pharynx in patients with sleep-disordered breathing. *Am J Respir Crit Care Med* 165(2):260–265, 2002.
384. Weatherly RA, Mai EF, Ruzicka DL, Chervin RD: Identification and evaluation of obstructive sleep apnea prior to adenotonsillectomy in children: A survey of practice patterns. *Sleep Med* 4:297–307, 2003.
385. Weaver TE, Grunstein RR: Adherence to continuous positive airway pressure therapy. *Proc Am Thorac Soc* 5:173–178, 2008.
386. Wenzel A, Williams S, Ritzau M: Changes in head posture and nasopharyngeal airway following surgical correction of mandibular prognathism. *Eur J Orthod* 11:37–42, 1989.
387. Wheatley JR, Tangel DJ, Mezzanotte WS, White DP: Influence of sleep on response to negative airway pressure of tensor palatini muscle and retropalatal airway. *J Appl Physiol* 75:2117–2124, 1993.
388. Winnberg A, Pancherz H, Westesson PL: Head posture and hyomandibular function in man. A synchronized electromyographic and videofluorographic study of the open-close-clench cycle. *Am J Orthod Dentofacial Orthop* 94:393–404, 1988.
389. Witt PD, Marsh J, Muntz H, et al: Acute obstructive sleep apnea as a complication of sphincter pharyngoplasty. *Cleft Palate Craniofac J* 33(3):183–189, 1996.
390. Wittenborn W, Panchal J, Marsh J, et al: Neonatal distraction surgery for micrognathia reduces obstructive sleep apnea and the need for tracheotomy. *J Craniofac Surg* 15:623, 2004.
391. Woodson BT: Predicting which patients will benefit from surgery for obstructive sleep apnea: *The ENT exam. ENT J* 78:792, 1999.
392. Yaggi HK, Concato J, Kernan WN, et al: Obstructive sleep apnea as a risk factor for stroke and death. *N Engl J Med* 353:2034–2041, 2005.
393. Young T, Palta M, Dempsey J, et al: The occurrence of sleep-disordered breathing among middle-aged adults. *N Engl J Med* 328:1230–1235, 1993.
394. Young T, Peppard P, Palta M, et al: Population-based study of sleep-disordered breathing as a risk factor for hypertension. *Arch Intern Med* 157:1745–1752, 1997.
395. Young T, Peppard PE, Taheri S: Excess weight and sleep-disordered breathing. *J Appl Physiol* 99:1592–1599, 2005.
396. Ysunza A, Garcia-Velasco M, Garcia-Garcia M, et al: Obstructive sleep apnea secondary to surgery for velopharyngeal insufficiency. *Cleft Palate Craniofac J* 30(4):387–390, 1993.
397. Yu CC, Hsiao HD, Lee LC, et al: Computational fluid dynamic study on obstructive sleep apnea syndrome treated with maxillomandibular advancement. *J Craniofac Surg* 20:426–430, 2009.
398. Yu LF, Pogrel MA, Ajayi M: Pharyngeal airway changes associated with mandibular advancement. *J Oral Maxillofac Surg* 52:40–43, 1994.
399. Zhao Y, Nguyen M, Gohl E, et al: Oropharyngeal airway changes after rapid palatal expansion evaluated with cone-beam computed tomography. *Am J Orthod Dentofacial Orthop* 137(4 Suppl):S71–S78, 2010.
400. Zucconi M, Strambli LF, Pestalozza G, et al: Habitual snoring and obstructive sleep apnea syndrome in children: Effects of early tonsil surgery. *Int J Pediatr Otorhinolaryngol* 26:235–243, 1993.

27

Treacher Collins Syndrome: Evaluation and Treatment

JEFFREY C. POSNICK, DMD, MD

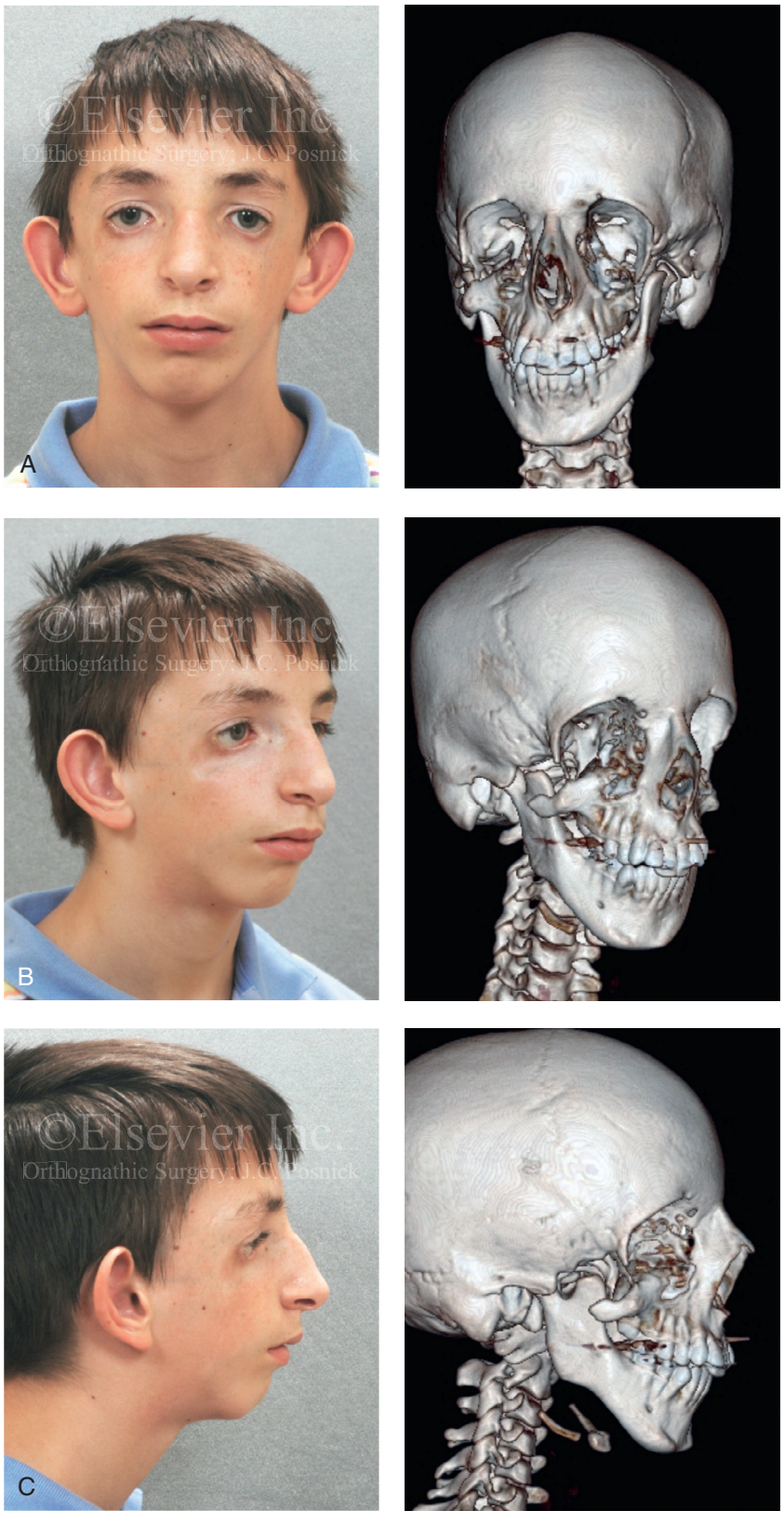
- Inheritance, Genetic Markers, and Testing
- Considerations during Infancy and into Early Childhood
- Dysmorphology with Treacher Collins Syndrome
- Facial Growth Potential with Treacher Collins Syndrome
- Classification of Temporomandibular Joint and Mandibular Malformation
- Staging of Skeletal Reconstruction: Timing and Techniques
- Indications for First-Stage Mandibular Reconstruction in the Newborn
- Indications for First-Stage Mandibular Reconstruction during the Mixed Dentition
- Nasal Reconstruction
- Facial Soft-Tissue Reconstruction
- External Ear Reconstruction
- External Auditory Canal and Middle-Ear Reconstruction
- Conclusions

Treacher Collins syndrome (TCS), which is also known as *mandibulofacial dysostosis,* is an autosomal dominant condition with variable expressivity.* It is generally characterized by bilaterally symmetric abnormalities of the structures within the first and second branchial arches. Early descriptions are attributed to Berry,[10] Treacher Collins,[51] and Franceschetti and Klein.[41] To date, the physical findings of many hundreds of individuals and families with TCS have been published in the world literature.

*References 6, 9, 24, 26-28, 38, 41, 42, 46, 50, 51, 56, 57, 65, 78, 79, 84, 96, 116, 117, 126, 128, 138, 157

The adult patient with fully expressed TCS has a convex horizontally deficient facial profile with a prominent nasal dorsum above a retrusive lower jaw and chin (Fig. 27-1). The eye region is characterized by an antimongoloid slant of the palpebral fissure as a result of colobomata and hypoplasia of the lower lids and the lateral canthi, including the partial absence of the eyelid cilia and inferolateral orbital hypoplasia and dystopia. Tongue-shaped processes of hair frequently extend into the preauricular region. The external ears are absent, malformed, or malposed and hearing is impaired as a result of variable degrees of hypoplasia of the external auditory canals and the ossicles of the middle ears. A characteristic finding is hypoplasia of the malar bones, often with clefting through the arches and limited formation of the residual zygomas, including the glenoid fossa component. The maxilla and mandible bones are also characteristically hypoplastic, with variable effects on the temporomandibular joints (TMJs), the muscles of mastication, and the muscles of facial expression. Interestingly, hypoplasia of the centrally located soft tissues of the face is not seen with TCS. In general, there is an Angle Class II anterior open-bite malocclusion and a steep clockwise-rotated maxillomandibular complex. The posterior facial height is short, and the anterior lower facial height is long. The A-point–to–B-point relationship in profile is consistent with the clockwise rotation of the jaws. The presence of cleft palate (with or without cleft lip and choanal atresia of the nasal cavity) is variable. Dental anomalies are present in 60% of individuals; these include tooth agenesis (33.3%), enamel opacities (20%), and the ectopic eruption of the maxillary first molars (13.3%).

Individuals with TCS have a reduced cranial base angle (i.e., basilar kyphosis). Craniosynostosis is not a feature of TCS, but the neurocranium may have an abnormal shape (i.e., decreased anteroposterior length and diminished bitemporal width) that is evident during childhood and remains through adulthood. [110] The degree of craniofacial malformation present at birth is believed to be relatively stable and non-progressive with age (this is discussed in more detail later in this chapter). TCS should not be



• **Figure 27-1** An 11-year-old boy with fully expressed Treacher Collins syndrome. His eyes are characterized by an antimongoloid slant of the palpebral fissures as a result of colobomata and hypoplasia of the lower lids and the lateral canthi, including the partial absence of the eyelid cilia and inferolateral orbital dystopia. The appearance of the malpositioned adnexal structures is a reflection of both zygomatic–orbital dystopia and hypoplasia of the soft tissues. The maxilla and the mandible are also deficient and malformed. The patient demonstrates a Type IIA mandibular malformation. The external ears are malformed and lack antihelical folds. **A,** Frontal facial and three-dimensional computed tomography scan views. **B,** Oblique facial and three-dimensional computed tomography scan views. **C,** Profile facial and three-dimensional computed tomography scan views.

confused with similar entities such as oculo–auriculo–vertebral dysplasia or Goldenhar syndrome. Distinguishing facial features of patients with TCS often include coloboma of the lower eyelids; the upper eyelids are more likely affected among patients with Goldenhar syndrome.

Inheritance, Genetic Markers, and Testing

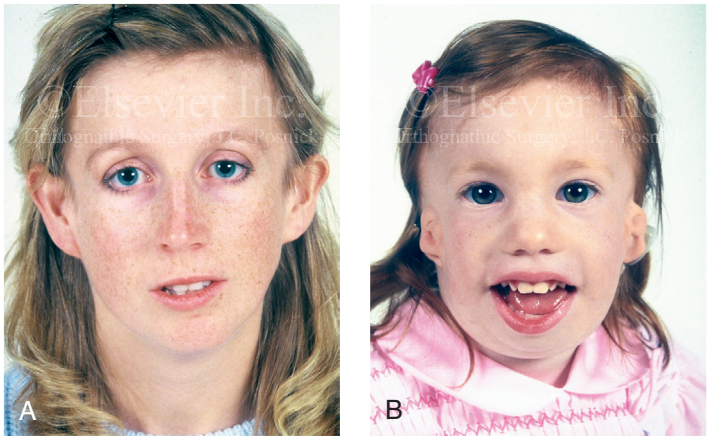
The occurrence of TCS is in the range of 1 in 25,000 to 1 in 50,000 live births. Inheritance is autosomal dominant, and male and female individuals are equally affected.[51] About 60% of probands with TCS have the disorder as the result of a de novo gene mutation. Each child of an individual with TCS has a 50% chance of inheriting the mutation (Fig. 27-2). Teber and colleagues identified TCOF1 mutations in 28 out of 36 (78%) individuals with a clinically unequivocal diagnosis of TCS.[147] The most frequent clinical findings were downward-slanting palpebral fissures, hypoplasia of the zygomatic complex, hypoplasia of the mandible, conductive deafness, any degree of microtia, and atresia of the external ear canal. Although there were interfamilial and intrafamilial variations that ranged from mild to severe, there were no genotype or phenotype correlations. Four clinically unaffected parents were heterozygous for the TCOF1 mutation. The authors concluded that modifying factors are important for phenotypic expression.

Genetic counseling for TCS is the process of providing individuals and families with information about the nature, inheritance, and implications of the syndrome to help them make informed medical and personal decisions. Molecular testing should be considered for any individual who presents with at least two major features or three minor features of TCS.[30,32,34,62,63,72,85,88,89,154,161,165] Until recently, penetrance in TCS was believed to be complete. Marres and colleagues confirmed the absence of clinical and radiographic findings in an individual with a pathogenic TCOF1 mutation. For this reason, testing is acceptable for individuals with any degree of severity of TCS features to confirm or refute the diagnosis.

Prenatal diagnosis for pregnancies that are at increased risk for TCS is possible via the analysis of DNA extracted from fetal cells obtained by amniocentesis; this is usually performed at about 15 to 18 weeks' gestation. Chorionic villus sampling can also be used for diagnosis at about 10 to 12 weeks' gestation. The malformation-causing allele of an affected family member must be identified before prenatal testing can be performed.

Considerations during Infancy and into Early Childhood

At the time of the birth of a child with TCS, concerns will center on the adequacy of the airway, swallowing, feeding, hearing, vision (corneal protection), the presence of cleft palate (with or without cleft lip), any other associated malformations, and the psychosocial well-being of the family.[8,17,27,31,77,94,95,101,103,104,120,160] The airway may be compromised as a result of multiple factors. The first is maxillary hypoplasia, which occurs with a degree of choanal stenosis or atresia with the blockage of the nasal airway. Second, mandibular micrognathia with a retropositioned tongue that at least partially obstructs the oropharyngeal and hypopharyngeal spaces will be present. In addition, tracheomalacia, vocal cord anomalies, or neuromotor involvement may also negatively affect the upper airway. Depending on the severity of the anomalies, a spectrum of upper airway compromise may necessitate, at a minimum, special infant positioning, an extended hospital stay, and pulse oximetry monitoring. Surgical considerations for the management of airway compromise in the infant or young adult may include tongue–lip adhesion, the use of a custom-made oral



• **Figure 27-2** This mother and daughter demonstrate the extent of variation in the expression of Treacher Collins syndrome within a family. The mother was not aware that she carried the Treacher Collins gene until after the birth of her daughter. *From Posnick JC: Treacher Collins syndrome: perspectives in evaluation and treatment,* J Oral Maxillofac Surg *55:1120, 1997.*

appliance, immediate or delayed tracheostomy, or an urgent mandibular advancement procedure.

As a result of any compromise in nasal and oral breathing and associated musculoskeletal malformations, the newborn with TCS may also have difficulty with swallowing and with achieving an adequate lip seal to the extent that urgent gavage-assisted feedings or the placement of a gastrostomy tube may be required to ensure adequate nutrition.

When a cleft of the secondary palate is documented, potential additive effects on the airway before and after repair must be considered. When Nager syndrome is present, the soft palate is both clefted and severely hypoplastic to the extent that the achievement of a functioning palate is not feasible.[65,96] A primary pharyngeal flap—although helpful for velopharyngeal function—is rarely indicated, because further obstruction of the airway would likely result. Evaluation by a *medical geneticist* is indicated to clarify the diagnosis and all presenting anomalies, to discuss family planning issues, and to analyze chromosomal studies.

Within a few days of birth, the results of an examination by an experienced *pediatric otolaryngologist* in combination with formal *audiology testing* will clarify the extent of conductive hearing loss and allow for the early fitting of hearing aids to assist the infant with the acquisition of communication skills and encourage the normal bonding process with the family.

At some point early in life, a complete craniofacial *computed tomography (CT) scanning* from the top of the skull through the cervical spine with three-dimensional reformation will be useful to document the extent of craniofacial skeletal anomalies. Special *focused cuts through the petrous temporal bones* are also required to document external auditory canal and middle and inner ear anatomy. Appropriate craniofacial, maxillofacial, cleft lip and palate, and auricular reconstructive surgeons should also be consulted, as indicated.

An assessment by a *pediatric ophthalmologic* is necessary to determine associated eye anomalies, extraocular muscle function, corneal exposure difficulties, and visual acuity. Ophthalmologic issues may include vision loss (37%), amblyopia (33%), refractive errors (58%), anisometropia (17%), and strabismus (37%). Interestingly, it has been the impression of many clinicians and scientists that the individual with TCS has a tendency toward a short stature, at least early in life, and that above-average intelligence is generally observed.

Dysmorphology with Treacher Collins Syndrome

Cranio–Orbito–Zygomatic Region

In earlier published studies, 14 reproducible cranio-orbito-zygomatic measurements taken from each of 26 standard axial CT scans of individuals with symmetric forms of TCS who had not been operated on were compared with the measurements of age-matched controls.[110] The interorbital measurements (i.e., medial and lateral orbital wall separations) of the patients with TCS were at the mean when compared with their cohort group (i.e., no measurable orbital hypertelorism or hypotelorism), whereas the zygomatic measurements were significantly less than normal, thereby confirming the extent of malar hypoplasia. The congenitally deficient lateral aspect of the orbits in individuals with TCS was confirmed by the greater than normal values measured for globe protrusion and medial orbital wall protrusion in conjunction with diminished lateral orbital wall lengths. These measurements use the lateral orbital rim as a reference point and confirm the limited depth of the lateral orbits as a result of hypoplasia of the lateral orbital rims. The lateral orbital rims are components of the hypoplastic zygomatic complex. The abnormal shape of the anterior cranial vault in patients with TCS was documented as a diminished intercoronal (bitemporal) distance (width) and decreased cephalic length as compared with values for normal age-matched controls. In general, the CT measurements documented in the described study agree with the clinically observed morphology of TCS. The measurements carried out in the zygomatic structures confirmed the extent of hypoplasia of the zygomas for the group as a whole and in each individual patient; they also documented the extent of globe protrusion and decreased upper face width in patients with TCS. It appears as though the bones immediately adjacent to the involved zygomatic complex also experience a degree of hypoplasia or at least distortion.[4,28,39,87,110,113,125,158,159,167,169]

Maxillomandibular Region

In 1975, Roberts and colleagues completed a radiographic cephalometric study of TCS. Serial cephalometric radiographs were available for eight patients who presented with the full range of features that are characteristic of this syndrome.[124] The expected facial convexity in patients with TCS was substantiated by cephalometric measurements. The extent of convexity was attributed to the severity of mandibular retrognathia (i.e., a decreased sella–nasion–B-point angle). Interestingly, the horizontal projection of the maxilla to the cranial base (i.e., the sella–nasion–A-point angle) remained within normal limits in the patients that were measured. Over time and with growth, the facial convexity remained relatively constant, thus confirming that the facial profile morphology of the infant with TCS was remarkably similar to that of the adult. Anterior facial heights were measured as "upper facial height" (i.e., the nasion to the anterior nasal spine) and "total facial height" (i.e., the nasion to the menton). The upper facial height was found to be relatively normal, whereas the total facial height was often excessive. This is the result of a combination of anterior open-bite malocclusion, mandibular retrognathism, and chin dysplasia characterized by increased vertical length and horizontal retrusion. The angle between the sella–nasion plane and the mandibular plane was obtuse,

which confirmed the steepness of the clockwise rotation of the mandibular plane. The angle between the sella–nasion plane and the palatal plane was also obtuse, thus confirming the steepness of the clockwise rotation of the maxillary plane. The posterior facial height was markedly shorter for patients with TCS, with values in five of the eight patients found to be a minimum of four standard deviations less than those of the normal cohort.

The effective horizontal length of the mandible was markedly reduced, with values in most of the patients falling below four standard deviations as compared with those in the normal cohort. The decreased mandible size was evident in both the ramus height and the body length. The gonial angle was confirmed to be obtuse, with values of two standard deviations from the mean as compared with control values. The extent of antigonial notching was also documented in patients with TCS and was found to be similar to that observed among patients with condylar arrest early during their lives (e.g., as a result of juvenile rheumatoid arthritis or TMJ trauma at a young age). The steepness of the clockwise rotation of the maxillary and mandibular planes combined with the anterior and posterior vertical height disproportions and severe horizontal deficiency of the mandible is also indirectly seen in the A-point–to–B-point discrepancy. All of these morphologic findings explain the observed facial dysmorphology.[4, 39,49,78,79,87]

Facial Soft Tissues

Soft-tissue anomalies in individuals with TCS are manifested primarily in four clinical regions: the external ears; the eyelid–adnexal structures; the preauricular–cheek skin; and the temporal fossa (e.g., temporalis muscle hypoplasia).[10,11,22,35,36,64,76,86,132,133,143,148,156] The extent of deficiency within the soft-tissue envelope is variable and, to large extent, reflective of the skeletal involvement. Each patient is unique and must be evaluated individually. The extent of soft-tissue anomalies continues to represent the most difficult obstacle to a favorable reconstruction.

External Auditory Canal, Middle and Inner Ear Structures, and Audiologic Findings

Relatively symmetric (i.e., from side to side) outer and middle ear malformations are characteristic of TCS. Middle-ear abnormalities (e.g., hypoplastic or absent cavities and ossicles) are an almost universal finding.[18,55,58,66,118] The majority of patients have a unilateral or bilateral moderate or greater conductive hearing loss, with a flat or reverse sloping configuration on the audiogram. Most of the hearing loss in these patients is attributable to middle-ear malformations. In 1993, Pron and colleagues reported the results of a detailed CT radiologic and audiologic investigation of a large series of pediatric patients with TCS.[118] The objectives of the study were 1) to determine the degree and symmetry of the external auditory canal and middle-ear abnormalities and evaluate hearing loss; and 2) to explore the relationship between hearing loss and external auditory canal and middle-ear abnormalities in patients with TCS. Data were available from 29 children who had not yet undergone any ear interventions and who had undergone audiometric testing and focused standardized petrous temporal bone CT scans. A review of the CT scans allowed for the evaluation of the external ear canal and the middle-ear anatomy. The majority of patients (88%) had largely symmetric external auditory canal abnormalities that were either stenotic (31%), atretic (54%), or normal (15%). Middle-ear cavity ossicular deformities were generally symmetric (96%), and cavities were either hypoplastic (85%) or missing (4%). Cavities that were hypoplastic were also deformed and assumed a rectangular rather than an oval shape. Ossicle deformities were generally completely symmetric and consisted largely of hypoplastic (46%) or missing (46%) ossicles. Ossicles (both the malleus and the incus) that were hypoplastic also tended to be ankylosed (82%) to either the lateral or medial wall of the tympanic recess. The structures of the external auditory canal and the middle ear are generally related. Increasing degrees of external ear canal malformations (i.e., normal, stenotic, or atretic) are directly associated with ossicle and cavity malformations. Of the patients with atretic canals, 67% had missing ossicles, and 33% had small or ankylosed ossicles. No abnormalities were noted in the inner-ear structures (i.e., cochlea, vestibule, canals, and internal auditory meatus) in the patients with TCS who were studied. According to the study by Pron and colleagues, hearing loss for the patient with TCS ranges from mild to severe (average, 58 dB of hearing loss).[118] The majority of individuals (96%) have a moderate or greater degree of hearing loss.

Relationships exist between the external auditory canal, the middle-ear ossicles, and the degree and configuration of hearing loss.[18,55,58,66,118] There is an association between the degree of external ear canal malformation and the degree of hearing loss in patients with TCS. In addition, hypoplastic, ankylosed, and missing ossicles appear to be associated with increasing levels of hearing loss; on average, the hearing loss is 52 dB, 56 dB, and 60 dB of hearing loss, respectively.

Facial Growth Potential with Treacher Collins Syndrome

The degree of malformation present at the time of birth in a newborn with TCS is believed to be relatively stable and non-progressive with age (Figs. 27-3 through 27-6).[53,102,109,114,115,124] Roberts and associates reviewed serial cephalometric radiographs from individuals who were known to have TCS.[124] They documented the stability of the mandible, which continued to grow but not to catch up. Through independent research, Garner reported findings of the cephalometric analysis of three patients of varied ages who were confirmed to have TCS.[49] He also

Text continued on p. 1069

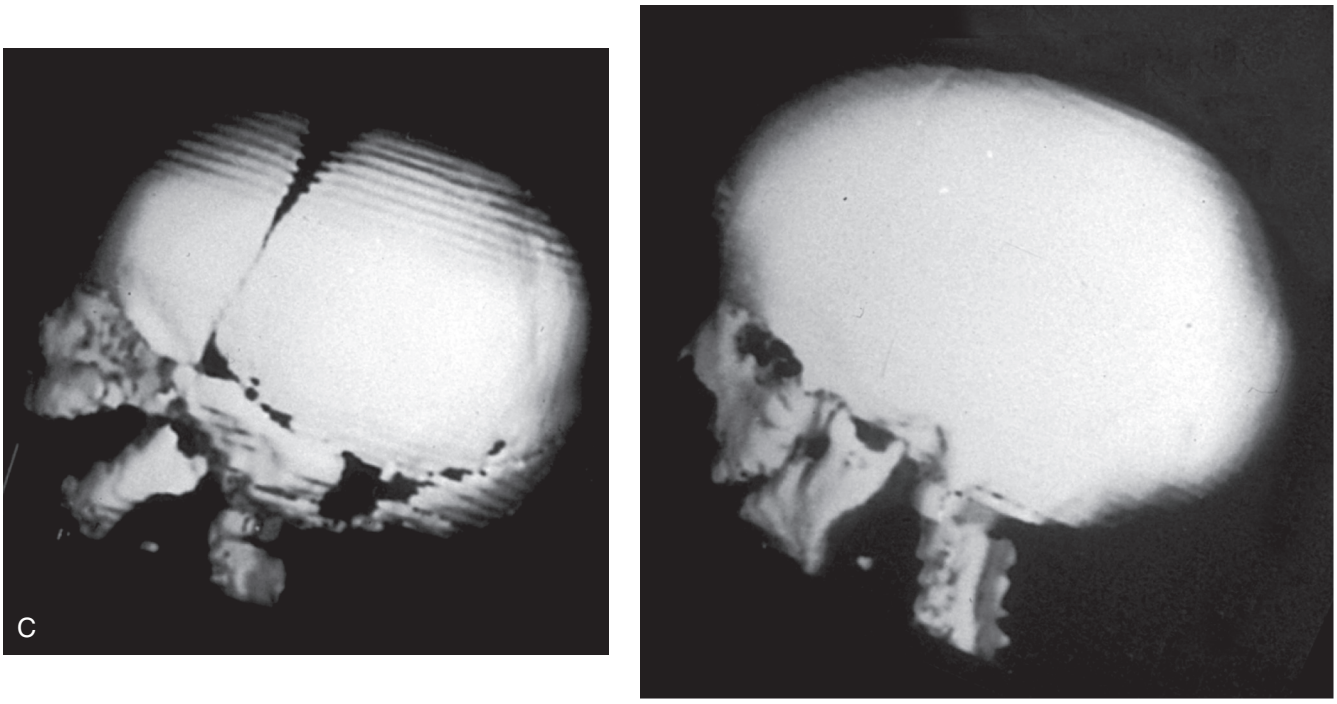
2 months

2 years

2 months

2 years

• **Figure 27-3** A child with a severe form of Treacher Collins syndrome was followed up longitudinally without treatment intervention. No progression of the deformity was seen during clinical or computed tomography scan examination. **A,** Frontal and **B,** profile views at 2 months and 2 years of age.



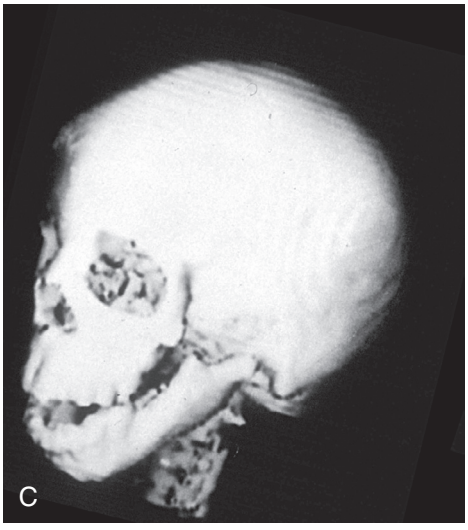
• **Figure 27-3, cont'd C** and **D,** Computed tomography scan views of the craniofacial region at 2 months and 2 years, respectively, demonstrating the consistent growth of the malformed condyles, the coronoid processes, and the ascending rami of the mandible. *From Posnick JC: Treacher Collins syndrome: perspectives in evaluation and treatment,* J Oral Maxillofac Surg *55:1120, 1997.*

1 year

5 years

1 year

5 years

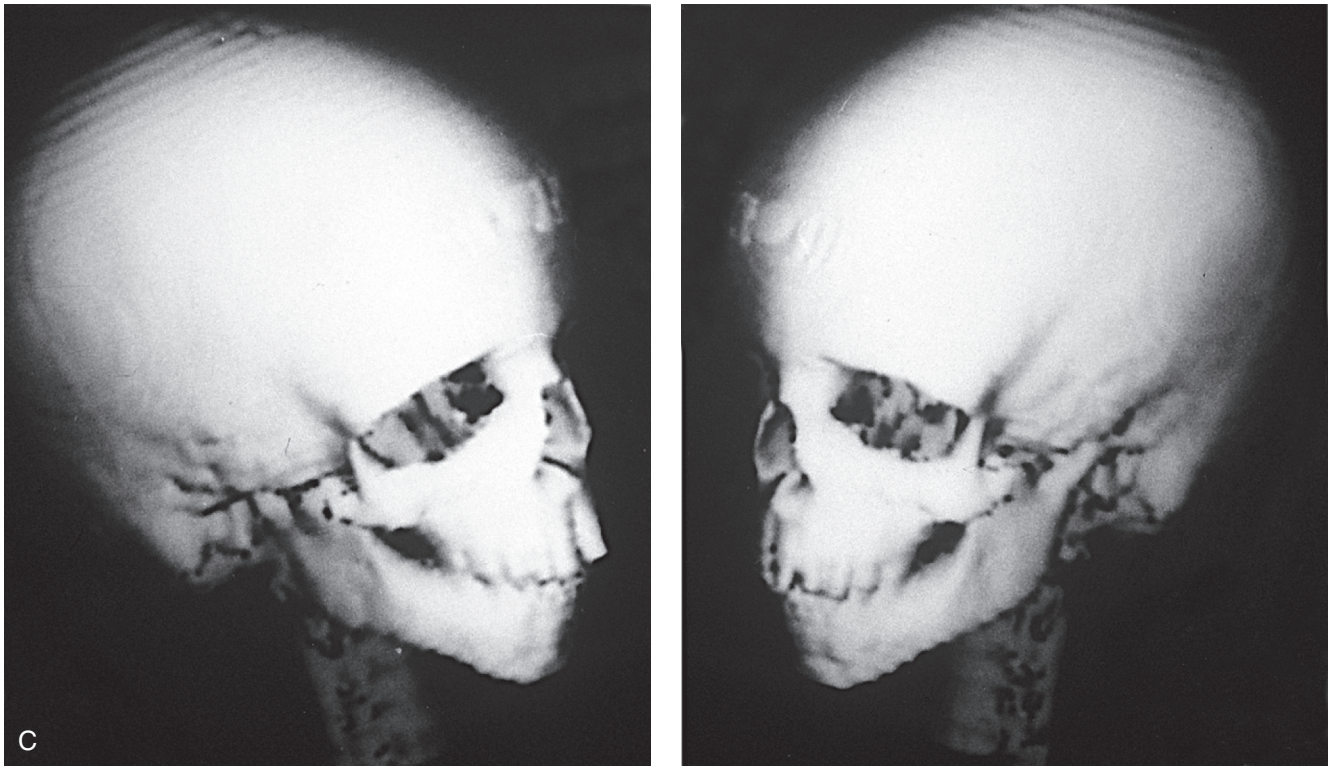


5 years of age (Kaban type II A)

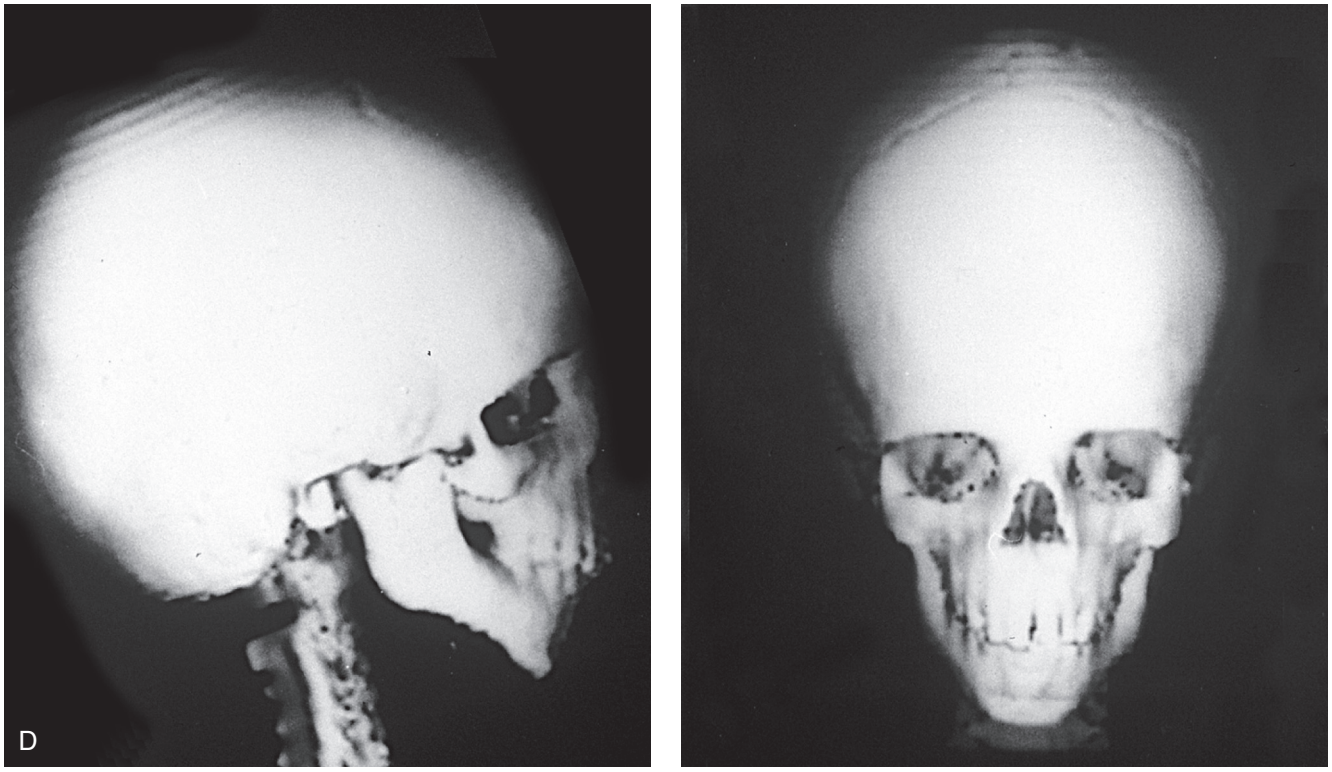
• **Figure 27-4** A girl who was born with Treacher Collins syndrome was followed up longitudinally at 1 and 5 years of age without treatment intervention. There is consistent growth of the craniofacial region without progression of the deformity. As a result of chronic nasal obstruction and an open-mouth posture, excess vertical facial growth can be seen. **A,** Frontal views at 1 and 5 years of age. **B,** Profile views at 1 and 5 years of age. **C,** Computed tomography scan views at 5 years of age. There is severe hypoplasia of the zygomatic complex with lateroinferior orbital deficiency. The mandible demonstrates a Type IIA malformation.

• **Figure 27-5** A child who was born with Treacher Collins syndrome was followed up longitudinally without treatment intervention. No progression of the facial deformity occurred with growth. **A,** Frontal views at 10 months and 6 years of age. **B,** Profile views at 10 months and 6 years of age.

Continued

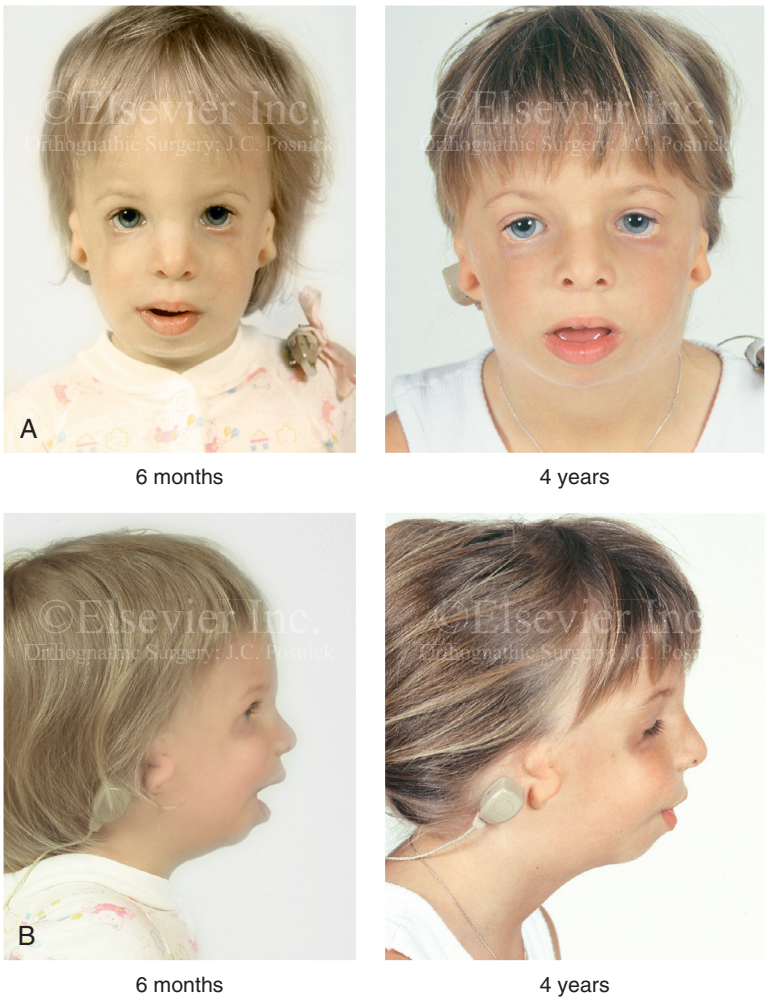


6 years of age (Kaban type II A)



6 years of age (Kaban type II A)

• **Figure 27-5, cont'd C,** and **D,** Computed tomography scans.



• **Figure 27-6** A child who was born with Treacher Collins syndrome was followed up longitudinally without treatment intervention. No progression of the craniofacial deformity has occurred. **A,** Frontal views at 6 months and 4 years of age. **B,** Profile views at 6 months and 4 years of age.

documented the relative stability of the anomalies without the progression of deformity at various ages.

To date, no convincing evidence demonstrates a clinically significant worsening of the TCS dysmorphology with facial growth. Early surgical interventions with mandibular osteotomies involving the use of either osteodistraction or standard orthognathic techniques are believed to adversely affect growth, thereby necessitating revision osteotomies at maturity. For this reason, waiting until the majority of jaw growth is complete before reconstructing the maxillomandibular components is recommended, if feasible. In any case, early mandibular surgery should not be expected to prevent the need for definitive maxillofacial reconstruction at maturity.

Classification of Temporomandibular Joint and Mandibular Malformation

The extent of TMJ–mandibular malformation in patients with TCS will influence the timing and techniques of reconstruction.[69,137] A lack of clinically relevant definitions of the presenting glenoid fossa–condyle–ascending ramus malformations has resulted in confusion and miscommunication. Kaban and colleagues revised and clarified the previously described Pruzansky classification system for the degree of TMJ–mandibular malformation observed in patients with hemifacial microsomia.[69,119] This classification is also useful to define the presenting anomalies and then to direct reconstruction in the patient with TCS (Fig. 27-7).

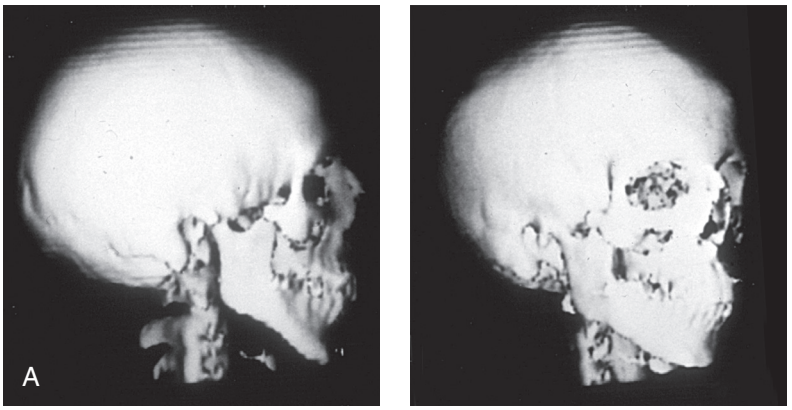
A Type I TMJ–mandibular malformation involves minimal hypoplasia of the glenoid fossa and the condyle–ascending ramus on each side. All of the skeletal components are present (see Fig 27-7, *A*). Mandibular retrognathia and a mild anterior open bite are likely to be present, but TMJ function is essentially normal. The muscles of mastication are intact, and the condyle and the glenoid fossa are also fully intact. There is no need or advantage to constructing a neo-condyle or neo-glenoid fossa.

A Type IIA TMJ–mandible malformation involves a moderate degree of glenoid fossa and condyle–ascending

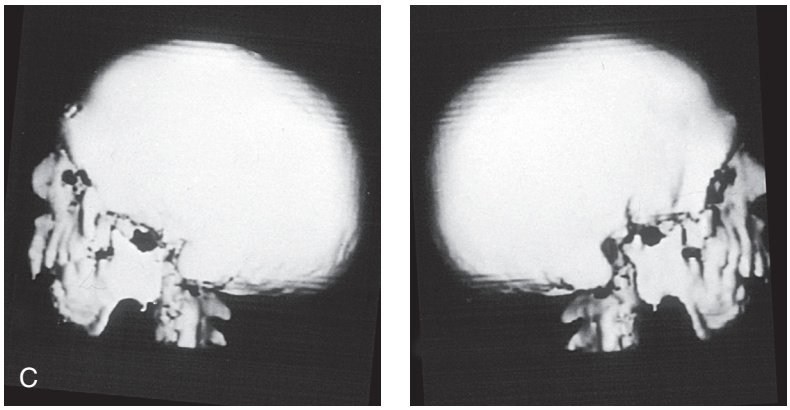
ramus hypoplasia (see Fig. 27-7, *B, C,* and *D*). The anatomic location of each joint is anterior and medial, but TMJ function remains satisfactory. One or more of the paired muscles of mastication are likely to be hypoplastic. By definition, in Type IIA malformations, the condyle and the glenoid fossa have function that is at a level deemed better than could reliably be achieved through TMJ replacement. Even in patients with more severe Type IIA malformations, when applying superior and posterior pressure to the chin, the condyles can be seated into a consistent location against the skull base–glenoid fossa. By completing ramus osteotomies, the proximal segments can be positioned into a stable location against the skull base, and the distal mandible can then be reoriented into the preferred location. This will avoid the need to construct neo-condyles (e.g., costochondral grafts). Coronoidectomies may be required to accommodate the short condyles.

The Type IIB TMJ–mandibular malformation involves severe hypoplasia of the condyles. The mandible is without a stable centric relation. The glenoid fossa may be hypoplastic, but they are deemed functional. The hypoplastic condyles do not consistently seat in the glenoid fossa, and the mandible functions within a limited range of motion. The extent of mandibular retrognathia and anterior open bite is marked. With Type IIB TMJ–mandibular malformations, the rudimentary condyles are deemed too far medially and anteriorly displaced to be useful. The construction of a neo-condyle ascending ramus with the use of a bone graft is required (see discussion to follow). Coronoidectomies and recontouring of the surgically advanced mandible may be required. Fortunately, this pattern of malformation occurs in only a small percentage of patients with TCS.

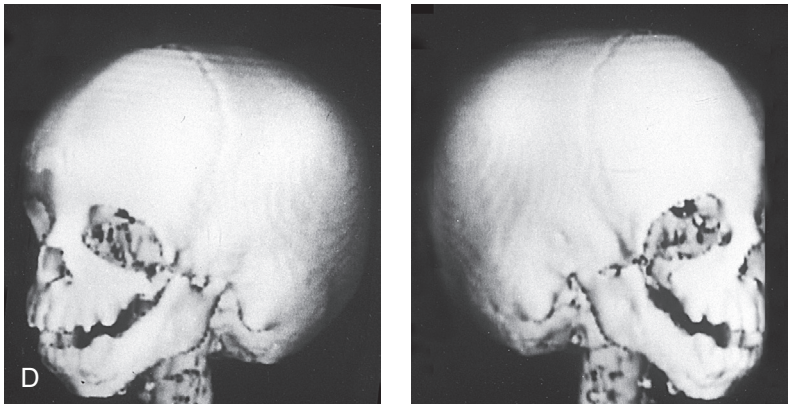
The Type III glenoid fossa–mandibular malformation involves deficiency to the extent that no posterior stop of the lower jaw against the cranial base is possible (see Fig. 27-7, *E*). The condyles and the ascending rami are not present. The disc, the TMJ capsule, and the glenoid fossa on each side are also not developed. Severe mandibular dysmorphology is present, including horizontal



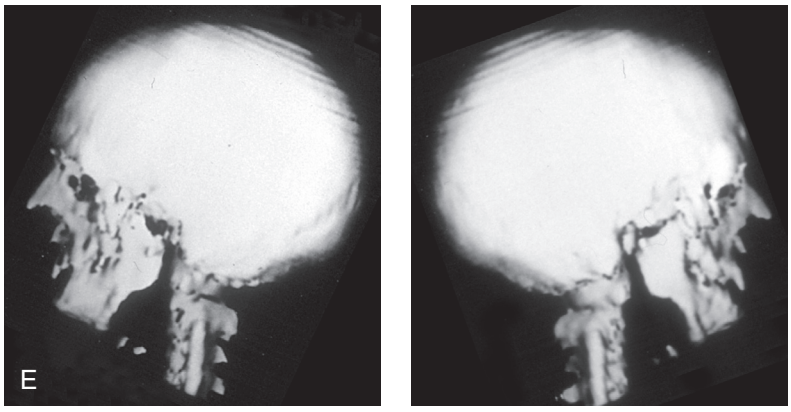
• **Figure 27-7** Kaban and colleagues described a classification system to define the degree of temporomandibular joint–mandibular malformation observed in patients with hemifacial microsomia. By definition, a Type IIB condylar deficiency will require the construction of a neo-condyle. The Type III malformation will also require neo-glenoid fossa construction. The same classification system is used for Treacher Collins syndrome. Craniofacial computed tomography (CT) scans demonstrate the classification system. **A,** CT scan views of a Type I mandibular malformation. **B, C,** and **D,** CT scan views of a Type IIA mandibular malformation.



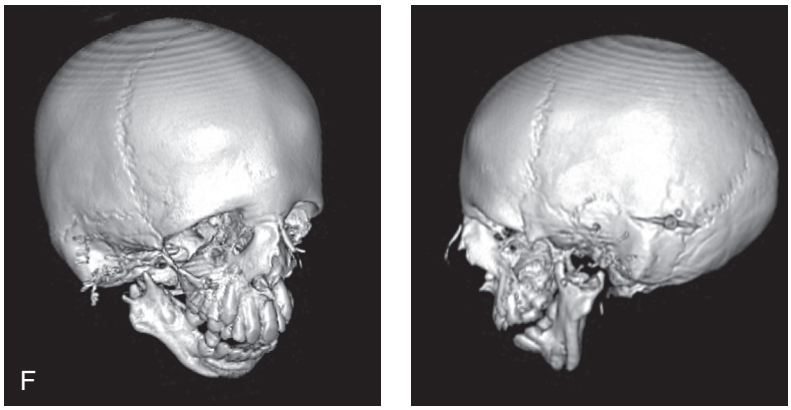
Kaban II A



Kaban II A



Kaban III



Kaban III

• **Figure 27-7, cont'd** **E** and **F,** CT scan views of a Type III glenoid fossa–mandibular malformation.

retrognathia and decreased posterior ramus height. A tracheostomy is generally required shortly after birth to manage the airway. Eventual construction of both a neo-glenoid fossa and a neo-condyle ascending ramus will be required on each side. Reconstruction with bone grafts (e.g., cranial grafts for the zygomatic complex and either costochondral or a vascularized fibula flap for the mandible) will be required (see discussion to follow).

Staging of Skeletal Reconstruction: Timing and Techniques

The reconstruction and rehabilitation of the Treacher Collins malformation must address unique and specific components of the anomalies, including the following: 1) the zygomatic and orbital region; 2) the maxillomandibular region; 3) the nasal region; 4) the soft-tissue envelope; 5) the external ears; 6) the external auditory canals; and 7) the middle-ear structures (Figs. 27-8 through 27-10).*

Zygomatic and Orbital Reconstruction

In previously published studies, we have documented the efficacy of reconstructing the malar and orbital deficiencies for moderate to severe forms of TCS with the use of full-thickness calvarial bone grafts that have been contoured three-dimensionally to form a new zygomatic complex (see Figs. 27-8 and 27-9 through 27-13).[113] The neo-zygoma is then inset and stabilized with microplate and screw fixation. Exposure for the reconstruction is provided by a coronal scalp incision without the need for additional periorbital incisions. Orbital floor defects are reconstructed with fixed split-thickness autogenous cranial bone. Cranial vault donor sites are repaired either with autogenous split cranial grafts or artificial material. For non-autogenous reconstruction of the small skull donor sites, we prefer the use of a titanium mesh base fixed to the adjacent skull. The mesh is then filled in with a bone substitute. Lateral canthopexies are completed through the coronal scalp incision and secured to each new lateral orbital rim. In our study, computed tomography scanning that was performed before surgery and immediately afterward demonstrated significant increases in lateral orbital wall length (depth), lateral orbital (rim) width, interzygomatic arch width, and zygomatic arch length for patients with TCS. The late (i.e., ≥1 year) postoperative scans document that these changes were maintained. No complications occurred during surgery, and repaired skull donor sites healed without clinical defect. Zygomatic and orbital morphologic improvement was achieved in all patients, with follow-up intervals ranging from 24 to 50 months (mean, 35 months) at the close of the study. Unfortunately, over time, I have observed that partial resorption of the zygomatic complex graft occurs in some patients.

*References 1-3, 5, 16, 21, 25, 81, 97-100, 105-107, 109, 110, 112, 114, 115, 121, 122, 127, 153, 151

We believe that reconstructing the malar and orbital deficiencies before the patient is 7 years old is not advisable unless severe corneal exposure problems occur and skeletal deficiency is believed to be the cause. By this age, the cranio–orbito–zygomatic bony maturation is nearly complete. Therefore, adult-sized cheekbones may be constructed and placed with limited concern about how further growth will effect the initial result. In addition, skull donor-site reconstruction is simpler than it is at a younger age, because the presence of bicortical cranial bone in those who are more than 7 years old allows for the splitting of adjacent bone to assist with reconstruction (see Figs. 27-11 and 27-12).

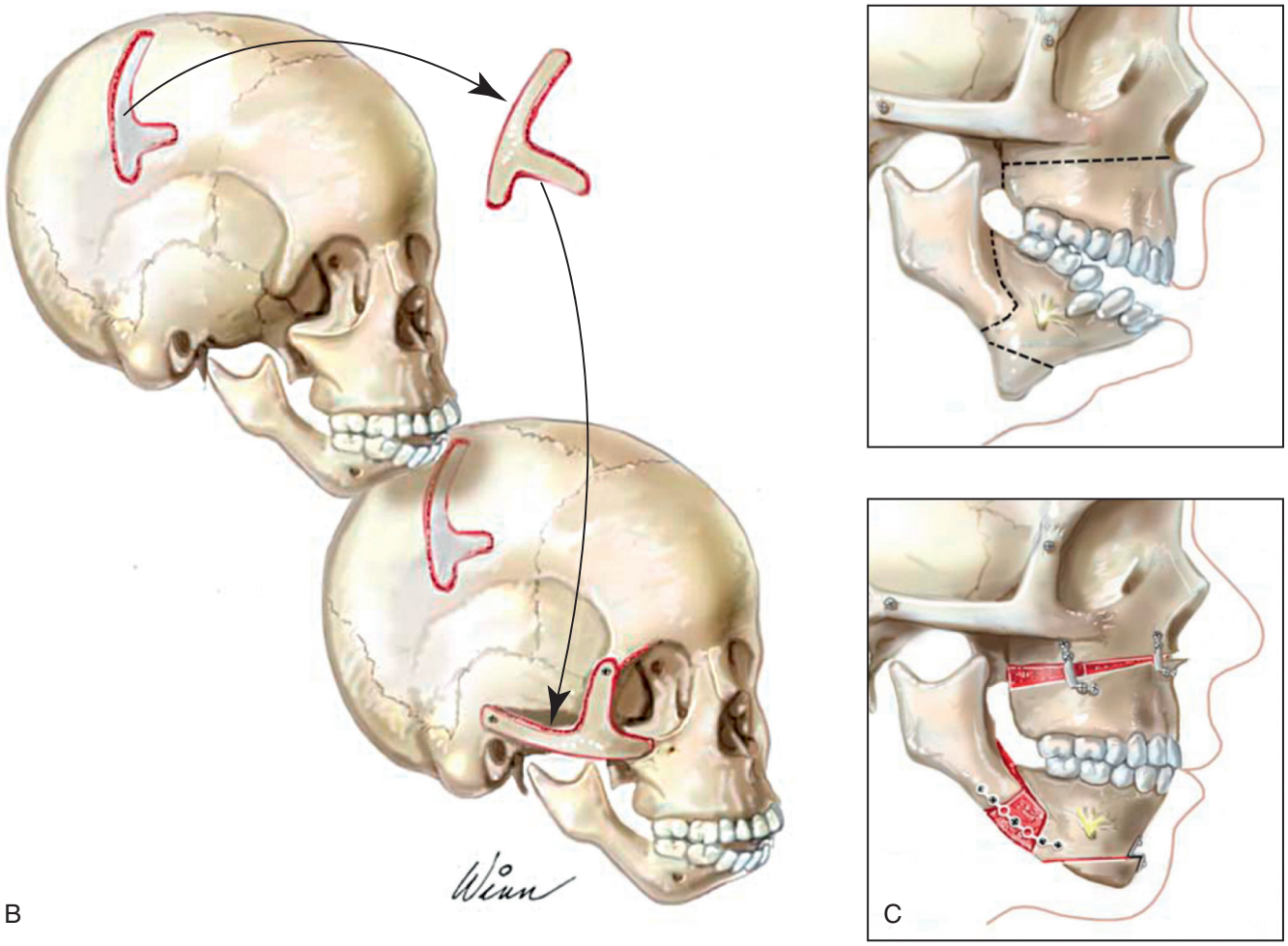
Other surgeons have recommended various methods and time frames for the zygomatic and orbital deformities associated with TCS.[20,45,60,113,125,149,150,152,167,169] We believe that the construction of the complete zygomatic complex from full-thickness autogenous cranial bone maintains volume and shape better than onlay grafts, which have been universally disappointing. Over time, onlay autogenesis bone grafts that have been placed in the craniofacial region (e.g., supraorbital ridge, zygoma, anterior maxilla, angle of mandible, chin) from all tried donor sites (e.g., skull, hip, rib) have demonstrated significant and unpredictable resorption. Furthermore, no commensurate growth (i.e., volume expansion) of the graft with the underlying bone (e.g., zygoma) should be expected. The belief that "even if the graft partially resorbs, at least something has been gained" should be abandoned; surface irregularities routinely result, which further detract from the baseline malformation.

Maxillomandibular Reconstruction

A primary consideration for the timing and technique of jaw reconstruction is to clarify any special airway needs. This will influence whether or not first-stage early mandibular reconstruction is necessary prior to growth maturity. The second consideration is to define the extent of TMJ–mandibular malformation. With Type I and IIA malformations, there is no need for glenoid fossa or condyle construction. In patients with these conditions, adequate TMJ anatomy is present so that mandibular reconstruction can be achieved through ramus osteotomies (e.g., sagittal split, inverted L, distraction [DO]) with the preservation of the working TMJ.

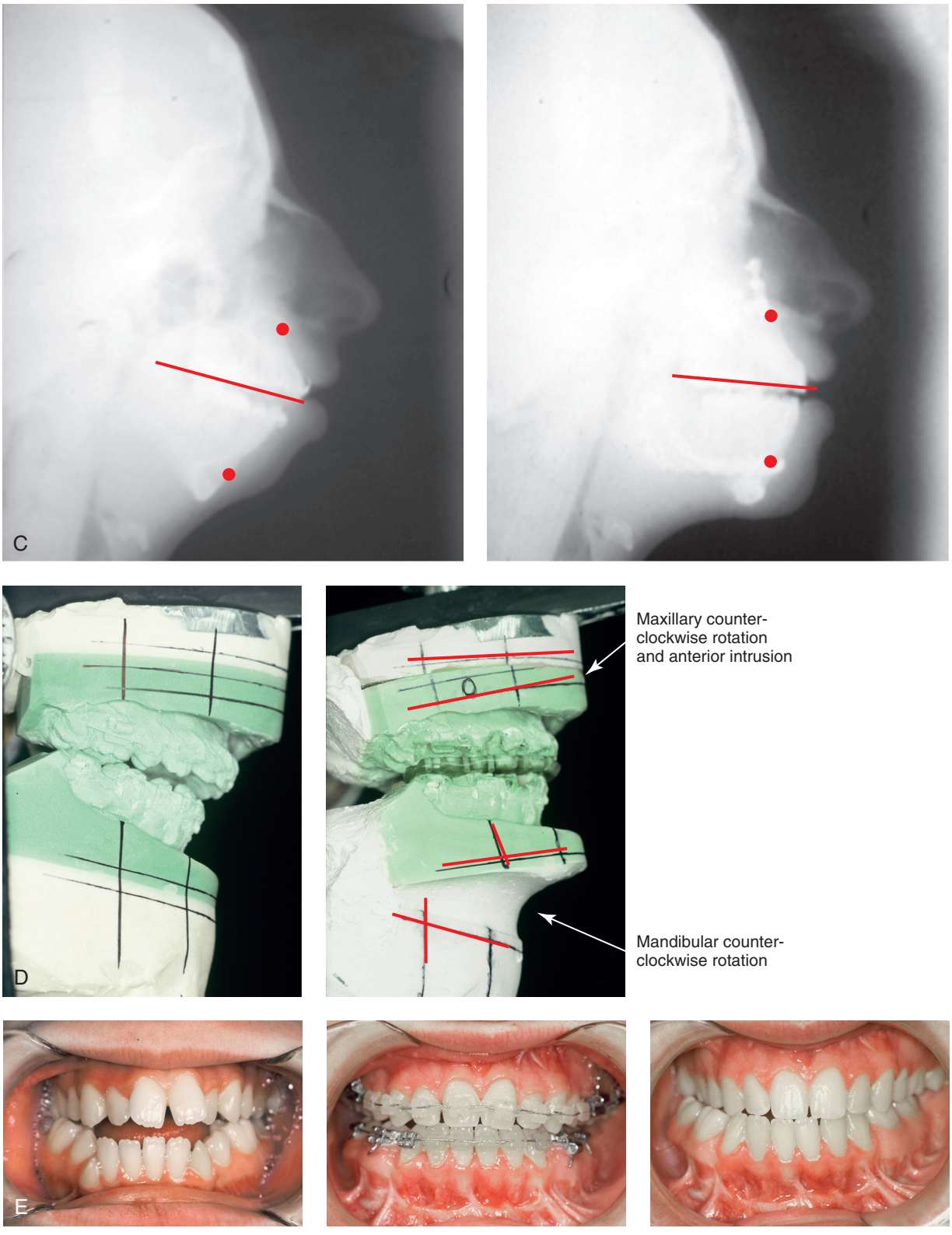
The basic dysmorphologies of the maxillomandibular complex that require reconstruction in patients with TCS are as follows: 1) altered facial heights (increased anterior lower facial height and decreased posterior facial height); 2) horizontal jaw deficiency (primarily in the mandible but also in the maxilla); and 3) chin dysplasia (increased vertical length and horizontal retrusion).[29,43,44,54,61,70,150,152,168] These malformations result in an excessive clockwise rotation of the maxillomandibular complex and an unfavorable A-point–to–B-point relationship when viewed in profile. In general, there is an Angle class II anterior open-bite malocclusion. Interestingly, some individuals will present with

Text continued on p. 1081



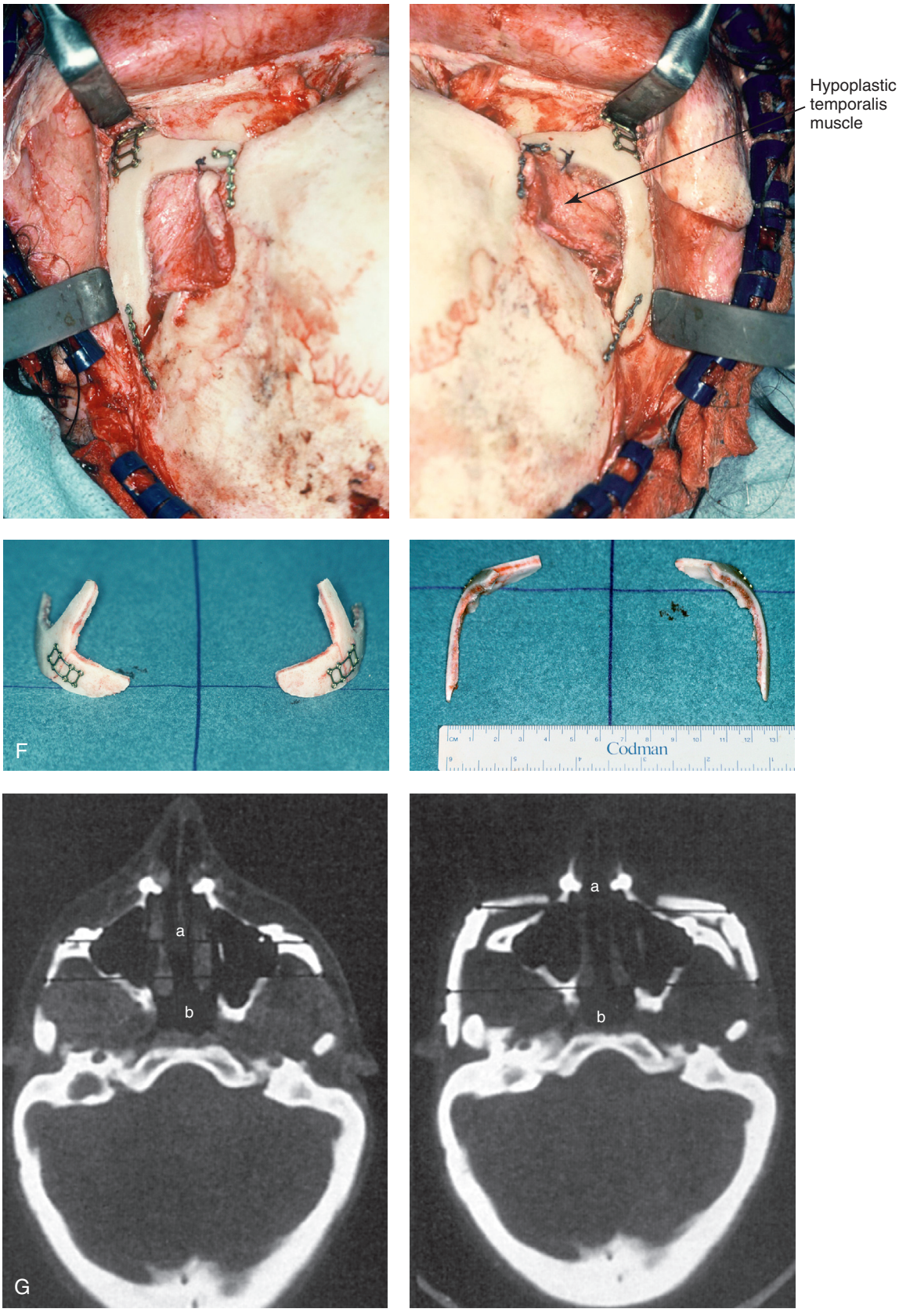
• **Figure 27-8 A,** A child who was born with Treacher Collins syndrome is shown during the mixed dentition. She has severe zygomatic complex and orbital deficiency as well as a Type IIA condylar malformation. **B,** Illustration of the location of the full-thickness cranial bone graft donor site for zygomatic complex reconstruction. The zygomatic complex graft is also shown inset at the recipient site. **C,** Illustrations of a maxillofacial complex seen in a teenager with Treacher Collins syndrome (Type IIA mandibular malformation). The cheekbone orbital reconstruction has previously been completed. The proposed maxillary, mandibular, and chin osteotomies are marked out and then completed. Counterclockwise rotation of the maxillomandibular complex is demonstrated as part of the reconstruction.

• **Figure 27-9** Additional images of the patient from Fig. 27-8 showing the stigmata of Treacher Collins syndrome. At the age of 17 years, the patient was referred to this surgeon and agreed to a staged reconstruction that included three operations. The initial preoperative orthodontic treatment included the extraction of mandibular first premolars with the retraction of the anterior dentition to remove compensations. Orthognathic surgery included a Le Fort I osteotomy (counterclockwise rotation and anterior maxillary intrusion); bilateral sagittal split ramus osteotomies (advancement and counterclockwise rotation); and an osseous genioplasty (vertical reduction and advancement). During a second operation, the patient underwent zygomatic complex reconstruction with fixed full-thickness autogenous cranial bone grafts as described in this chapter and as illustrated in Fig. 27-8. This was followed a third operation: septorhinoplasty (open approach) to in-fracture the nasal bones, reduce the dorsal hump (bone and cartilage), and reshape the tip, including septal (caudal strut) grafting (see Chapter 38). **A,** Right profile views before and after reconstruction. **B,** Left profile views before and after reconstruction.



• **Figure 27-9, cont'd** **C,** Lateral cephalometric radiographs before and after reconstruction. **D,** Articulated dental casts that indicate analytic model planning. **E,** Occlusal views before treatment, early after reconstruction, and 3 years after reconstruction. A degree of maxillary relapse (skeletal versus dental) has occurred but with a retained functional occlusion and favorable facial aesthetics.

Continued



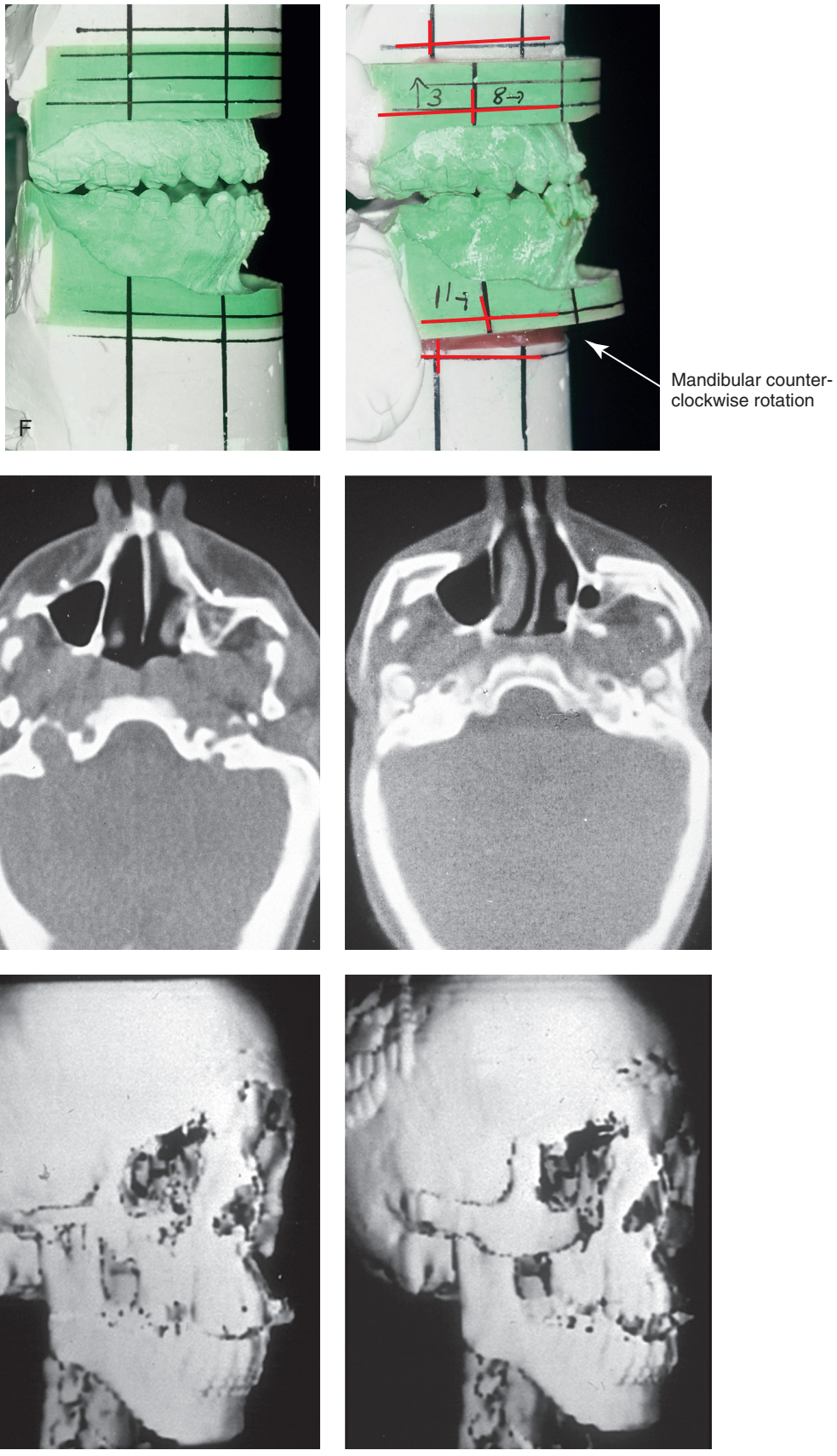
• **Figure 27-9, cont'd** **F,** Crafted autogenous cranial grafts shown before and then after inset into the zygomatic defects. The advancement of the hypoplastic temporalis muscles is also demonstrated. The soft-tissue hypoplasia involves multiple layers (i.e., skin, subcutaneous tissue, fascia, and fat tissue) and therefore can not be fully corrected simply by advancing the hypoplastic temporalis muscle. **G,** Axial computed tomography scan views shown through the zygomas before and after malar reconstruction indicating the improved interzygomatic buttress width, interzygomatic arch width, and zygomatic arch length. *A, From Posnick JC, Treacher Collins syndrome: Perspectives in evaluation and treatment, J Oral Maxillofac Surg 55:1120, 1997. F (bottom left, bottom right), G, From Posnick JC, Goldstein JA, Waitzman A: Surgical correction of Treacher Collins malar deficiency: quantitative CT scan analysis of long-term results,* Plast Reconstr Surg *92:12, 1993.*

• **Figure 27-10** A 16-year-old boy born with Treacher Collins syndrome with a Type I mandibular malformation was followed up longitudinally to confirm no progression of the deformity. He then underwent two operations, including orbitozygomatic and orthognathic reconstruction, as described previously. In preparation for orthognathic surgery, orthodontic treatment included maxillary first bicuspid extractions with the retraction of the anterior dentition to correct the arch form. The mandibular second molars are congenitally absent. The patient's orthognathic surgery included Le Fort I osteotomy (vertical intrusion and horizontal advancement); bilateral sagittal split ramus osteotomies (horizontal advancement); and an osseous genioplasty (horizontal advancement). He is shown before and after orbitozygomatic and orthognathic surgery. **A,** Frontal views before and after orthognathic surgery. **B,** Left profile views before and after orthognathic surgery. **C,** Right profile views before and after reconstruction. *Continued*

Prior to treatment

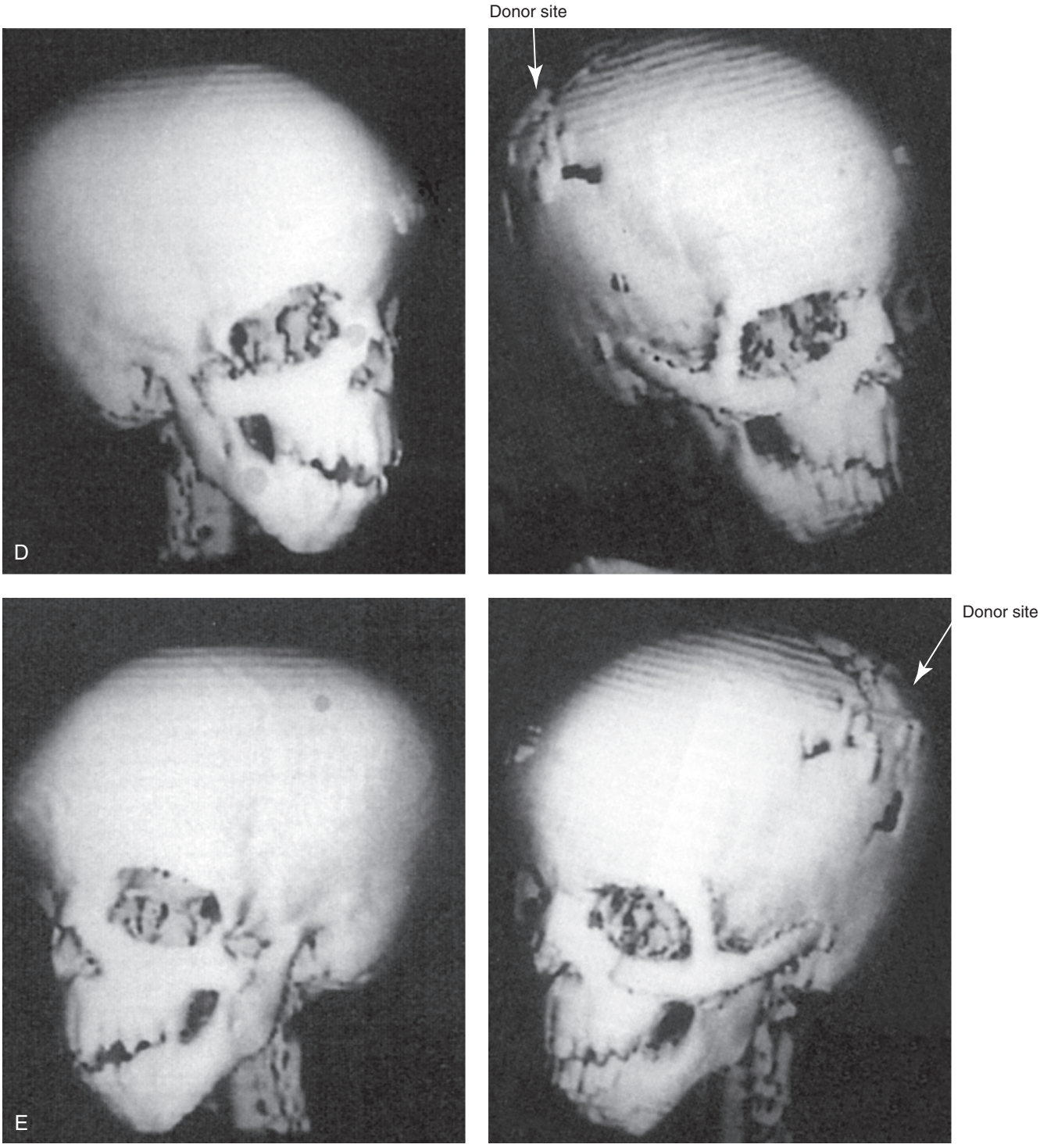
After treatment with maxillary bicuspid extractions

• **Figure 27-10, cont'd D,** Occlusal views before and after orthognathic surgery. Note that, with the upper bicuspid extractions and the absent mandibular second molars, the maxillary second molars are no longer in occlusion. **E,** Palatal views before and after orthognathic surgery.



• **Figure 27-10, cont'd F,** Articulated dental casts that indicate analytic model planning. **G,** Axial computed tomography slices before and after malar reconstruction. **H,** Three-dimensional computed tomography scans before and after malar reconstruction (i.e., before orthognathic surgery).

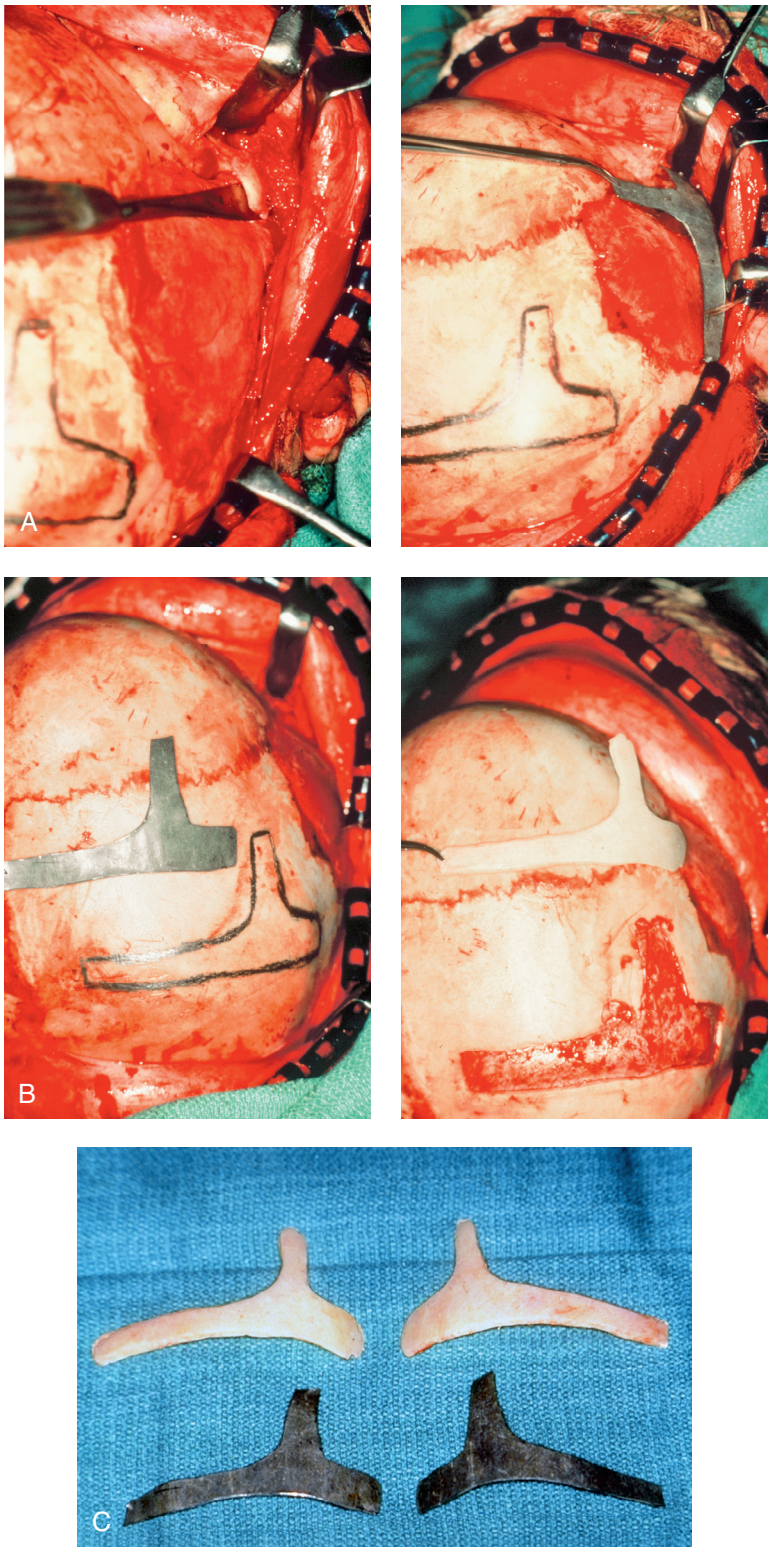
• **Figure 27-11** A 6-year-old boy who was born with Treacher Collins syndrome and a Type IIA mandibular malformation is shown before and after zygomatico–orbital reconstruction carried out by the technique described. He also underwent bilateral otoplasty to set the ears back (see Chapter 39). **A,** Frontal views with smile before and after reconstruction. **B,** Oblique views before and after reconstruction. **C,** Worm's-eye views before and after reconstruction.



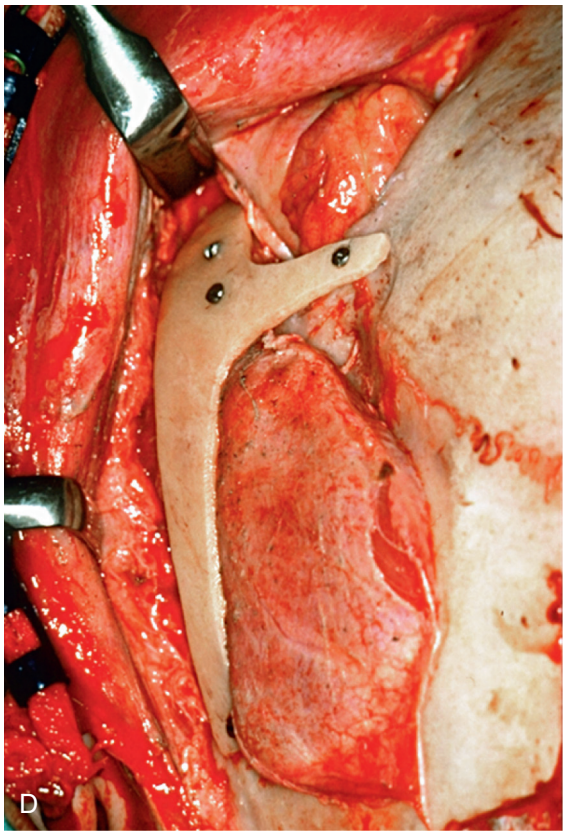
• **Figure 27-11, cont'd D** and **E,** Computed tomography scan views before and after reconstruction.

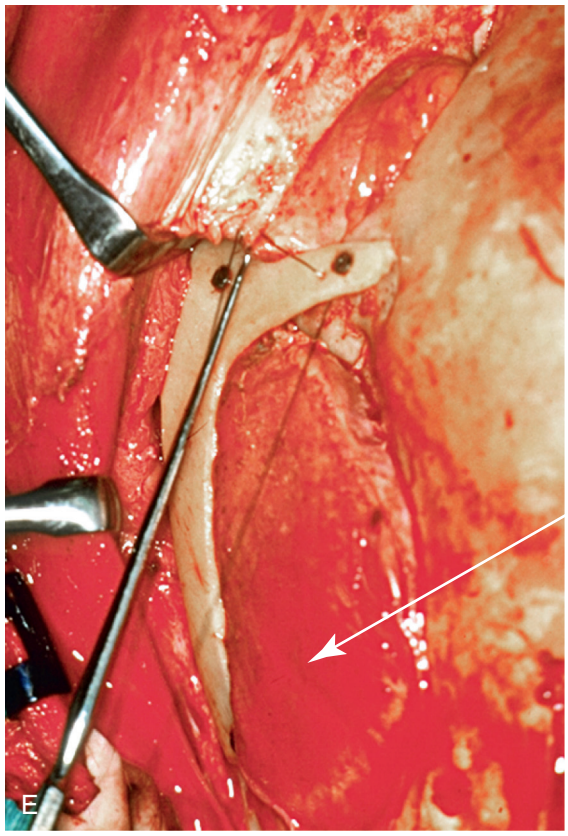
either a Class I or Class III relationship. These aspects must be addressed with the incorporation of counterclockwise rotation of the maxillomandibular complex to increase the posterior airway space, to correct the malocclusion, and to restore facial balance.

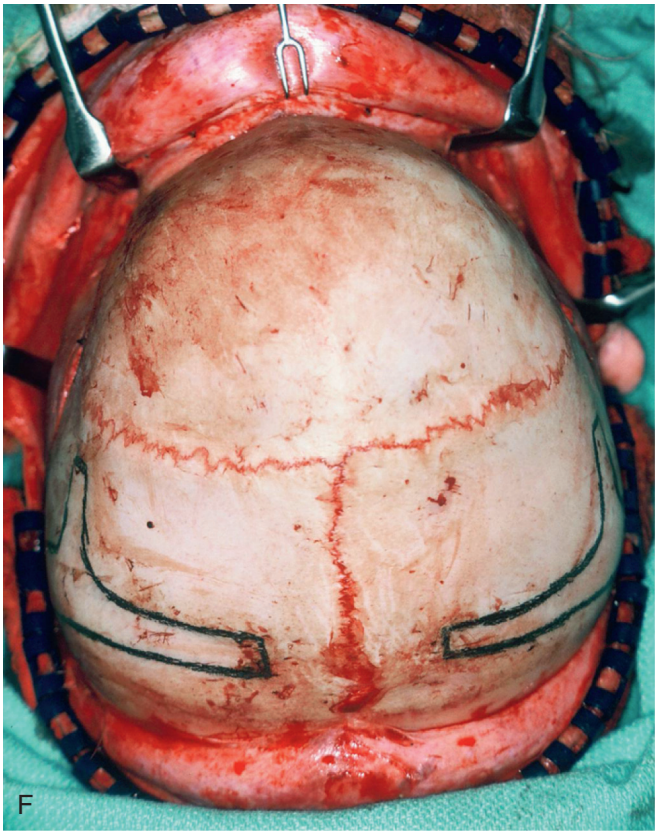
The most favorable aesthetic results are generally achieved in patients who undergo definitive reconstructive jaw surgery only after early maxillofacial skeletal maturity (i.e., after the age of 13 to 15 years) and when these procedures are carried out in combination with effective definitive orthodontic treatment during the permanent dentition (see Figs. 27-8 through 27-10). Mandibular and possibly maxillary premolar extractions are often required to orthodontically unravel dental root crowding, to normalize the

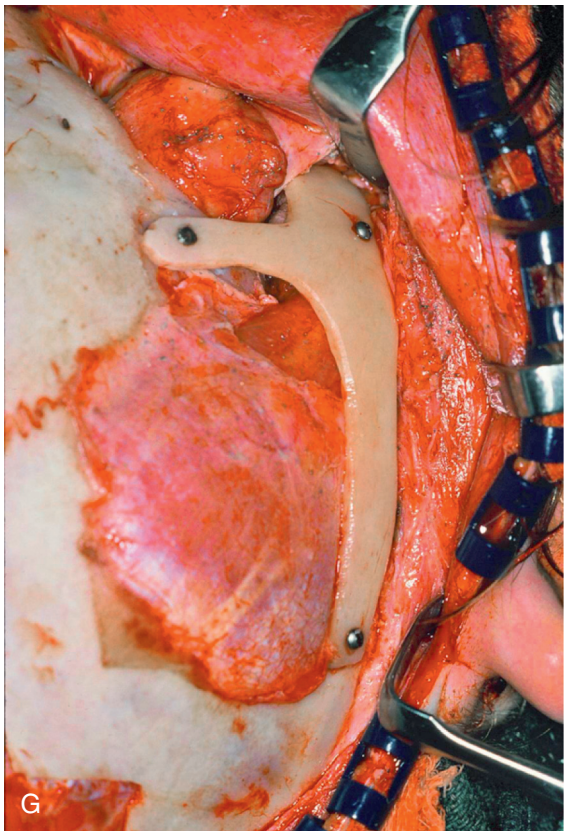


• **Figure 27-12** Intraoperative views demonstrate the technique for zygomatico–orbital reconstruction in a 5-year-old child as it would be carried out for a patient with Treacher Collins syndrome. Working through a coronal (scalp) incision, the rudimentary zygomatic and orbital structures are exposed. Full-thickness cranial bone grafts are harvested, and each zygoma is crafted from such a graft. Each zygoma graft is inset and stabilized with microplates and screws. The orbital defects are reconstructed with cranial bone graft, and the orbital rims are reshaped with a rotary drill. Lateral canthopexies are completed through the coronal scalp incision and fixed to the new frontozygomatic suture regions. **A,** A view of the dissected rudimentary zygomatic remnant; the periosteal elevation is deep to the remnant. A template of the proposed zygomatic complex has been made. **B,** The template is taken to the mid-skull region, and its outline is marked out for craniotomy. The location for graft harvesting is posterior to the coronal suture, lateral to the sagittal suture, and anterior to the lambdoid suture. Through a craniotomy, the full-thickness cranial bone graft is harvested for zygomatic complex reconstruction. The exact same procedure is completed on the contralateral side. **C,** The two full-thickness cranial bone grafts are then crafted. The two templates and the two full-thickness crafted cranial bone graft–zygomatic complexes are shown.



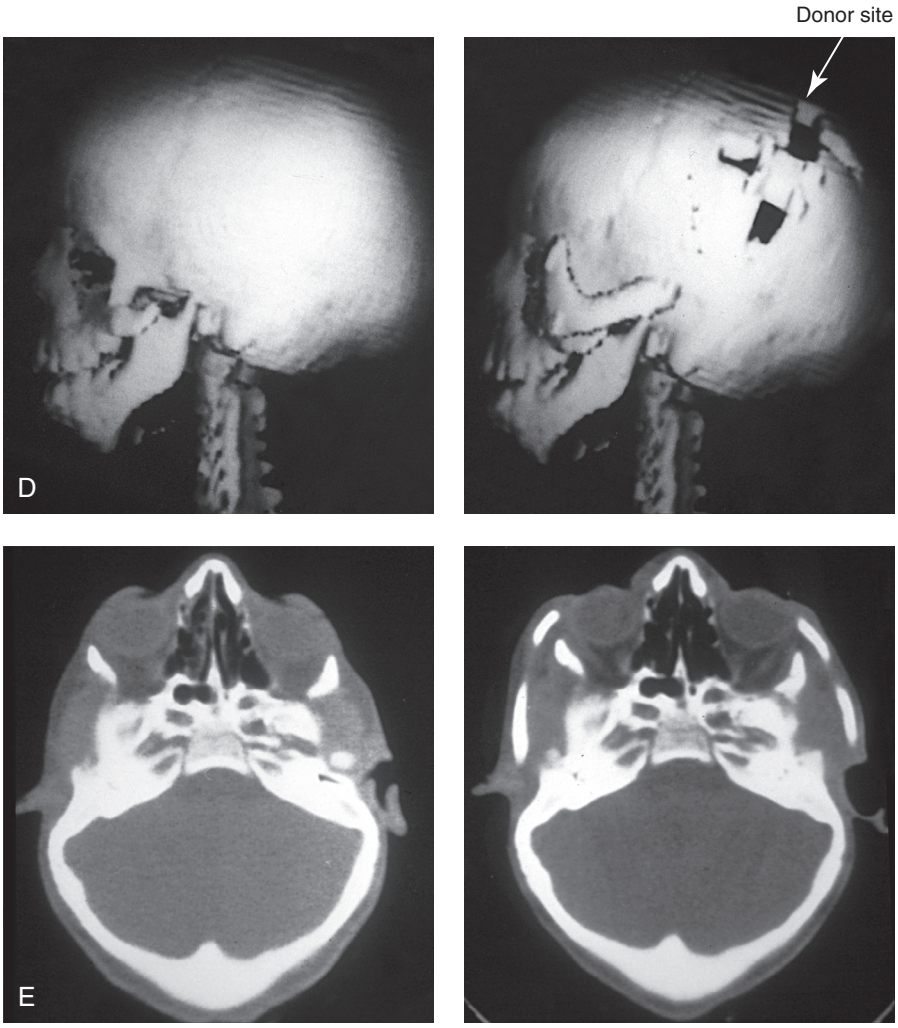






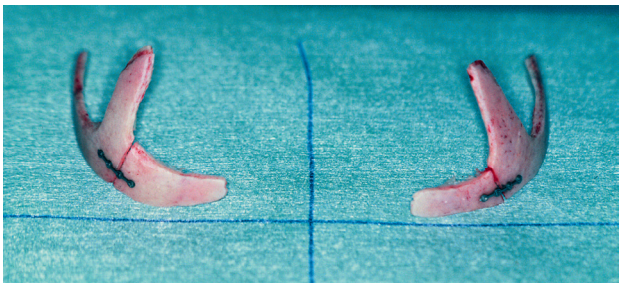
• **Figure 27-12, cont'd** **D,** The full-thickness cranial bone graft is fixed in place with microscrews. **E,** The lateral canthus is identified through the coronal incision and a 28-gauge stainless steel wire is passed through. In the patient shown here, the temporalis muscle has minimal hypoplasia. Thus, minimal temporal hallowing is expected after zygomatic reconstruction. The lateral canthus is pexed to the new frontozygomatic region. **F,** The locations for graft harvesting are outlined. **G,** The right side cranial graft is shown in place. *A,B, From Posnick JC, Goldstein JA, Waitzman A: Surgical correction of Treacher Collins malar deficiency: Quantitative CT scan analysis of long–term results.* Plast Reconstr Surg *92:12, 1993.*

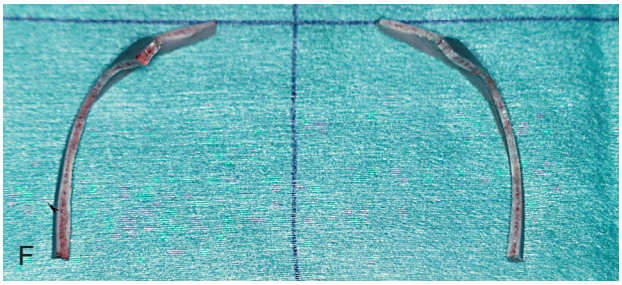
• **Figure 27-13** A 7-year-old girl who was born with Treacher Collins syndrome is shown before and after zygomatico–orbital reconstruction that was carried out via the technique described in this chapter. **A,** Frontal views before and after reconstruction. **B,** Oblique views before and after reconstruction. **C,** Worm's-eye views before and after reconstruction.

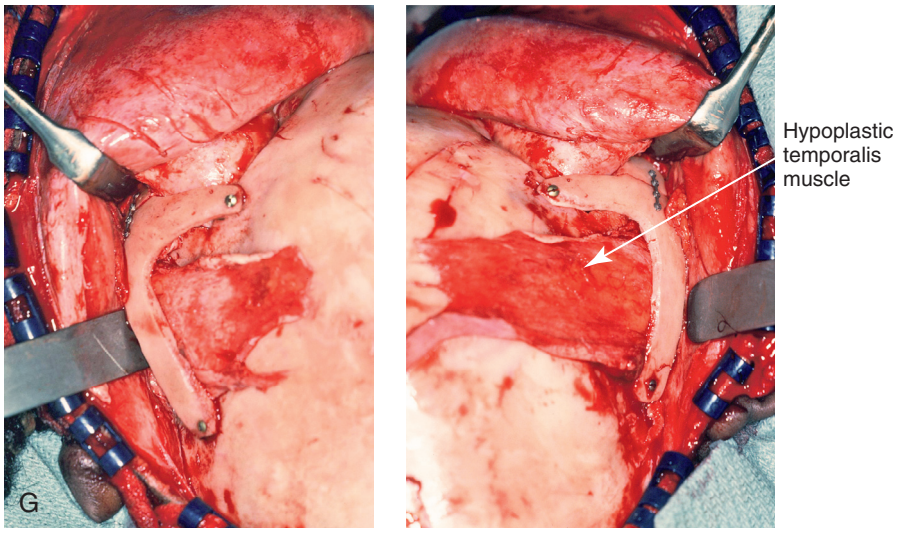


• **Figure 27-13, cont'd D,** Computed tomography scan views before and after zygomatico–orbital reconstruction. **E,** Axial computed tomography scan views through the mid orbits before and after cranial graft reconstruction.

Continued







• **Figure 27-13, cont'd F,** Intraoperative views of full-thickness crafted zygomatic complex before inset. **G,** Left and right intraoperative views of zygomatic complex reconstruction fixed with microplates and screws. The hypoplastic temporalis muscles will be rotated anteriorly as part of the reconstruction. A degree of temporal hallowing is to be expected as a result of the congenital soft-tissue hypoplasia. It is important to not over correct the zygomatic–orbital reconstruction, because this would accentuate the soft-tissue deficiency. *A (left), From Posnick JC, Treacher Collins syndrome: Perspectives in evaluation and treatment, J Oral Maxillofac Surg 55:1120, 1997. A (right), From Posnick JC, Goldstein JA, Waitzman A: Surgical correction of Treacher Collins malar deficiency: quantitative CT scan analysis of long-term results,* Plast Reconstr Surg *92:12, 1993.*

inclination of the anterior teeth, and to fully decompensate the occlusion in preparation for the effective aesthetic and airway repositioning of the jaws (see Fig. 27-7, *A, B, C,* and *D*). The surgical technique involves sagittal ramus osteotomies of the mandible in combination with a Le Fort I (maxillary) osteotomy and an oblique osteotomy of the chin. The incorporation of counterclockwise rotation of the maxillomandibular complex is generally required to successfully accomplish certain objectives. Any intranasal airway obstructions are simultaneously managed (e.g., septoplasty, inferior turbinate reduction, recontouring of the nasal floor, widening of the aperture).

Type IIB mandibular malformations will require surgical construction of the congenitally missing condyle. Osteotomies alone—with the immediate repositioning or gradual DO of skeletal segments—will not be adequate. For the Type IIB malformation, despite inherent limitations, mandibular reconstruction with a costochondral graft remains the preferred alternative when feasible (i.e. this will avoid the need to harvest fibula composite flaps).

The incorporation of the counterclockwise rotation of the entire maxillomandibular complex will also be required to achieve facial balance and an adequate airway. Effective reconstruction can only be predictably carried out with neo-condylar construction followed by maxillary and mandibular osteotomies at or near skeletal maturity. Intranasal procedures to open the airway (i.e., septoplasty, inferior turbinate reduction, recontouring of the nasal floor and walls) are also carried out at the time of definitive jaw reconstruction.

The Type III TMJ–mandibular malformation will require surgical construction of the congenitally missing glenoid fossa and condyle on each side (see Fig. 27-7, *E*). Construction of the neo-glenoid fossa is required and best carried out as part of the zygomatic and orbital reconstruction (see the discussion of zygomatic–orbital reconstruction earlier in this chapter). For the Type III condyle-ascending ramus malformation, construction of a neo-condyle on each side is also required. Mandibular osteotomies alone, with the immediate repositioning or gradual distraction of skeletal segments, will not be adequate. Despite limitations, neo-condylar construction with a costochondral graft remains the preferred alternative when feasible. Occasionally the combined extent of the deficiency of the skeleton and the soft tissues will require the microvascular transfer of a crafted fibula composite flap for each side (i.e., bone and soft tissue). The advantages of a vascularized fibular composite flap are obvious but the donor site morbidity (i.e., need for bilateral lower extremity harvesting) and recipient site requirements (e.g., need for bilateral neck microvascular anastomosis) are significant. In either case, at operation, the distal mandible is repositioned anteriorly to the preferred position of the maxilla. This is assisted by a prefabricated interocclusal acrylic splint, with the distal mandible secured by intermaxillary fixation. The proximal mandible on each side is then constructed with an autogenous costochondral graft or a fibula composite microvascular flap. Contouring of the native mandible with a rotary drill before placing the rib or fibula graft on the lateral side of or adjacent to the edge of the distal mandible is generally required to avoid lateral flaring. Fixation of the graft to the native distal mandible is done with an extended titanium plate from the graft forward along the inferior border of the body of the distal mandible on each side. Graft placement and fixation are generally carried out through an extraoral neck incision. The limited use of intraoral incisions in the ramus regions during this procedure is preferred to minimize the incidence of infection and wound breakdown with subsequent graft loss or TMJ ankylosis.

Indications for First-Stage Mandibular Reconstruction in the Newborn

TCS is a clefting syndrome that involves both sides of the face (i.e., the first and second branchial arches) in which upper airway anomalies occur with both regularity and variation. Hypoplasia of the mandible is one of the constant but variable components of the syndrome.* Individuals with TCS are known to be at risk for sleep apnea, which has been reported to occur in more than 25% of affected individuals. For those with OSA who are tracheostomy dependent, an alternative method of treatment may include the sagittal advancement of the mandible to bring the tongue forward, to create space in the oral cavity, and to open up the retromandibular airway. DO techniques to gradually advance and then hold the mandibular segments until consolidation occurs have been used to improve the airway for almost two decades.* The use of this technique has been reported by several authors as an effective method of decannulating otherwise tracheostomy-dependent individuals with TCS. The implications of this early mandibular advancement approach for long-term airway success and as a definitive jaw reconstruction option for patients with TCS have undergone little critical analysis.[37]

Anderson and colleagues reported the long-term results of mandibular lengthening with a DO approach that was carried out during the patient's childhood for the management of airway obstruction in an individual with TCS.[2] They documented that the airway improved in a 6-year-old patient with TCS after DO of the mandible to the extent that decannulation and the early resolution of airway obstruction were achieved. They showed that the minimum cross-sectional airway in this patient increased during the period of distraction but did not reach age-adjusted normal values. Subsequent serial scans over 10 progressive years of growth demonstrated that the minimal cross-sectional airway did not increase further. Specifically, measurements of the cross-sectional area that were taken at a fixed point of the upper airway (i.e., the base of the C2 vertebral body) demonstrated no increase in the cross-sectional area during growth. The published longitudinal case study from these authors demonstrates a failure of a sustained satisfactory cross-sectional area of the upper airway with chronologic age. They also used three-dimensional CT scans to document that mandibular growth continued in the same dysmorphic TCS pattern as it had before the distraction intervention. This resulted in an abnormally steep (clockwise rotated) mandibular plane angle. The combination of an increasingly steep mandibular plane angle and the failure of the distraction procedure to produce long-term positive horizontal projection was believed to have created the skeletal basis for ongoing upper airway obstruction. The authors conclude that a mandibular DO procedure carried out in a child with TCS who is experiencing OSA maybe useful for opening the retromandibular airway. However, the initial improvement in the upper airway did not continue during chronologic growth. The clinical consequences were the reemergence of the symptoms of OSA that required further treatment (i.e., continuous positive pressure airway mask at night or tracheostomy). It is also known that a mandibular

*References 17, 21, 47, 48, 53, 59, 61, 67, 70, 71, 90-93, 131, 134, 136, 139, 141, 142, 160, 166

lengthening procedure carried out during early childhood will have other detrimental effects, including injury to the developing teeth; deformational growth of the mandible; facial soft-tissue scarring; injury to the inferior alveolar nerve; a significant incidence of TMJ ankylosis (both bony and fibrous); perioperative cardiovascular and respiratory complications in some patients; expense to the family and medical health care system; and the need for definitive jaw reconstruction at maturity.[7] Despite almost two decades of accumulated worldwide experience, the role of mandibular DO in the treatment protocol for infants and children with TCS remains to be determined through long-term outcome studies. In the absence of sound data, the use of mandibular osteotomies and lengthening procedures—by whatever means—during infancy and childhood should be used cautiously and selectively.

Indications for First-Stage Mandibular Reconstruction during the Mixed Dentition

When a first-stage mandibular reconstruction during the mixed dentition for the Type IIB condylar malformation is felt to be indicated, it is preferably carried out after the child is 7 to 10 years old and only after the eruption of the mandibular permanent first molars.[105-110,112,114,115] Unfortunately, a first-stage early mandibular reconstruction will not provide a long-term resolution of the mandibular and other maxillofacial deformities.* Problems with costochondral graft overgrowth, undergrowth, and asymmetrical growth as well as with infection and TMJ ankylosis continue to plague the reconstructive surgeon (see Chapter 26). The final orthognathic correction of the upper jaw, the lower jaw, and the chin region will be required later, after early skeletal maturity (i.e., after 13 to 15 years of age).

The rationale for postponing the correction of the maxillomandibular deformity until the time of early skeletal maturity (i.e., 13 to 15 years of age) is the same as that for patients with standard dentofacial deformities, for those with other associated syndromes, and for those with a cleft lip and palate jaw deformity. The reasons that mandibular surgery during the mixed dentition is generally not preferred include the following: 1) to avoid injury to the developing permanent dentition and the inferior alveolar nerves[7,164]; 2) to avoid soft-tissue (cutaneous) scarring; 3) to limit perioperative airway and infection complications; 4) to limit the patient's negative psychosocial memories; and (5) to avoid any iatrogenic deformity of the TMJs and the mandible that may add difficulty to the achievement of a long-term favorable reconstruction.

In addition, safe and effective osteotomies in the malformed upper jaw and chin are not carried out during childhood as a result of the position of the developing teeth and the expected induced growth restrictions that would occur. The literature now confirms a lack of continued horizontal maxillary growth after early Le Fort I osteotomy. In addition, the favorable surgical placement of the jaws during the mixed dentition to ensure long-term functional occlusion and enhanced aesthetics is not realistic.

In individuals with TCS, a Le Fort I osteotomy will be required to correct vertical, horizontal, and transverse maxillary deformities. The maxillomandibular complex invariably benefits from counterclockwise rotation for the correction of pitch orientation. Intrusion of the anterior maxilla is generally needed to establish a more normal upper-lip–to–tooth relationship during smile and in repose. The posterior maxillary height may require lengthening, but more frequently it can remain close to a neutral position. The planned maxillary plane change (i.e., pitch orientation) is carried out to achieve appropriate counterclockwise rotation of the mandible and the chin. The objectives are 1) to normalize the anterior facial height; 2) to open the upper airway; and 3) to establish an aesthetically improved A-point–to–B-point relationship when the patient is viewed in profile. In 1993, Rosen documented the long-term stability of the counterclockwise rotation of the lower jaw when this is required to improve function and enhance the facial aesthetics in patients with mandibular micrognathia. Others have carried out similar clinical studies that have confirmed the long-term skeletal stability of mandibular counterclockwise rotation as part of an orthognathic correction.[127,168]

For the Type I and IIA mandibular malformations, bilateral intraoral sagittal split osteotomies are generally the preferred ramus procedures, because they allow for effective horizontal advancement, counterclockwise rotation of the mandible (without the need to actually lengthen the posterior facial height); and sufficient bone contact across each osteotomy site (see Fig. 27-8, *C*). Stabilization at each mandibular osteotomy site is accomplished with titanium plates and screws as needed. Interposed (e.g., autogenous iliac) corticocancellous bone graft is used when required.

For the Type IIB and Type III rib or fibula graft-constructed mandible that was created earlier during the patient's life, splitting in the ramus region at the time of definitive reconstruction is preferred when feasible. The degree of bone contact across the osteotomy site after the advancement of the distal mandible is generally limited in these circumstances. The need for an interpositioned corticocancellous bone graft (e.g., autogenous iliac) should be anticipated (see Chapter 28).

For most patients with TCS, DO osteogenesis techniques have failed to demonstrate lower morbidity or improved soft-tissue response as compared with standard osteotomies.* Data have indicated significant relapse during the consolidation phase of DO techniques and no ongoing jaw growth when the procedure is carried out during childhood. Kaban and colleagues and others continue to refine the application of curvilinear DO for the correction of the

*References 1, 2, 16, 29, 33, 38, 47, 48, 53, 61, 67, 70, 71, 90-94, 108, 123, 131, 136, 139, 141, 142

*References 1, 2, 16, 29, 33, 38, 47, 48, 53, 61, 67, 70, 71, 80, 90-94, 108, 123, 131, 136, 139, 141, 142, 163

complex mandibular deformities of severe forms of TCS.[69] Despite their best efforts when using DO techniques, reliable three-dimensional vector control to achieve preferred proportions and symmetry of the osteotomized skeletal segments remains a work in progress. For these reasons, until DO technology and its clinical applications improve, we prefer the standard orthognathic correction of the maxillomandibular imbalance in patients with TCS when feasible.

Nasal Reconstruction

The bridge of the nose in the adult with TCS often has mild to moderate increased width, and a mid-dorsal hump is always present. The length of the nose is usually normal, but it seems long as a result of the imbalance with the upper and lower skeletal thirds of the face; Also, the tip often droops and lacks the preferred projection. Most patients benefit from a rhinoplasty that includes osteotomies with in-fracture; the conservative reduction of the skeletal and cartilaginous dorsal hump; the removal of the cephalic portions of the lower lateral cartilages; and septal cartilage grafting (i.e., a caudal strut) sutured to the anterior septum to improve tip projection and definition. To achieve the most favorable aesthetic results, it is best to postpone the rhinoplasty until after effective orthognathic procedures have been performed. Exposure is provided through an open (i.e., columella-splitting incision) technique (see Chapter 38 and Fig. 27-9).

Facial Soft-Tissue Reconstruction

Despite several decades of well-intentioned surgical attempts to improve the soft-tissue deficiencies of the eyelid–adnexal regions, few aesthetically pleasing soft-tissue eyelid reconstructive results in patients with TCS have been reported.[64,82,83,129,135,143,148,156] The transposition of pedicled upper-eyelid-skin–muscle flaps to deficient regions of the lower eyelid is not technically difficult; unfortunately, the adnexal scarring that inevitably occurs often results in an "operated" look that generally detracts from any positive advantages of the procedure. The placement of full-thickness skin grafts to the lower eyelids is another frequently mentioned option. It predictably leaves a "patchy" look, and it should be reserved only for patients with recalcitrant corneal exposure problems. The lateral canthi are inferiorly displaced as a result of both orbital dystopia and hypoplasia of the lateral canthi and other components of the eyelid structures. Although the correction of the orbital dystopia and direct lateral canthopexies are helpful and should be carried out, they rarely normalize the multilevel soft-tissue hypoplasia of the specialized adnexal structures (i.e., skin, cartilage, eyelashes, ligament, fascia, and tendon). For these reasons, it is best to consult with an experienced oculoplastic surgeon when considering even minor eyelid procedures for patients with TCS.

The observed temporal fossa hollowing that is frequently encountered with TCS is also a reflection of multilevel hypoplasia that includes the skin, the temporoparietal fascia, the temporalis muscle, and the decreased bitemporal width of the anterior cranial vault. For this reason, transposing pericranial or temporoparietal congenital hypoplastic flaps will have limited utility for the management of either temporal or adnexal region subcutaneous deficiency.

When the procedure is meticulously designed and expertly executed, the transfer of soft-tissue flaps from other body regions, with microvascular reanastomosis, should be considered for reconstruction of TCS.[64,82,83,129,135,143,148,156] When this level of facial soft tissue replacement is required, parascapular free flaps have become the workhorse since they were first described by Dos Santos. Siebert and colleagues found that the parascapular flap allows for the reasonable correction of contour defects in the lateral aspects of the face; this yields improved aesthetic results in selected patients with TCS by providing vascularized soft tissue in the subcutaneous plane while minimizing scarring.[135] Fortunately, hypoplasia of the centrally located soft tissues of the face (i.e., forehead, nose, upper and lower lips, chin, and submental region) is not seen in patients with TCS. Dermal fat grafts, autogenous fat injections, and other forms of soft-tissue augmentation have gained popularity. The results of autogenous fat injection are variable and considered technique sensitive; they are generally not harmful, and they are clearly promising. Tanna and colleagues investigated the use of serial autologous fat grafting to restore soft-tissue contour in patients with hemifacial microsomia.[144] Patients with moderate to severe hemifacial microsomia were divided into two groups: Group I included those undergoing microvascular free-flap reconstruction (i.e., inframammary extended circumflex scapular flaps; n = 10); Group II included those undergoing multiple staged autogenous fat grafting (n = 21). The two patient groups had similar OMENS scores (2.4 and 2.3, respectively) and similar pre-reconstruction facial symmetry scores (74% and 75%, respectively). At the final evaluation, facial symmetry scores were 121% for the microvascular free-flap group and 99% for the fat-grafting group. Furthermore, no statistically significant difference between the microvascular and fat-graft groups in either patient or physician rating of overall satisfaction was noted. The extrapolation of these study results to the individual with TCS is appropriate.[52,73,144]

External Ear Reconstruction

The surgical reconstruction of the auricle can be satisfactorily achieved through a staged approach in the hands of a few experts.[12-15,19,40,74,75,86,111,130,140,145,146,155,162] The methods described by Brent represent the reference standard for external ear reconstruction.[12-15] The successful grafting of a well-sculpted cartilage framework is the foundation for sound auricular repair. According to Brent, most children have adequate rib cartilage for the repair by 6 years of age. At that time, the synchondrotic region of ribs 6 and 7 will provide an ample cartilage block to form the framework of the ear; this is taken from the side contralateral to the ear being constructed. With the use of the preoperatively

determined measurements and a prefabricated template, the position of the ear is determined, and a small preauricular incision is made. Unusable vestigial cartilage is excised. A thin-skinned pocket is developed, with the dissection carried beyond the marked auricular outline to recruit sufficient tension-free skin coverage. For patients with bilateral microtia, Brent prefers that the harvesting of cartilage grafts for each ear be separated by several months. Simultaneous bilateral auricular reconstruction would necessitate bilateral chest wounds with severe splinting and the potential for respiratory distress. Other stages of the auricular construction include lobule transposition; the detachment of the auricle with a skin graft; the management of the hairline; and tragus construction. Brent suggests that bilateral microtia repair be completed with five procedures that are carried out at 3-month intervals.

When completing microtia reconstruction in the patient with TCS, eventual external auditory canal and middle-ear surgery must be considered. Auricular construction should precede any middle-ear surgery because, after an attempt is made to open the canal, the chances of obtaining a satisfactory auricular repair are severely compromised as a result of the scarring of the skin envelope. When middle-ear surgery is an option, Brent suggests that the cartilaginous framework be sculpted with a wider concha to accommodate a future surgically created canal.

Other options for external ear reconstruction include the use of alloplastic and homologous frameworks. Alloplastic materials that are currently in use include silicone and Medpor (Porex Surgical, Inc., College Park, Ga). These foreign substances are more susceptible to infection, soft-tissue wound dehiscence, and susceptibility to minor trauma, even decades after reconstruction. Innovative techniques of tissue engineering involving bovine cartilage cells that are grown in the laboratory and seeded on a synthetic biodegradable ear template that is then implanted beneath the skin of an immunocompetent mouse have shown promise for the future. Unfortunately, until tissue engineering evolves beyond the currently encountered immunogenic problems, sculpted autogenous rib cartilage will remain the material of choice for the surgical repair of the ear.

At the same time, unless the option of sculpted autogenous rib cartilage is carried out by an experienced auricular reconstructive surgeon, the chance of achieving an aesthetically acceptable result is slim. For this reason, the use of a prosthetic external ear or no reconstruction at all in most individuals with TCS should not be discounted automatically.

External Auditory Canal and Middle-Ear Reconstruction

Although some of the hearing loss in the patient with TCS is attributable to external auditory canal stenosis or atresia, most of the loss (i.e., an average of 44 dB) is attributable to hypoplastic middle-ear cavities and ankylotic or missing ossicles.[18,58,66] Ankylosed or non-functioning ossicles appear to limit hearing conduction to the same extent as if they were absent.[55] Attempting reconstruction of the middle ear in patients with TCS can be expected to result in gains in hearing. However, even if the ossicles are mobilized, middle-ear dysmorphology remains; this results in residual hearing loss and leaves the patient dependent on some form of amplification. Jahrsdoerfer reported on a group of patients with TCS who underwent surgery for hearing rehabilitation.[66] He confirmed the generally unsuccessful attempts to satisfactorily improve "non-aided" long-term hearing. A form of bone-conduction hearing aid is required for proper amplification in most patients, including those who have undergone reconstruction as well as those who have not. The bone-conduction hearing aid may be a traditional unit, a modified amplification system, or a bone-anchored conduction unit.

According to Brent, for the gains from any middle-ear surgery to outweigh the risks and complications of the procedure, it should be reserved for selected patients with bilateral and unilateral microtia in whom personal motivation is high, in whom the favorable radiologic evidence of middle-ear development is present, and in whom a favorable outcome is expected according to an experienced otologist.[12-15] Furthermore, it must be planned through a team approach with an otologist and an auricular/reconstructive surgeon who are competent and experienced with ear-canal atresia, middle-ear malformations, and microtia repair. At the time of middle-ear surgery, the auricular/reconstructive surgeon initiates the procedure by lifting the reconstructed ear from its bed while carefully preserving both the connective tissue on the framework's undersurface and the overall circulation of the flap. The otologist proceeds by drilling a bony external auditory canal, completing ossiculoplasty, and then repairing the tympanum with a temporal fascia graft. The auricular/reconstructive surgeon then excises the soft tissues to exteriorize the meatus through the chondral region and harvests a skin graft that the otologist uses to line the new external auditory canal.

The Bone-Anchored Hearing Aid Option

A bone-anchored hearing aid (BAHA) consists of a permanent titanium fixture that is surgically implanted into the skull bone behind the ear and a small detachable sound processor that clips onto the fixture. BAHAs are suitable for people with conductive or mixed hearing loss who cannot adequately benefit from or prefer not to use conventional hearing aids.

Colquitt and colleagues completed a systematic review and an economic evaluation of BAHAs for people who are bilaterally deaf.[23] Previous prospective studies of adults or children with bilateral hearing loss were eligible for review. Twelve studies were included; there were seven cohort pre-post studies and five cross-sectional audiologic comparison studies. Comparisons were made between BAHAs and the

following: 1) conventional hearing aids, which are also known as air-conduction hearing aids (ACHAs); 2) bone-conduction hearing aids (BCHAs); and 3) unaided hearing and ear surgery. The study also looked at 4) unilateral versus bilateral BAHAs. Outcomes reviewed included hearing measures, validated measures of quality of life, adverse events, and measures of cost-effectiveness.

There appeared to be some audiologic benefits of BAHAs for this group of patients, including 1) improvements in speech as compared with BCHAs; and 2) understanding and noise as compared with ACHAs (however, ACHAs may produce better audiologic results for other outcomes). In addition, 3) hearing was found to be improved with the use of BAHAs as compared with unaided hearing; and 4) studies that compared unilateral with bilateral BAHAs suggested the benefits of bilateral BAHAs in many situations. However, prospective case series of BAHAs reported rates of 6.1% to 19.4% for the loss of the implants over time. In addition, the financial analysis suggests that BAHAs are unlikely to be a cost-effective option in terms of hearing gain and probability of needing alternative aids. Interestingly, the greater the benefit from aided hearing and the greater the difference in the proportion of people using the hearing aid for more than 8 hours per day, the more likely the BAHAs were a cost-effective option.

Janssen and colleagues completed a systematic review of bilateral BAHAs for bilateral permanent conductive hearing loss.[68] This was a review of the literature from between 1977 and 2011, and it included studies in which subjects of any age had permanent conductive hearing loss and bilateral implanted BAHAs. Eleven studies met the criteria for data analysis. Bilateral BAHAs were found to provide audiologic benefit as compared with unilateral BAHAs (i.e., improved thresholds for tones, speech in quiet and in noise, improved localization/lateralization, and patient's perceived subjective benefit). Disadvantages of bilateral BAHAs included 1) listening in noise in some condition; 2) presumed additional cost; and 3) presumed increasing adverse event risk. The authors concluded that bilateral BAHAs provided additional objective and subjective benefit as compared with unilateral BAHAs.

Conclusions

The current approach to the correction of malformations associated with TCS is to stage the reconstruction to coincide with facial growth patterns, visceral function (i.e., vision, breathing, chewing, swallowing, speech, hearing), and psychosocial needs. Precise morphologic analysis of each patient and the recognition of the need for a staged reconstructive approach serve to clarify the objectives at each interval for both the clinicians and the family. By continuing to define a rationale for the timing, methods, and extent of surgical, medical, and dental interventions and then objectively evaluating head and neck function, facial aesthetics, and psychosocial outcomes, the outlook for individuals affected by TCS will be greatly improved.

References

1. Acosta H, Stelnicki E, Boyd B, et al: Vertical mesenchymal distraction and bilateral free fibula transfer for severe Treacher Collins syndrome. *Plast Reconstr Surg* 113:1209, 2004.
2. Anderson P, Netherway D, Abbott A, et al: Mandibular lengthening by distraction for airway obstruction in Treacher-Collins syndrome: The long-term results. *J Craniofac Surg* 15:47, 2004.
3. Argenta LC, Iacobucci JJ: Treacher Collins syndrome: Present concepts of the disorder and their surgical correction. *World J Surg* 13:401, 1989.
4. Arvystas M, Shprintzen RJ: Craniofacial morphology in Treacher Collins syndrome. *Cleft Palate J* 28:226, 1991.
5. Arvytas AH, Netherway DJ, David DJ, Brown T: Application and comparison of techniques for three-dimensional analysis of craniofacial anomalies. *J Craniofac Surg* 1:119–134, 1990.
6. Arystas M, Shprintzen RJ: Craniofacial morphology in Treacher Collins syndrome. *Cleft Palate Craniofac J* 28:226, 1991.
7. Baas EM, Horsthuis RBG, Lange JD: Subjective alveolar nerve function after bilateral sagittal split osteotomy or distraction osteogenesis of mandible. *J Oral Maxillofac Surg* 70:910–918, 2012.
8. Beaune L, Forrest CR: Adolescents' perspectives on living and growing up with Treacher Collins syndrome: A qualitative study. *Cleft Palate Craniofac J* 41:343, 2004.
9. Behrents RG, McNamara JA, Avery JK: Prenatal mandibulofacial dysostosis (Treacher Collins syndrome). *Cleft Palate J* 14:13, 1977.
10. Berry GA: Note on a congenital defect (coloboma) of the lower lid. *R Lond Ophthalmic Hosp Rep* 12:255, 1889.
11. Bregeat P: Thirty-six years after a mandibulofacial dysostosis operation. *Ophthalmic Paediatr Genet* 6:313, 1985.
12. Brent B: Auricular repair using autogenous rib cartilage grafts: Two decades of experience with 600 cases. *Plast Reconstr Surg* 90:355, 1992.
13. Brent B: Advances in ear reconstruction with autogenous rib cartilage grafts: Personal experience with 1200 cases. *Plast Reconstr Surg* 104:319, 1999.
14. Brent B: The team approach to treating the microtia atresia patient. *Otolaryngol Clin North Am* 33:1353, 2000.
15. Brent B: Microtia repair with rib cartilage grafts: A review of personal experience with 1000 cases. *Clin Plast Surg* 29:257, 2002.
16. Brevi B, Lagana F, Piazza F, Sesenna E: Mandibular distraction osteogenesis with a small semiburied device in neonates: Report of 2 cases. *Ear Nose Throat J* 85:102, 2006.
17. Burstein FD, Cohen SR, Scott PH, et al: Surgical therapy for severe refractory sleep apnea in infants and children: Application of the airway zone concept. *Plast Reconstr Surg* 96:34, 1995.
18. Caldarelli DD, Hutchinson JG, Jr, Pruzansky S, et al: A comparison of microtia and temporal bone anomalies in hemifacial microsomia and mandibulofacial dysostosis. *Cleft Palate J* 17:103, 1980.
19. Cao Y, Vacanti JP, Paige KT, et al: Transplantation of chondrocytes utilizing a polymer-cell construct to produce tissue-engineered cartilage in the shape of a human ear. *Plast Reconstr Surg* 100:297, 1997.
20. Clauser L, Curioni C, Spanio S: The use of the temporalis muscle flap in facial and craniofacial reconstructive surgery: A review of 182 cases. *J Craniomaxillofac Surg* 23:203, 1995.

21. Codvilla A: On the means of lengthening, in the lower limbs, the muscles and tissues which are shortened through deformity. *Am J Orthop Surg* 2:353, 1905.
22. Collins ET: Cases with symmetrical congenital notches in the outer part of each lower lid and defective development of malar bones. *Trans Ophthalmol Soc UK* 20:190, 1900.
23 Colquitt JL, Jones J, Harris P, et al: Bone anchored hearing aids (BAHAs) for people who are bilaterally deaf: a systematic review and economic evaluation. *Health Technol Assess* 15(26):1–200, iii–iv, 2011.
24. Converse JM, Wood-Smith D, McCarthy JG, et al: Bilateral facial microsomia. *Plast Reconstr Surg* 54:413, 1974.
25. Cope JB, Samchukov ML, Cherkashin AM: Mandibular distraction osteogenesis: A historic perspective and future directions. *Am J Orthod Dentofacial Orthop* 115:448–460, 1999.
26. Da Silva Dalben G, Costa B, Gomide MR: Prevalence of dental anomalies, ectopic eruption and associated oral malformations in subjects with Treacher Collins syndrome. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 101:588, 2006.
27. Da Silva Dalben G, Teixeira das Neves L, Ribeiro Gomide M: Oral health status of children with Treacher Collins syndrome. *Spec Care Dentist* 26:71–75, 2006.
28. Dahl E, Kreiborg S, Bjork A: A morphologic description of a dry skull with mandibulofacial dysostosis. *Scand J Dent Res* 83:257, 1975.
29. Daniels S, Ellis E, III, Carlson DS: Histologic analysis of costochondral and sternoclavicular grafts in the TMJ of the juvenile monkey. *J Oral Maxillofac Surg* 45:675, 1987.
30. Dixon J, Edwards SJ, Anderson I, et al: Identification of the complete coding sequence and genomic organization of the Treacher Collins syndrome gene. *Genome Res* 7:223, 1997.
31. Ebata T, Nishiki S, Masuda A, et al: Anaesthesia for Treacher Collins syndrome using a laryngeal mask airway. *Can J Anaesth* 38:1043, 1991.
32. Edwards S, Gladwin A, Dixon, MJ: The mutational spectrum in Treacher Collins syndrome reveals a predominance of mutations that create a premature-termination codon. *Am J Hum Genet* 60:515, 1997.
33. Ellis E, III, Carlson DS: Histologic comparison of the costochondral, sternoclavicular, and temporomandibular joints during growth in *Macaca mulatta*. *J Oral Maxillofac Surg* 44:312, 1986.
34. Ellis PE, Dawson M, Dixon MJ: Mutation testing in Treacher Collins syndrome. *J Orthod* 29:293, 2002.
35. Farkas LG, Posnick JC: Detailed morphology of the nose in patients with Treacher Collins syndrome. *Ann Plast Surg* 22:211, 1989.
36. Farkas LG, Posnick JC, Winemaker MJ: Orbital protrusion in Treacher Collins syndrome: a tool for determining the degree of soft tissue damage. *Dtsch Z Mund Kiefer Gesichtschir* 13(6):429–432, 1989.
37. Fan K, Andrews BT, Liao E, et al: Protection of the temporomandibular joint during syndromic neonatal mandibular distraction using condylar unloading. *Plast Reconstr Surg* 129:1151–1161, 2012.
38. Fernandez AO, Ronis ML: The Treacher Collins syndrome patient. *Arch Otolaryngol* 80:505, 1964.
39. Figueroa AA, Peterson-Falzone SJ, Friede H, et al: Neurocranial morphology in mandibulofacial dysostosis (Treacher Collins syndrome). *Cleft Palate Craniofac J* 30:369, 1993.
40. Firmin F: Ear reconstruction in cases of typical microtia: Personal experience based on 352 microtic ear corrections. *Scand J Plast Reconstr Surg Hand Surg* 32:35, 1998.
41. Franceschetti A, Klein D: The mandibulofacial dysostosis: A new hereditary syndrome. *Acta Ophthalmol* 27:143, 1949.
42. Francis-West PH, Robson L: Craniofacial development: The tissue and molecular interactions that control development of the head. *Adv Anat Embryol Cell Biol* 169:1, 2003.
43. Freihofer HPM: Four-step mandibular lengthening to correct a bird-face deformity. *Acta Stomatol Belg* 87:189, 1990.
44. Freihofer HPM: Variations in the correction of Treacher Collins syndrome. *Plast Reconstr Surg* 99:647, 1997.
45. Freihofer HPM, Borstlap WA: Reconstruction of the zygomatic area. *J Craniomaxillofac Surg* 17:43, 1989.
46. Fryns JP, Bonhomme A, Van Den Berge H: Nager acrofacial dysostosis: An adult male with severe neurologic deficit. *Genet Couns* 7:147, 1996.
47. Fuente del Campo A, Martinez Elizondo M, Arnaud E: Treacher Collins syndrome (mandibulofacial dysostosis). *Clin Plast Surg* 21:613, 1994.
48. Garcia-Cimbrelo E, Olsen B, Ruiz-Yague M, et al: Ilizarov technique: Results and difficulties. *Clin Orthop* 283:116, 1992.
49. Garner LD: Cephalometric analysis of Berry-Treacher Collins syndrome. *Oral Surg* 23:320, 1967.
50. Giugliani R, Pereira CH: Nager acrofacial dysostosis with thumb duplication: Report of a case. *Clin Genet* 26:228, 1984.
51. Gorlin RJ, Cohen MM, Jr, Levin LS: *Syndromes of the head and neck*, ed 3, New York, 1990, Oxford University Press, pp 649–652.
52. Grahovac TL, Rubin JP: Discussion: An analysis of the experiences of 62 patients with moderate complications after full-face fat injection for augmentation. *Plast Reconstr Surg* 129:1369, 2012.
53. Heller JB, Gabbay JS, Kwan D, et al: Genioplasty distraction osteogenesis and hyoid advancement for correction of upper airway obstruction in patients with Treacher Collins and Nager syndromes. *Plast Reconstr Surg* 117:2389, 2006.
54. Hennig TB, Ellis E, III, Carlson DS: Growth of the mandible following replacement of the mandibular condyle with the sternal end of the clavicle: An experimental investigation in *Macaca mulatta*. *J Oral Maxillofac Surg* 50:1196, 1992.
55. Herberts G: Otological observations on the "Treacher Collins syndrome." *Acta Otolaryngol* 54:457, 1962.
56. Herman TE, Siegel MJ: Special imaging case-book: Nager syndrome. *J Perinatol* 18:85, 1998.
57. Herring SW, Rowlett UF, Pruzansky S: Anatomical abnormalities in mandibulofacial dysostosis. *Am J Med Genet* 3:225, 1979.
58. Holborow JC, Jr, Caldarelli DD, Valvassori GE, et al: The otologic manifestations of mandibulofacial dysostosis. *Trans Sect Otolaryngol Am Acad Ophthalmol Otolaryngol* 84:520, 1977.
59. Hopper RA, Altug AT, Grayson BH, et al: Cephalometric analysis of the consolidation phase following bilateral pediatric mandibular distraction. *Cleft Palate Craniofac J* 40:233, 2003.
60. Hurwitz DJ: Long-term results of vascularized cranial bone grafts. *J Craniofac Surg* 5:237, 1994.
61. Ilizarov GA: The principles of the Ilizarov method. *Bull Hosp Joint Dis Orthop Inst* 48:1, 1988.
62. Jabs EW, Li X, Lovett M, et al: Genetic and physical mapping of the Treacher Collins syndrome locus with respect to loci in the chromosome 5q3 region. *Genomics* 18:7, 1993.
63. Jabs EW, Xiang L, Coss CA, et al: Mapping the Treacher Collins syndrome locus to 5q31.3–q33.3. *Genomics* 11:193, 1991.
64. Jackson IT: Reconstruction of the lower eyelid defect in Treacher Collins syndrome. *Plast Reconstr Surg* 67:365, 1981.
65. Jackson IT, Bauer B, Salech J, et al: A significant feature of Nager syndrome: Palatal agenesis. *Plast Reconstr Surg* 84:219, 1989.
66. Jahrsdoerfer RA, Aquilar EA, Yeakley JW, et al: Treacher Collins syndrome: An otologic challenge. *Ann Otol Rhinol Laryngol* 98:807, 1989.
67. James D, Ma L: Mandibular reconstruction in children with obstructive sleep apnea due to micrognathia. *Plast Reconstr Surg* 100:1131, 1997.
68. Janssen RM, Hong P, Chadha NK: Bilateral bone-anchored hearing aids for bilateral permanent conductive hearing loss: a systematic review. *Otolaryngol Head Neck Surg* 147:412–422, 2012.
69. Kaban LB, Moses ML, Mulliken JB: Surgical correction of hemifacial microsomia in the growing child. *Plast Reconstr Surg* 82:9, 1980.
70. Kaban LB, Seldin EB, Kikinis R, et al: Clinical application of curvilinear distraction osteogenesis for correction of mandibular deformities. *J Oral Maxillofac Surg* 67:996–1008, 2009.

71. Karp NS, Thorne CHM, McCarthy JG, et al: Bone lengthening in the craniofacial skeleton. *Ann Plast Surg* 24:231, 1990.
72. Katsanis S, Jabs EW: Treacher Collins syndrome. In Pagon RA, Bird TD, Dolan CR, Stephens K, Adam MP, editors: *SourceGeneReviews™ [Internet]*. Seattle (WA), 1993-2004 Jul 20 [updated 2012 Aug 30], University of Washington, Seattle.
73. Kim SM, Kim YS, Hong JW, et al: An analysis of the experiences of 62 patients with moderate complications after full-face fat injection for augmentation. *Plast Reconstr Surg* 129:1359, 2012.
74. Kobus K, Szczyt M, Latkowski I, Wojcicki P: Reconstruction of the auricle. *Br J Plast Surg* 55:645, 2002.
75. Kobus K, Wojcicki P: Surgical treatment of Treacher Collins syndrome. *Ann Plast Surg* 56:549, 2006.
76. Kolar JC, Farkas LG, Munro IR: Surface morphology in Treacher Collins syndrome: An anthropometric study. *Cleft Palate J* 22:266, 1985.
77. Kovacs AL: Use of the Augustine stylet anticipating difficult tracheal intubation in Treacher Collins syndrome. *J Clin Anaesth* 4:409, 1992.
78. Kreiborg S: The skeletal anatomy of mandibulofacial dysostosis (Treacher Collins syndrome) [discussion]. *Plast Reconstr Surg* 78:469, 1986.
79. Kreiborg S, Dahl E: The cranial base and face in mandibulofacial dysostosis. *Am J Med Genet* 47:753, 1993.
80. Gui L, Zhang Z, Zang M, et al: Restoration of facial symmetry in hemifacial microsomia with mandibular outer cortex bone grafting combined with distraction osteogenesis. *Plast Reconstr Surg* 127:1997–2004, 2011.
81. Longacre JJ, deStefano GA, Holmstrand KE: The surgical management of the first and second branchial arch syndromes. *Plast Reconstr Surg* 31:507, 1963.
82. Longaker MT, Flynn A, Siebert JW: Microsurgical correction of bilateral facial contour deformities. *Plast Reconstr Surg* 98:951, 1996.
83. Longaker MT, Siebert J: Microsurgical correction of facial contour in congenital craniofacial malformations: The marriage of hard and soft tissue. *Plast Reconstr Surg* 98:942, 1996.
84. Maran AGD: The Treacher Collins syndrome patient. *J Laryngol* 78:135, 1964.
85. Marres HA, Cremers CW, Dixon MJ, et al: The Treacher Collins syndrome: A clinical, radiological, and genetic linkage study on two pedigrees. *Arch Otolaryngol Head Neck Surg* 121:509, 1995.
86. Marres HA, Cremers CW, Marres EH: Treacher Collins syndrome: Management of major and minor anomalies of the ear. *Rev Laryngol Otol Rhinol* 116:105, 1995.
87. Marsh JL, Celin SE, Vannier MW, et al: The skeletal anatomy of mandibulofacial dysostosis (Treacher Collins syndrome). *Plast Reconstr Surg* 78:460, 1986.
88. Marsh K, Dixon J, Dixon MJ: Mutations in the Treacher Collins syndrome gene lead to mislocalization of the nucleolar protein treacle. *Hum Mol Genet* 7:1795, 1998.
89. Marszalek B, Wojckicki P, Kazimierz K, et al: Clinical features, treatment and genetic background of Treacher Collins syndrome. *J Appl Genet* 43:223, 2002.
90. McCarthy JG, Schreiber J, Karp N, et al: Lengthening the human mandible by gradual distraction. *Plast Reconstr Surg* 89:1, 1992.
91. Molina F: A CT scan technique for quantitative volumetric assessment of the mandible after distraction osteogenesis [discussion]. *Plast Reconstr Surg* 99:1248, 1997.
92. Molina F, Ortiz-Monasterio F: Mandibular elongation and remodeling by distraction: A farewell to major osteotomies. *Plast Reconstr Surg* 96:825, 1995.
93. Moore MH, Guzman-Stein G, Proudman TW, et al: Mandibular lengthening by distraction for airway obstruction in Treacher Collins syndrome. *J Craniofac Surg* 5:22, 1994.
94. Moos KF: Mandibular reconstruction in children with obstructive sleep apnea due to micrognathia [discussion]. *Plast Reconstr Surg* 100:1138, 1997.
95. Mukhopadhyay P, Mukherjee P, Adhikary M: Problems in the anaesthetic management of Pierre Robin and Treacher Collins syndrome. *Indian Pediatr* 29:1120, 1992.
96. Nager FR, deRaynier JP: Das gehororgan bei den angeborenen kopf-missbildungen. *Pract Otorhinolaryngol* 10(Suppl 2):1, 1948.
97. Obwegeser HL: Zur korrektur der dysostosis otomandibularis. *Schweiz Monatsschr Zahnheilkd* 80:331, 1970.
98. Obwegeser HL: Correction of skeletal anomalies of oromandibular dysostosis. *J Maxillofac Surg* 2:73, 1974.
99. Obwegeser HL, Hadjiianghelou O: Two ways to correct bird face deformity. *Oral Surg* 64:507, 1987.
100. Opitz C, Ring P, Stoll C: Orthodontic and surgical treatment of patients with congenital unilateral and bilateral mandibulofacial dysostosis. *J Orofac Orthop* 65:150, 2004.
101. Perkins JA, Sie KCY, Milczuk H, et al: Airway management in children with craniofacial anomalies. *Cleft Palate Craniofac J* 34:135, 1997.
102. Peterson-Falzone S, Figueroa AA: Longitudinal changes in cranial base angulation in mandibulofacial dysostosis. *Cleft Palate J* 26:31, 1989.
103. Pope AW, Speltz ML: Research on psychosocial issues of children with craniofacial anomalies: Progress and challenges. *Cleft Palate Craniofac J* 34:371, 1997.
104. Pope AW, Ward J: Self-perceived facial appearance and psychosocial adjustment in preadolescents with craniofacial anomalies. *Cleft Palate Craniofac J* 34:396, 1997.
105. Posnick JC: Occlusal plane rotation: aesthetic enhancement in mandibular micrognathia [discussion]. *Plast Reconstr Surg* 91:1241, 1993.
106. Posnick JC: Treacher Collins syndrome: In Aston SJ, Beasley RW, Thorne CAM, editors: *Grabb and Smith plastic surgery, ed 5*. Philadelphia, 1997, Lippincott-Raven, pp 313–319.
107. Posnick JC: Treacher Collins syndrome: Perspectives in evaluation and treatment. *J Maxillofac Surg* 155(10):1120–1133, 1997.
108. Posnick JC: Discussion: Midface growth after costochondral graft reconstruction of the mandibular ramus in hemifacial microsomia. *J Oral Maxillofac Surg* 56:127–128, 1998.
109. Posnick JC: Treacher Collins syndrome: Evaluation and treatment. In Posnick JC, editor: *Craniofacial and maxillofacial surgery in children and young adults.* Philadelphia, 2000, WB Saunders Co, Vol I, pp 391–418.
110. Posnick JC, Al-Qattan MM, Moffat S, Armstrong D: Cranio-orbito-zygomatic measurements from standard CT scans in unoperated Treacher Collins syndrome patients: comparison with normal controls. *Cleft Palate Craniofac J* 32(1):20–24, 1995.
111. Posnick JC, Al-Qattan MM, Whitaker LA: Assessment of the preferred vertical position of the ear. *Plast Reconstr Surg* 91(7):1198–1203, 1993.
112. Posnick JC, Fantuzzo J, Orchin J: Deliberate operative rotation of the maxillo-mandibular complex to alter the A-point to B-point relationship for enhanced facial esthetics. *J Oral Maxillofac Surg* 64:1687–1695, 2006.
113. Posnick JC, Goldstein JA, Waitzman A: Surgical correction of the Treacher Collins malar deficiency: quantitative CT scan analysis of long-term results. *Plast Reconstr Surg* 92(1):12–22, 1993.
114. Posnick JC, Ruiz R: Treacher Collins syndrome: current evaluation, treatment and future directions. *Cleft Palate Craniofac J* 37(5):434, 2000.
115. Posnick JC, Ruiz R, Tiwana P: Treacher Collins syndrome: comprehensive evaluation and treatment. *Oral Maxillofac Surg Clin North Am* 16:503–523, 2004.
116. Poswillo DE: Otomandibular deformity: pathogenesis as a guide to reconstruction. *J Maxillofac Surg* 2:64, 1974.
117. Poswillo DE: The pathogenesis of the Treacher Collins syndrome (mandibulofacial dysostosis). *Br J Oral Surg* 13:1, 1975.
118. Pron G, Galloway C, Armstrong D, Posnick JC: Ear malformation and hearing loss in patients with Treacher Collins syndrome. *Cleft Palate Craniofac J* 30:97–103, 1993.
119. Pruzansky S: Not all dwarfed mandibles are alike. *Birth Defects* 1:120, 1969.
120. Rasch DK, Browder F, Barr M, et al: Anaesthesia for Treacher Collins and Pierre Robin syndromes: a report of three cases. *Can Anaesth Soc J* 33:364, 1986.
121. Raulo Y: Treacher Collins syndrome: analysis, principles of treatment. In Caronni EP, editor: *Craniofacial surgery*. Boston, 1985, Little, Brown.

122. Raulo Y, Tessier P: Mandibulofacial dysostosis. *Scand J Plast Reconstr Surg* 15:251, 1981.
123. Raustia A, Pernu H, Pyhtinen J, et al: Clinical and computed tomographic findings in costochondral grafts replacing the mandibular condyle. *J Oral Maxillofac Surg* 54:1393, 1996.
124. Roberts FG, Pruzansky S, Aduss H: An x-radiocephalometric study of mandibulofacial dysostosis in man. *Arch Oral Biol* 20:265, 1975.
125. Roddi R, Vaandrager J, van der Meulen J: Treacher Collins syndrome: early surgical treatment of orbitomalar malformations. *J Craniofac Surg* 6:211, 1995.
126. Rogers BO: Berry-Treacher Collins syndrome: a review of 200 cases. *Br J Plast Surg* 17:107, 1964.
127. Rosen HM: Occlusal plane rotation: aesthetic enhancement in mandibular micrognathia. *Plast Reconstr Surg* 91:1231, 1993.
128. Rotten D, Levaillant JM, Martinez H, et al: The fetal mandible: a 2D and 3D sonographic approach to the diagnosis of retrognathia and micrognathia. *Ultrasound Obstet Gynecol* 19:122, 2002.
129. Saadeh P, Reavey PL, Siebert JW: A soft-tissue approach to midfacial hypoplasia associated with Treacher Collins syndrome. *Ann Plast Surg* 56:522–525, 2006.
130. Saadeh PB, Brent B, Mehrara BJ, et al: Human cartilage engineering: chondrocyte extraction, proliferation, and characterization for construct development. *Ann Plast Surg* 42:509, 1999.
131. Sengezer M: Mandibular lengthening by gradual distraction [letter]. *Plast Reconstr Surg* 92:372, 1993.
132. Shprintzen RJ: Palatal and pharyngeal anomalies in craniofacial syndromes. *Birth Defects* 18:53–78, 1982.
133. Shprintzen RJ, Croft C, Berkman MD, Rakoff SJ: Pharyngeal hypoplasia in Treacher Collins syndrome. *Arch Otolaryngol* 105:127–131, 1979.
134. Sidman J, Sampson D, Templeton B: Distraction osteogenesis of the mandible for airway obstruction in children. *Laryngoscope* 111:1137, 2001.
135. Siebert J, Anson G, Longaker M: Microsurgical correction of facial asymmetry in 60 consecutive cases. *Plast Reconstr Surg* 97:354, 1996.
136. Silla Freitas R, Tolazzi ARD, Alonso N, Cruz GA: Evaluation of molar teeth and buds in patients submitted to mandibular distraction: Long-term results. *J Plast Reconstr Surg* 121:1335–1342, 2010.
137. Singh D, Bartlett S: Congenital mandibular hypoplasia: analysis and classification. *J Craniofac Surg* 16:291, 2005.
138. Snyder CC: Bilateral facial agenesis (Treacher Collins syndrome). *Am J Surg* 92:81, 1956.
139. Snyder CC, Levine GA, Swanson HM, et al: Mandibular lengthening by gradual distraction. *Plast Reconstr Surg* 51:506, 1973.
140. Somers T, De Cubber J, Govaerts P, Offeciers FE: Total auricular repair: Bone anchored prosthesis or plastic reconstruction? *Acta Otorhinolaryngol Belg* 52:317, 1998.
141. Stelnicki E, Boyd J, Nott R, et al: Early treatment of severe mandibular hypoplasia with distraction mesenchymogenesis and bilateral free fibula flaps. *J Craniofac Surg* 12:337, 2001.
142. Stelnicki E, Lin W, Lee C: Long-term outcome study of bilateral mandibular distraction: A comparison of Treacher Collins and Nager syndromes to other types of micrognathia. *Plast Reconstr Surg* 109:1819, 2002.
143. Stenstrom SJ, Sundmark ES: Contribution to the treatment of the eyelid deformities in dysostosis mandibulofacialis. *Plast Reconstr Surg* 38:567, 1966.
144. Tanna N, Wan DC, Kawamoto HK, Bradley JP: Craniofacial microsomia soft-tissue reconstruction comparison: Inframammary extended circumflex scapular flap versus serial fat grafting. *Plast Reconstr Surg* 127:802, 2011.
145. Tanzer RC: Total reconstruction of the external ear. *Plast Reconstr Surg* 23:1, 1959.
146. Tanzer RC: Total reconstruction of the auricle: the evolution of a plan of treatment. *Plast Reconstr Surg* 47:523, 1971.
147. Teber OA, Gillessen-Kaesbach G, Fischer S, et al: Genotyping in 46 patients with tentative diagnosis of Treacher Collins syndrome revealed unexpected phenotypic variation. *Eur J Hum Genet* 12:879–890, 2004.
148. Tessier P: Chirurgische behandlung der genetisch bedingten palpebralen und orbito-facialen missbildungen. *Klin Monatschr Augenheilkd* 50:82, 1968.
149. Tessier P: Autogenous bone grafts taken from the calvarium for facial and cranial application. *Clin Plast Surg* 9:531, 1982.
150. Tessier P: Facial contouring in the Treacher Collins-Franceschetti syndrome. In Brent B, editor: *The artistry of reconstructive surgery*. St. Louis, 1987, CV Mosby, p 343.
151. Tessier P: Complications associated with the harvesting of cranial bone grafts [discussion]. *Plast Reconstr Surg* 95:14, 1995.
152. Tessier P, Tulasne J: Treacher Collins syndrome. In Marchac D, editor: *Craniofacial surgery*. New York, 1985, Springer-Verlag.
153. Tessier P, Tulasne JF: Stability in the correction of hypertelorbitism and Treacher Collins syndromes. *Clin Plast Surg* 16:195, 1989.
154. The Treacher Collins Syndrome Collaborative Group: Positional cloning of a gene involved in the pathogenesis of Treacher Collins syndrome. *Nat Genet* 12:130, 1996.
155. Tollefson TT: Advances in the treatment of microtia. *Curr Opin Otolaryngol Head Neck Surg* 14:412, 2006.
156. Van der Meulen JCH, Hauben DJ, Vaandrager JM, et al: The use of a temporal osteoperiosteal flap for the reconstruction of malar hypoplasia in Treacher Collins syndrome. *Plast Reconstr Surg* 74:687, 1984.
157. Vento AR, LaBrie RA, Mulliken JB: The OMENS classification of hemifacial microsomia. *Cleft Palate Craniofac J* 28:68–76, 1991.
158. Waitzman AA, Posnick JC, Armstrong D, et al: Craniofacial skeletal measurements based on computed tomography: I. Accuracy and reproducibility. *Cleft Palate Craniofac J* 29:112, 1992.
159. Waitzman AA, Posnick JC, Armstrong D, et al: Craniofacial skeletal measurements based on computed tomography: II. Normal values and growth trends. *Cleft Palate Craniofac J* 29:118, 1992.
160. Walker JS, Dorian RS, Marsh NJ: Anaesthetic management of a child with Nager syndrome. *Anaesth Analg* 79:1025, 1994.
161. Walker MB, Trainor PA: Craniofacial malformations: Intrinsic vs extrinsic neural crest cell defects in Treacher Collins and 22q11 deletion syndromes. *Clin Genet* 69:471, 2006.
162. Walton RL, Beahm EK: Auricular reconstruction for microtia: Part II. Surgical techniques. *Plast Reconstr Surg* 110:234, 2002.
163. Wan D, Taub P, et al: Distraction osteogenesis of costocartilaginous rib grafts and treatment algorithm for severely hypoplastic mandibles. *Plast Reconstr Surg* 127:2005–2013, 2011.
164. Wijbenga JG, Verlinden CR, Jansma J, et al: Long lasting neurosensory disturbance following advancement of the retrognathic mandible: distraction osteogenesis versus bilateral sagittal split osteotomy. *Int J Oral Maxillofac Surg* 38:719, 2009.
165. Winokur ST, Shiang R: The Treacher Collins syndrome (TCOF1) gene product, treacle, is targeted to the nucleolus by signals in its C-terminus. *Hum Mol Genet* 7:1947, 1998.
166. Wittenborn W, Panchal J, Marsh J, et al: Neonatal distraction surgery for micrognathia reduces obstructive sleep apnea and the need for tracheotomy. *J Craniofac Surg* 15:623, 2004.
167. Wolfe SA, Vitenas P, Jr: Malar augmentation using autogenous materials. *Clin Plast Surg* 18:39, 1991.
168. Wolford L: Occlusal plane alteration in orthognathic surgery–Part I: Effects on function and esthetics. *Am J Orthod Dentofacial Orthop* 106(3):304–316, 1994.
169. Zanini S, Viterbo F, Parro F, et al: Bone graft of the zygoma in a patient with Treacher Collins syndrome. *J Craniofac Surg* 5:270, 1994.

28

Hemifacial Microsomia: Evaluation and Treatment

JEFFREY C. POSNICK, DMD, MD

- Inheritance Pattern
- Considerations during Infancy and into Early Childhood
- Dysmorphology Associated with Hemifacial Microsomia
- Facial Growth Potential with Hemifacial Microsomia
- Classification of Temporomandibular Joint–Mandibular Malformation
- Staging of Skeletal Reconstruction: Timing and Techniques
- Consideration of First-Stage Mandibular Reconstruction during the Mixed Dentition
- Facial Soft-Tissue Reconstruction
- External Ear Reconstruction
- External Auditory Canal and Middle-Ear Reconstruction
- Conclusions

Hemifacial microsomia (HFM) is a craniofacial malformation that results in varying degrees of hypoplasia of the structures within the first and second branchial archs.[34,35,38,53,62,65,66,81,84,174,227] Congenital hypoplasia is generally unilateral, although bilateral (asymmetric) involvement occurs in 5% to 15% of patients. Most cases of this condition are sporadic, but there are rare familial causes that exhibit autosomal dominant inheritance. The term *hemifacial microsomia* was first used by Gorlin and Pindborg during the early 1960s, although the first recorded cases may have been those of Canton in 1861 and Von Arlt in 1881.[62] Many terms have been used for this malformation, thus indicating the wide spectrum of anomalies observed and emphasized by authors from various disciplines. In addition to HFM, the malformation has been called *craniofacial microsomia, oculo–auriculo–vertebral dysplasia, first and second branchial arch syndrome, lateral facial dysplasia, unilateral oto–mandibular dysostosis, facio–auriculo–vertebral sequence,* and *oculo–auriculo–vertebral spectrum.*

Although there are no agreed-upon minimal diagnostic criteria, the facial phenotype is characteristic when enough manifestations are present. In most patients, microtia or other auricular or preauricular abnormalities (i.e., preauricular tags or sinus pits) may present the mildest manifestation. Involvement is not always limited to the facial structures. Cardiac, renal, additional skeletal (vertebral), central nervous system, and other more unusual anomalies may also occur. Goldenhar syndrome is considered a variant of HFM, and it is present in about 10% of HFM patients. In addition to maxillofacial malformation, it presents with epibulbar dermoid cysts and cervical spine involvement.

Inheritance Pattern

The occurrence of HFM has been estimated to range from 1 in 3500 to 1 in 26,550 live births. Cohen suggests that the birth prevalence is likely to be around 1 in 5600 live births.[62,26] Morrison and colleagues stated a prevalence rate of conditions on the oculo–auriculo–vertebral spectrum of 1 in 45,000 in Northern Ireland.[26]

Kelberman and colleagues performed a genome-wide search for linkage in two families with features of hemifacial microsomia.[105] In one of these families, the data were highly suggestive of linkage to a region of approximately 10.7 cM on chromosome 14q32, with a maximum multipoint LOD score of 3.00 between microsatellite markers D14S987 and D14S65. Linkage to this region was excluded in the second family, thereby suggesting genetic heterogeneity. With the use of an animal model.

Poswillo demonstrated that early vascular disruption with expanding hematoma formation in utero resulted in the destruction of differentiating tissues in the region of the ear and the jaw.[214-218] The degree of local destruction

appeared to be related to the severity of tissue damage caused by the hematoma. The constellation of anomalies seen in patients with HFM suggests an origin that occurs at about 30 to 45 days' gestation. Naora and colleagues described a transgenic mouse line that carried an autosomal dominant insertion mutation that resulted in facial anomalies that resemble HFM, including microtia and abnormal occlusion.[164]

First and second branchial arch malformations similar to HFM have been observed in infants who are born to women who have been exposed to thalidomide and primidone. The administration of isotretinoin (Accutane) may also injure neural crest cells and prevent their normal development, thereby resulting in hemifacial hypoplasia similar to that observed in patients with HFM. It has been documented that acute exposure to ethanol during gastrulation (i.e., the transformation of the blastula into the gastrula) may induce asymmetric facial development.

Discordance in monozygotic twins has been frequently reported. Concordance with variable expression has also been rarely documented in monozygotic twins. The rarity of the reports of the concordance of the malformation in twins supports the suggestion that the condition is sporadic in most families. Familial instances have been observed with variable expressivity. Overall, the empiric recurrence risk is on the order of 2% to 3%; however, 1% to 2% of cases suggest an autosomal dominant inheritance pattern.

Engiz and colleagues reviewed the clinical and laboratory findings of 31 individuals with Goldenhar syndrome (15 boys and 16 girls) between the ages of 1 day and 16 years.[47] The characteristic features were preauricular skin tags (90%), microtia (52%), hemifacial microsomia (77%), and epibulbar dermoids (39%). Vertebral anomalies were noted in 70%, cardiac malformations were found in 39%, genitourinary anomaly were noted in 23%, and various central nervous system malformations were found in 47%.

Several classification systems for HFM have focused on one or more fundamental anatomic features of this anomaly. In 1991, Vento and colleagues described the OMENS classification for HFM. This system substratifies each of five anatomic manifestations of HFM in accordance with their dysmorphic severity on a scale from 0 to 3. The five manifestations each constitute one letter of the *OMENS* acronym: *orbital asymmetry; mandibular hypoplasia; ear deformity; nerve dysfunction;* and *soft-tissue deficiency.* Scoring was done on the basis of conventional radiographs (e.g., posteroanterior, lateral, submental, panoramic), physical examinations, and photographs. Although the OMENS classification does allow for the objective cataloguing of a range of the abnormalities that constitute the spectrum of HFM, it falls short in terms of providing effective communication when determining the timing and technique that are best suited for the reconstruction of each component of the anomaly (i.e., maxillo-mandibular, orbito–zygomatic, soft tissues, external ear). It also is of limited value with regard to overall patient management.[276]

At the present time, HFM should be regarded as a nonspecific symptom complex that is etiologically and pathogenetically heterogeneous.* Extreme variability of expression is the characteristic finding.

Considerations during Infancy and into Early Childhood

At the time of birth, for a child with HFM, concerns will center on the adequacy of the airway; swallowing and feeding; hearing; vision; the presence of other associated malformations; and family unit psychosocial issues.[3,16,27,28,56,57,112,125,131,165,175,193,196,197,199,207,208]

The *airway* may be compromised as a result of 1) maxillary hypoplasia with choanal (nasal) obstruction 2) mandibular micrognathia with a retropositioned tongue obstructing the oropharyngeal/hypopharyngeal spaces 3) laryngomalacia/tracheomalacia or 4) neuromotor involvement.[3,16,27,28,57,195] Depending on the severity of these malformations, a spectrum of airway problems may be present and necessitate treatment that ranges from special infant positioning with an extended hospital stay to mandibular osteotomies with advancement; on rare occasions, immediate or delayed tracheostomy may be required.

When the airway is compromised, the newborn has difficulty *swallowing* enough fluids and taking in enough calories (i.e., maintaining hydration and positive nitrogen balance) while simultaneously breathing effectively (i.e., sufficient oxygenation). In this situation, gavage-assisted feedings or the placement of a gastrostomy tube may be necessary to ensure adequate nutrition without aspiration or respiratory distress.

When a *cleft* (of the lip, palate, or both) or *macrostomia* is present, the timing of correction of these anomalies is generally similar to that used for the patient with nonsyndromic cleft lip and palate (Fig. 28-1).

Soon after birth, examination by a *medical geneticist* and the procurement of a blood sample for chromosomal studies (i.e., genetic analysis) will help to discern the extent of birth defects and the risk of recurrence. Evaluation by the *pediatric otolaryngologist* in combination with the *speech and feeding specialist* will clarify the extent of breathing, swallowing, and feeding difficulties. Initial *audiologic testing* via auditory evoked brain responses is essential to clarify neurosensory hearing function. A *pediatric ophthalmologic* assessment is useful to assess extraocular muscle function, corneal exposure difficulties, visual potential, and adnexal region malformations.

When moderate to severe skeletal malformations are present, a *craniofacial computed tomography (CT) scan* from the top of the skull through the cervical spine with three-dimensional reformation will be useful to document the extent of craniofacial skeletal dysmorphology. A focused *petrous temporal bone CT scan* is also required to document external auditory canal anatomy as well as middle and inner

*References 9, 26, 36, 41, 42, 47, 50, 52, 64, 69, 75, 78, 83, 90, 104-106, 119, 164, 181, 198, 206, 214-218, 229-231, 247, 248, 255, 263, 264, 267, 276, 281, 282

• **Figure 28-1** A newborn with a mild form hemifacial microsomia, including macrostomia of the left oral commissure. She is shown before and 1 year after macrostomia repair. **A,** Facial views before macrostomia repair. **B,** Facial views after oral commissure reconstruction.

ear anatomy. Because of the potential increased risk of radiation exposure to infants, these imaging studies may be delayed until they are necessary for surgical treatment planning. Appropriate *craniofacial, maxillofacial, cleft lip and palate, and auricular reconstructive surgeons* should also be consulted as indicated.

Dysmorphology Associated with Hemifacial Microsomia

Facial Soft Tissues

From a clinical perspective, the soft-tissue deficiencies associated with HFM can be considered to affect four anatomic regions of the head and neck: 1) the external ear; 2) the eyelid–adnexal structures; 3) the preauricular–cheek–lip soft tissues; and 4) the temporal fossa (Figs. 28-2 and 28-3).[19,51,85,98,101,106,130,133,129,228,251] The soft tissues within each region that may be deficient and dysmorphic include the bulk of the cutaneous and subcutaneous tissue, the volume of fat, the muscles of mastication and facial expression, the cranial nerves, and the parotid and submandibular glands.

According to the research of Kane and colleagues, in patients with HFM, the extent of hypoplasia of specific muscles of mastication frequently predicts the extent of dysplasia of the osseous origin and insertion of those muscles.[98] If the temporalis muscle is hypoplastic, a deficiency of the coronoid process will be present. When the masseter muscle is hypoplastic, the gonial region of the mandible will also be deficient. When the lateral pterygoid muscle is deficient, the condylar head is deficient or absent.

Preauricular–cheek region soft-tissue thickness and skin surface area are generally related to the skeletal deficiencies. *Skin tags* are small vestigial rests of epithelial tissue that are generally found at the cleft between the first and second arches. Skin tags are frequently associated with small cartilaginous remnants that are found within the subcutaneous tissue. *Sinus tracts* may form inclusion cysts or result in

• **Figure 28-2** A 9-year-old boy who was born with left-sided hemifacial microsomia and a unilateral cleft lip and palate. He underwent lip and palate repair during early childhood at another institution. He is congenitally missing the left external ear and external auditory canal. The soft tissues of the eyelids and the cheek region are also deficient. The skeletal malformation involves the upper facial skeleton (i.e., the left zygomatic complex, the left orbit, and the left squamous temporal bone) and the lower facial skeleton (i.e., the maxilla and the mandible). He has a type III glenoid fossa mandibular malformation. **A,** Frontal facial and computed tomography scan views. **B,** Oblique facial and computed tomography scan views.

infection if they are obstructed. *Macrostomia* is a clefting or failure of fusion of the maxillary and mandibular processes and of the first branchial arch, respectively. This results in a cleft of the orbicularis oris muscle, the overlying skin, and the underlying mucosa directly through the oral commissure. *Anophthalmia* or *microphthalmia* may also occur. *Colobomata* of the iris or eyelids with the absence of the eyelashes is a frequent finding. *Ptosis of the upper eyelid* (i.e., as a result of levator palpebral muscle function) with narrowing of the vertical palpebral fissure is often seen. *Deficiencies of the lateral canthi* with a decreased horizontal fissure are common.

Epibulbar dermoid cysts are found in a significant percentage of patients; they appear as solid yellowish or pinkish-white ovoid masses that vary in size from that of a pinhead to 8 to 10 mm in diameter. The cysts occur most often at the inferotemporal quadrant of the limbus. The surface is usually smooth, and it frequently has fine hairs. These cysts can occur at any location on the globe or in the orbit, and they may be movable or fixed to the dermis. Unilateral epibulbar dermoid cysts are seen in a high percentage of patients, and multiple lesions may occur in each eye. Occasionally, vision is impaired by encroachment on the pupillary axis or by lipid infiltration of the cornea.

Abnormalities of the external ear are a consistent finding and range from *anotia* to a *mildly dysmorphic ear*. Farkas and James were unable to demonstrate a direct relationship between the degree of microtia and the extent of skeletal deformity.[51]

The External Auditory Canal, the Middle- and Inner-Ear Structures, and Audiologic Findings

A narrow external auditory canal is frequently found in patients with mild external ear deformity, whereas atretic canals are expected in more severe cases.[17,18,223,280] At times, a small external ear with normal middle-ear architecture is seen. Audiometry will delineate the nature of the hearing loss, which likely includes conductive and, less frequently,

• **Figure 28-3** A 14-year-old girl who was born with right-sided hemifacial microsomia, including microtia and an absent external auditory canal. There is soft-tissue deficiency on the right side of the face. The skeletal malformations involve the right zygomatic complex as well as the mandible and the maxilla. There is a Type IIB mandibular malformation. The patient had previously undergone right ear reconstruction and the placement of a bone-anchored hearing aid (BAHA) at another institution. **A,** Frontal facial and computed tomography scan views. **B,** Oblique facial and computed tomography scan views.

sensorineural loss in 15% of patients. Hypoplasia or agenesis of the ossicles may occur. In a comprehensive study, Caldarelli and colleagues used air and bone conduction audiometry and temporal bone tomography to evaluate 57 patients with hemifacial microsomia.[17,18] The authors were unable to correlate the degree of auricular (i.e., external ear) deformity with hearing function. Focused temporal bone CT scans offer the best documentation of the middle-ear structures. Assessment of the unaffected and presumed normal ear is an essential part of the evaluation.

Maxillomandibular Region

A variable degree of hypoplasia of the skeletal structures within the first and second branchial arches will occur. As a result, the anteroposterior, transverse, and vertical dimensions of the face are diminished on the affected side, with secondary deformities on the contralateral side. This is especially true in the maxillomandibular region (see Figs. 28-2 and 28-3).[134]

Cranio–Orbito–Zygomatic Region

A variable degree of hypoplasia of the zygomatic complex is a frequent finding. The consequences of zygomatic hypoplasia may be seen clinically as orbital dystopia; maxillary hypoplasia; deficiency of the glenoid fossa; and deficiency of the squamous aspect of the temporal bone. This is best visualized with a three-dimensional reconstruction of a craniofacial CT scan. As a result of the asymmetric nature of the deformity, identifying consistent reproducible landmarks is difficult in the upper face. This makes a quantitative assessment of the skeletal malformation difficult and imprecise.

Facial Growth Potential with Hemifacial Microsomia

The potential for longitudinal facial growth in a child who is born with HFM is an important factor when

considering the timing and techniques of reconstruction.[96,97,114,115,133,144-147,152-154,168,194,202-204,225,226,230-232] An additional pivotal consideration for the maxillofacial reconstruction of the individual with HFM is the integrity of the glenoid fossa–condyle–ascending ramus of the mandible (Fig. 28-4). Some authors speculate that a reduced mandibular growth potential secondarily leads to the observed maxillary deficiency on the ipsilateral side with progressive canting of the occlusal plane. Those who advocate mandibular surgery during the mixed dentition often do so with the belief that it will effectively correct the lower jaw malformation and that normal ongoing mandibular growth will continue with the prevention of secondary maxillary growth deformities in an otherwise normal upper jaw. There are objective clinical and radiographic studies in the literature that review the issue of the progression of deformity with HFM during growth.[87-89,194,225,226]

Rune and associates placed metallic implants in the jaws to study the facial growth of 11 patients with HFM.[225,226] They collected and studied serial roentgen stereophotogrammetry images from each patient. They found that, in five patients, the cant of the occlusal plane seemed to "slightly" increase; in the remaining six patients, the occlusal plane asymmetry either remained the same or improved with time. The authors also stated that the pattern of mandibular displacement showed no correlation with the severity of the presenting deformity. They concluded that the data "do not support the claim that the asymmetry of the jaws is invariably increased in time because of growth disparity between the affected and the unaffected sides."[225,226]

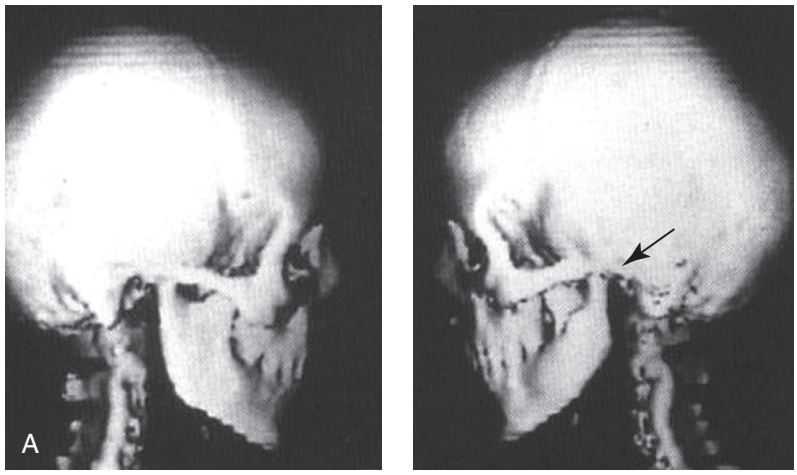
Polley and associates completed a longitudinal radiographic cephalometric study of the maxillofacial region in patients with HFM.[194] Twenty-six patients were included in the study and were followed longitudinally. Five patients (19%) had a Pruzansky grade I mandibular deformity, 14 patients (54%) had a Pruzansky grade II deformity, and 7 patient (27%) had a Pruzansky grade III deformity. The average age at the time that the initial cephalometric records for all patients were obtained was 3.5 years (range, 0.7 to 9.2 years), and the average age at the time that the final cephalometric records were obtained was 16.7 years (range, 10.1 to 22.5 years). None of the study patients had undergone surgical or orthopedic jaw manipulation during the time of the study, although nine patients underwent fixed appliance orthodontic treatment. Vertical and horizontal skeletal mandibular asymmetry was evaluated with posteroanterior cephalometry. The authors found that the growth of the affected side in patients with HFM parallels that of the contralateral side. The degree of mandibular asymmetry in the study patients remained relatively constant throughout craniofacial development. The mandibular skeletal deformity as measured in the study subjects did not progress with time. The growth rate of the affected side was similar to that of the contralateral side in each subject, irrespective of the degree of presenting mandibular deformity on the involved side. In the study group, subsequent growth occurred in both mandibular rami in each patient. This was true for all patients, irrespective of the Pruzansky grade and the side of the mandible that was affected.

Meazzini and colleagues conducted a long-term facial growth study of children affected by HFM with Type I or Type II mandibular malformations.[136A] The study group (N = 8) was not subjected to any treatment until adulthood. With the use of the panoramic radiograph analysis of ramus vertical heights, data confirmed that facial proportions in patients with HFM were maintained throughout growth when no treatment was undertaken. The authors concluded that HFM does not progress in the degree of facial asymmetry or deformity when left to mature naturally.

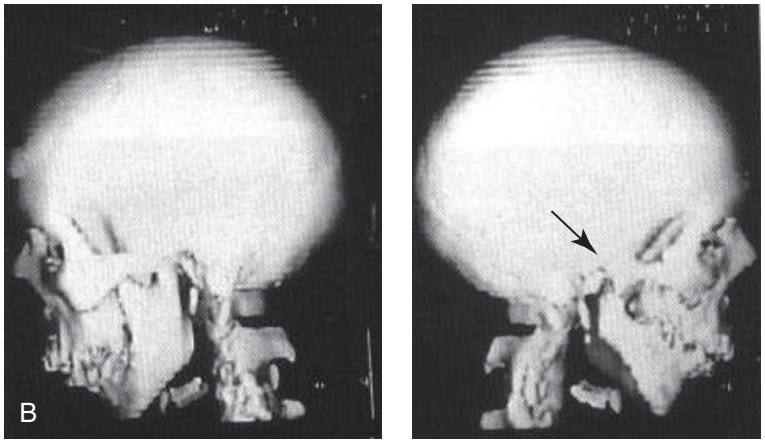
Ongkosuwito and colleagues studied mandibular growth in 84 consecutive patients with a confirmed diagnosis of unilateral HFM. The mandibular malformation for each subject was categorized into one of four grades on the basis of the Pruzansky/Kaban classification. The malformations were then regrouped to reflect those with a functional glenoid fossa and condyle (Type I and Type IIA) and those without (Type IIB and Type III). The study groups were compared with a normal age-matched Danish control group. For each subject, an orthopantomogram was obtained and used to perform measurements of ramal height (i.e., the distance measured in millimeters between the condylion and the gonion) on each side. The data confirmed that patients with HFM start with a shorter ramal height and end up with a shorter ramal height, although growth is the same in both patients with HFM (regardless of the extent of malformation) and controls. In patients with HFM, the ramal height is smaller on both sides, which means that growth is characterized not simply by unilateral underdevelopment but by a complex three-dimensional phenomenon.[161A]

According to the independent findings of Polley and colleagues, Rune and colleagues, Meazzini and colleagues, Ongkosuwito and colleagues, *the mandibular asymmetry associated with HFM is not progressive.* In these studies, progressive canting of the maxillary plane was not routinely observed (Figs. 28-5 through 28-7).

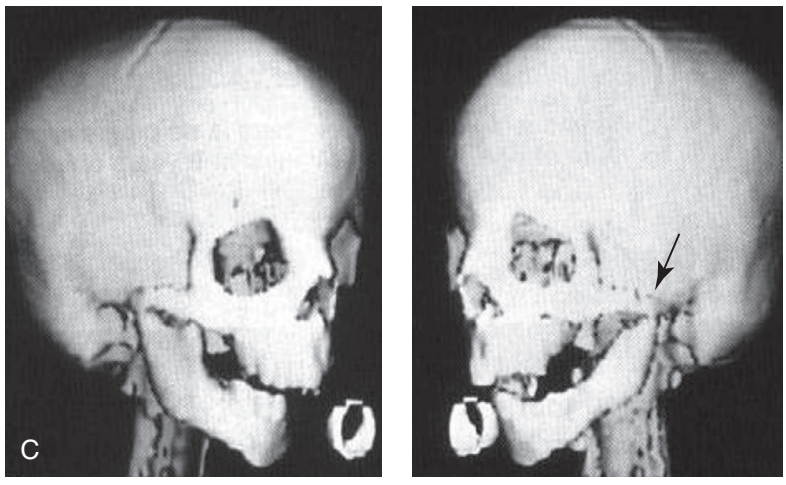
An alternative perspective is presented by **Kaban and colleagues.**[92,93,95] Their research suggests to them that, as a result of ongoing growth after birth in patients with HFM, there is progressive canting measured at the pyriform rims, the occlusal plane, and the intergonial angles, especially with the Type IIB and III mandibular malformations. The authors recommend that mandibular reconstruction during the mixed dentition be considered to correct the lower jaw deformity in anticipation of normal ongoing longitudinal growth; to prevent secondary maxillary deformities (in an otherwise normal maxilla); and to alleviate psychosocial issues that would otherwise occur during childhood. I believe that Kaban and colleagues' rationale for first-stage mandibular reconstruction during the mixed dentition in children with HFM are overly optimistic.



Kaban Type I



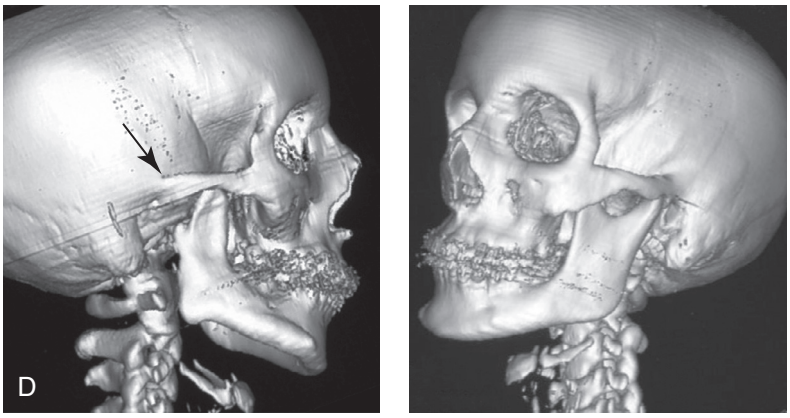
Kaban Type IIA



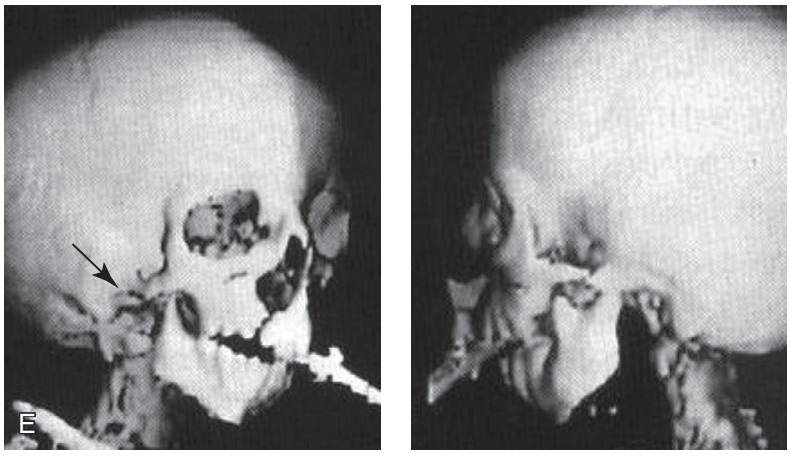
Kaban Type IIA

• **Figure 28-4** Kaban and colleagues described a classification system to define the degree of glenoid fossa–mandibular malformation observed in patients with hemifacial microsomia. Three-dimensional craniofacial computed tomography (CT) scans illustrate this classification system (see text for complete description). **A,** CT scans of a Type I mandibular malformation. **B** and **C,** CT scans of a Type IIA mandibular malformation.

Continued



Kaban Type IIB



Kaban Type III

• **Figure 28-4, cont'd D,** CT scans of a Type IIB mandibular malformation. **E,** CT scans of a Type III glenoid fossa–mandibular malformation in a patient who also has a unilateral cleft lip and palate.

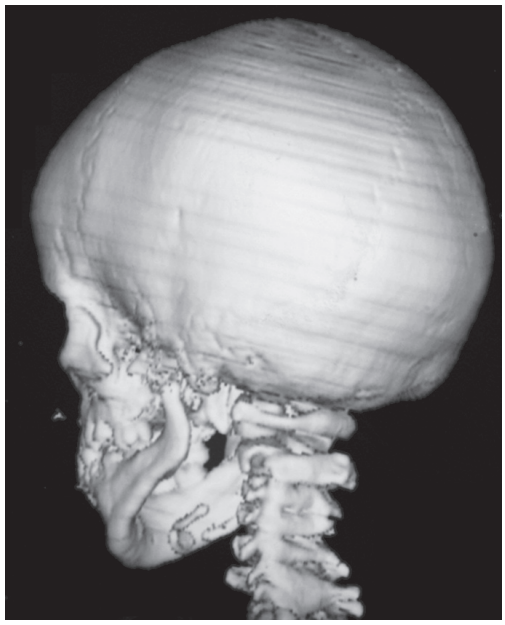
1. In perspective, even if a small degree of asymmetric jaw growth occurs during childhood in the patient with HFM, significant progressive dysfunction (i.e., in the areas of chewing, breathing, speech, lip control, or swallowing) that would justify a first-stage mandibular procedure is not generally seen.
2. The effectiveness of a first-stage mandibular procedure during the mixed dentition as a definitive long-term maxillofacial reconstruction has not been documented. Not only would a successful initial lengthening and derotation of the mandible be required, but the fabrication and then the periodic modification of an effective removable acrylic splint to create and maintain an ipsilateral posterior open bite (to allow for the supra-eruption of the maxillary posterior teeth) would be needed. The necessity of constant clinician monitoring and splint modification in conjunction with ongoing orthodontic treatment and rigorous patient compliance would be essential. This requires an experienced and dedicated orthodontist, a persistent surgical coordinator, and a zealous patient and family. All of the treating clinicians and the family must be in close geographic proximity and without socioeconomic barriers to treatment. Unfortunately, this is not a combination of events that is readily achievable.
3. Unfortunately, the costochondral graft construction of the neocondyle–ascending ramus required for the treatment of HFM Type IIB and III mandibular deformities carried out during the mixed dentition will result in a significant percentage of poorly growing mandibles (i.e., overgrowth or undergrowth). At minimum, a second definitive orthognathic reconstruction will be required (see the section to follow in this chapter about costochondral grafting).
4. Researchers have documented that, when a distraction osteogenesis (DO) approach is used as a first-stage mandibular procedure for HFM during the mixed dentition, symmetric lower jaw growth does not occur. The resulting residual mandibular asymmetry and deformity will, at minimum, require later

Text continued on p. 1110

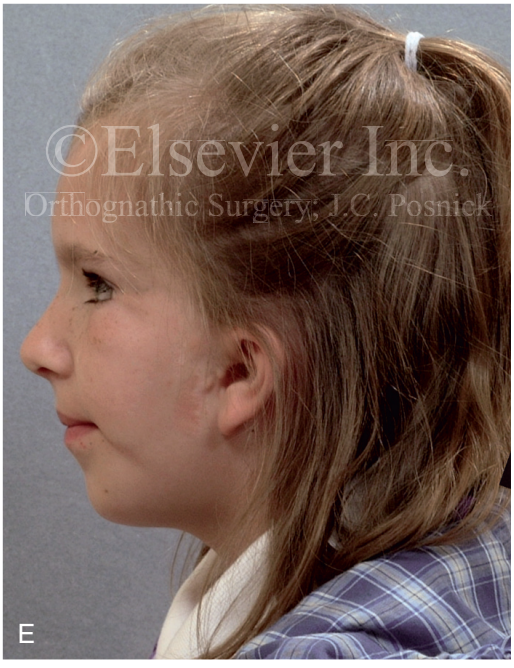
• **Figure 28-5** A girl who was born with left-sided hemifacial microsomia is followed up longitudinally at 5, 9, and 12 years of age without treatment intervention. There is a Type III glenoid fossa–mandibular malformation. No progression of the deformity has occurred. **A,** Frontal facial views at 5, 9, and 12 years of age. **B,** Oblique facial views at 5, 9, and 12 years of age. **C,** Profile views at 5, 9, and 12 years of age.

Continued

Age 9



Kaban III



Age 9

Kaban III

• **Figure 28-5, cont'd** **D** and **E,** Facial and computed tomography scan views at 9 years of age confirmed the extent of skeletal malformations involving the zygomatic–orbital complex, the maxilla, and the mandible.

Age 5

Age 12

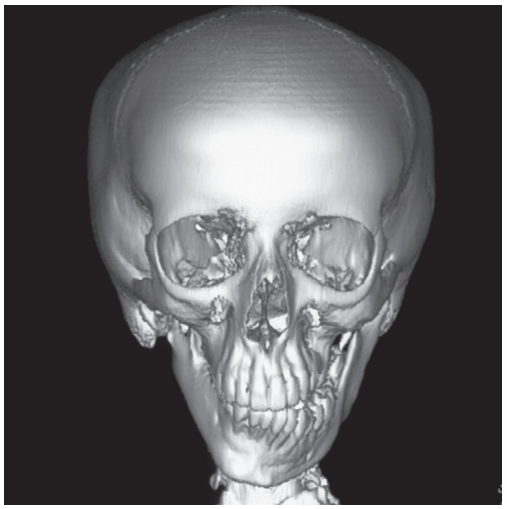
Age 5

Age 12

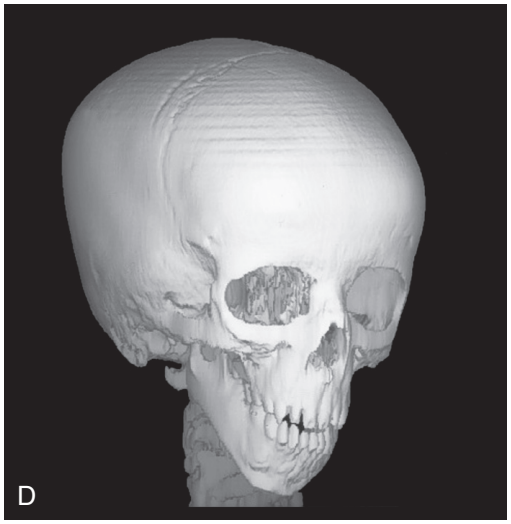
• **Figure 28-6** A girl who was born with right-sided hemifacial microsomia is followed up longitudinally at 5 and 12 years of age without treatment intervention. There is a Type IIB–mandibular malformation. No progression of the deformity is demonstrated. **A,** Frontal facial views in repose at 5 and 12 years of age. **B,** Profile views at 5 and 12 years of age.

Continued

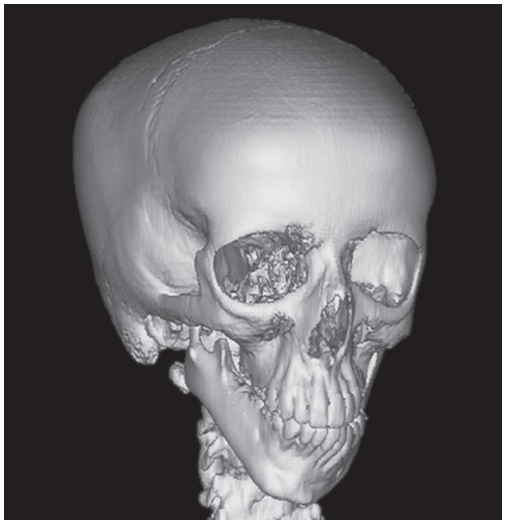
Age 5



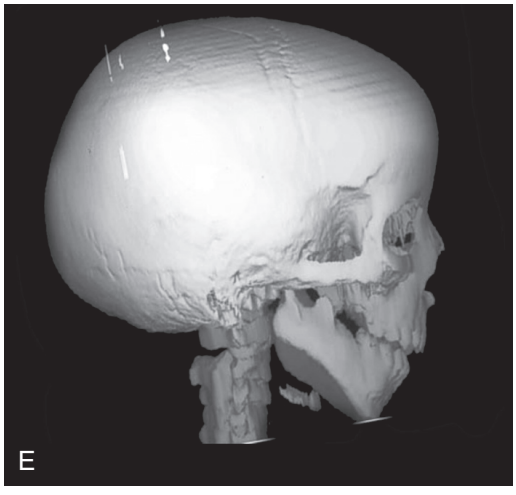
Age 12



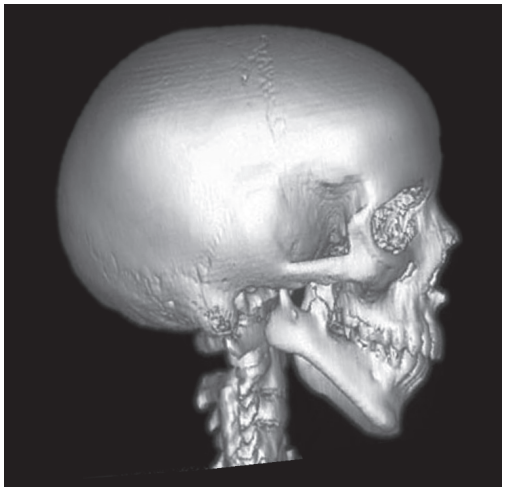
Age 5



Age 12



Age 5



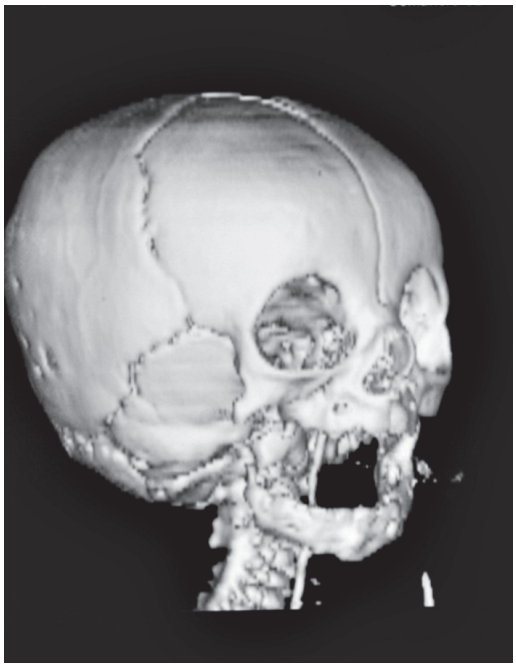
Age 12

• **Figure 28-6, cont'd C, D,** and **E,** Computed tomography scan views at 5 and 12 years of age that confirm no progression of the skeletal malformation.

6 months

Kaban III

6 months

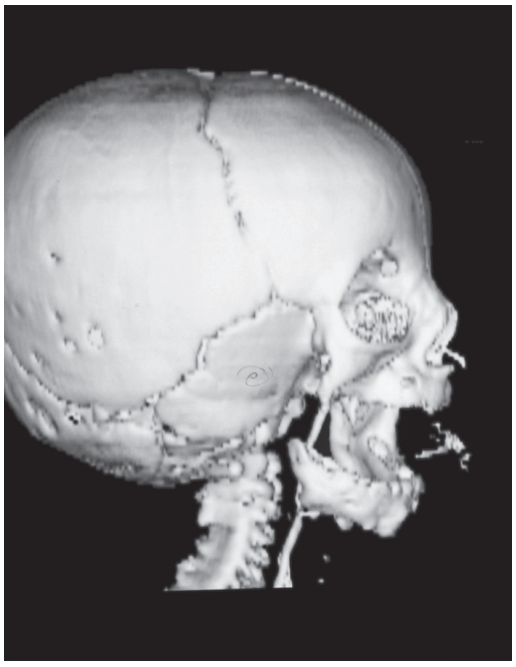


Kaban III

• **Figure 28-7** A child who was born with hemifacial microsomia of the right side. The extent of the soft-tissue and skeletal deficiencies of the structures within the first and second branchial arches is shown. There is a Type III glenoid fossa–mandibular malformation. The only treatment intervention was the excision of displaced redundant auricular tissue in the right cheek region. No progression of the skeletal malformation occurred between 6 months and 10 years of age. **A,** Frontal facial and computed tomography (CT) scan views at 6 months of age. **B,** Oblique facial and CT scan views at 6 months of age.

Continued

6 months



Kaban III

6 months

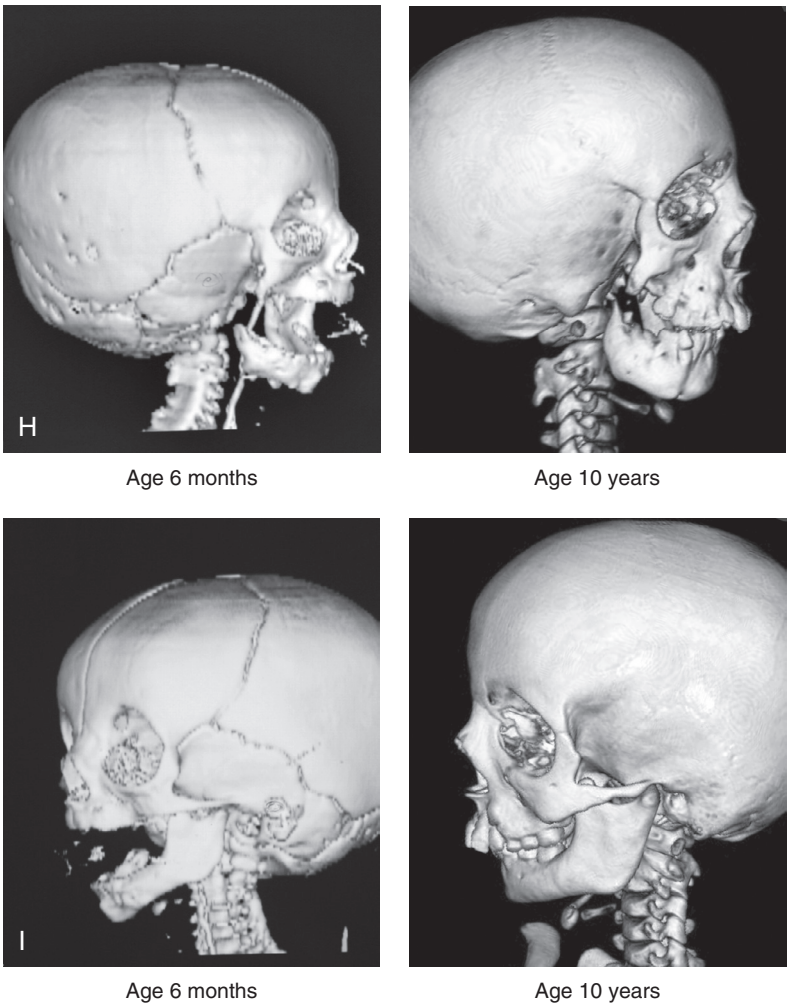
5 years

• **Figure 28-7, cont'd C,** Right profile and CT scan views at 6 months of age. **D,** Profile views at 6 months and 5 years of age. The excision of right cheek redundant soft tissue was carried out at 1 year of age with facial nerve preservation.

©Elsevier Inc.
Orthognathic Surgery; J.C. Posnick

• **Figure 28-7, cont'd** **E,** Frontal views at 4 years, 7 years, and 10 years of age without treatment intervention. **F,** Oblique facial views at 4 years, 7 years, and 10 years of age without treatment intervention. **G,** Profile views at 4 years, 7 years, and 10 years of age without treatment intervention.

Continued



• **Figure 28-7, cont'd H** and **I,** Left and right CT scan profile views at 6 months and 10 years of age.

NOTE: In this author's view, the benefits of mixed-dentition first-stage mandibular procedures rarely outweigh the downsides. The argument that first-stage mandibular surgery during the mixed dentition is necessary to allow the child to develop normal self-esteem is well intended but not realistic. Multiple doctor visits and procedures give a child a "sick kid" mentality and draw more attention to his or her differences.[199] The unfortunate reality is that, for a majority of HFM patients even with well-done mixed dentition first stage mandibular reconstruction, the residual external ear, soft tissue, and other skeletal malformations will continue to draw attention to the child's facial disproportion. The overall burden of care for the patient, the family, and the health care system is minimized when definitive single-stage maxillomandibular reconstruction is carried out at the time of early skeletal maturity (i.e., between the ages of 13 and 15) in conjunction with orthodontic treatment.

definitive orthognathic reconstruction. In addition, the completion of a mandibular ramus–body osteotomy and DO during the mixed dentition is documented to result in the destruction of ipsilateral developing permanent molars in a significant percentage of children who have undergone such an operation.

Classification of Temporomandibular Joint–Mandibular Malformation

The extent of temporomandibular joint (TMJ)–mandibular deficiency is an important factor when considering the timing of and techniques for reconstruction.[108,118,119,249] Imprecise definitions of the degrees of TMJ–mandibular anomalies have resulted in miscommunication among clinicians and in the literature. In an article published during the late 1960s, Pruzansky classified the presenting

mandibular anomalies of HFM into three grades (Type I through Type III) in accordance with the extent of hypoplasia of the mandibular condyle and ramus.[220] Kaban and colleagues refined the Pruzansky classification to further clarify the degree of glenoid fossa–condyle–ascending ramus malformation observed in patients with HFM.[94,157] The Kaban classification provides an excellent starting point for defining the reconstruction required in each individual patient (see Fig. 28-4).

A **Type I** mandible has only a minimal degree of hypoplasia of the glenoid fossa, the condyle, and the ascending ramus (see Fig. 28-4, *A*). All of the skeletal components are present, all of the masticatory muscles are present, and function is within normal limits. There is a degree of asymmetric mandibular retrognathia, with a shift of the chin off of the facial midline. A limited degree of maxillary cant can usually be documented. Note: With Type I malformations, the condyle and the glenoid fossa are fully intact. There is no need to construct a new condyle or a new glenoid fossa.

A **Type IIA** mandible has a moderate degree of glenoid fossa and condyle–ascending ramus hypoplasia (see Fig. 28-4, *B* and *C*). By definition, the extent of hypoplasia results in the TMJ complex being located anteriorly and medially as compared with the normal side; however, joint function remains satisfactory. The masticatory muscles have a variable degree of hypoplasia. The mandible is retrognathic, the chin is shifted to the ipsilateral side, and an anterior open bite is frequently seen. A moderate maxillary cant is usually present. With a Type IIA malformation, despite significant hypoplasia, a decision to retain the TMJ complex (i.e., the condyle and the glenoid fossa) as part of the maxillomandibular reconstruction has been made.

The **Type IIB** mandible involves severe hypoplasia of the condyle–ascending ramus (see Fig. 28-4, *D*). The glenoid fossa has an anterior and medial location, but its placement is adequate for the construction of a neocondyle. The hypoplastic condyle, if it is present at all, is rudimentary and not substantial enough to seat into the glenoid fossa. There is not a consistent "posterior stop" to the mandible against the glenoid fossa. The mandible functions with rotation but not with a condylar component in the glenoid fossa, and there is little or no translational jaw movement. Vertical mouth opening is present (albeit reduced), with a shift of the chin to the ipsilateral side. The extent of mandibular dysmorphology, asymmetric retrognathia, and anterior open bite is marked. Variable but definite deficiencies of the masticatory muscles are present. There is usually significant hypoplasia of the ipsilateral maxilla, with obvious canting. By definition, with a Type IIB mandibular deficiency and deformity, the condylar remnant is too far displaced and rudimentary to be useful. Construction of a *neocondyle–ascending ramus* into the current acceptable glenoid fossa is required.

The **Type III** mandible is free-floating on the ipsilateral side, with no "posterior stop" of the lower jaw against the skull base on the affected side (see Fig. 28-4, *E*). The condyle and all or part of the ramus on the ipsilateral side are absent. The disc, the TMJ capsule, and the glenoid fossa are also not developed. Mandibular dysmorphology is severe and includes ipsilateral retrognathia and decreased posterior (ramus) facial vertical height. The masticatory muscles are severely hypoplastic, and the lateral pterygoid remnant is not attached to the mandibular structures. There is significant hypoplasia of the maxilla, with obvious canting. Occasionally, a tracheostomy is required shortly after birth to manage the airway compromise that results from a combination of factors, including (but generally not limited to) the degree of mandibular hypoplasia. By definition, with a Type III glenoid fossa–mandibular malformation, the eventual construction of both a *neo–glenoid fossa* and a *neocondyle ascending ramus* will be required to achieve successful maxillofacial reconstruction.

Staging of Skeletal Reconstruction: Timing and Techniques

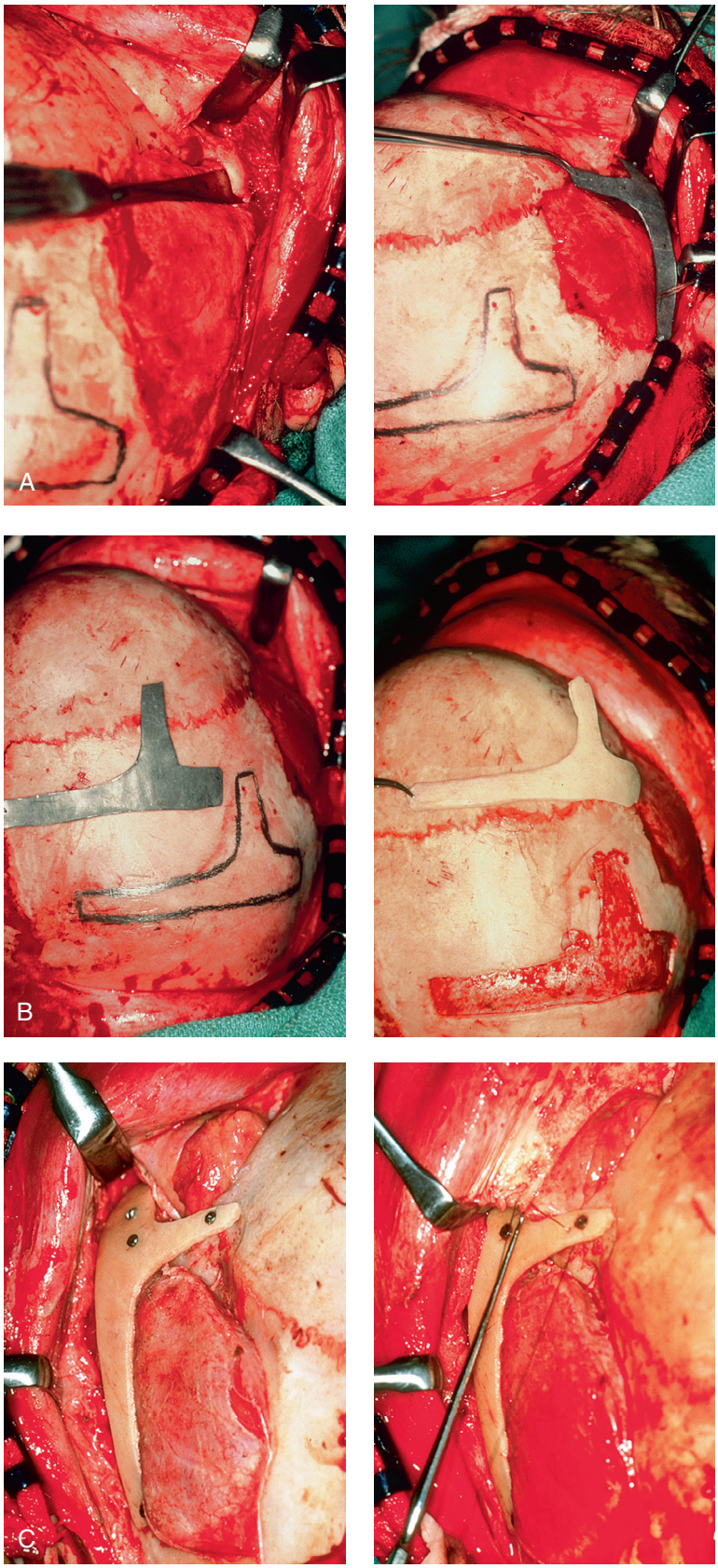
The facial rehabilitation of the patient with HFM must address unique and specific components of the malformation that may include the following: 1) the zygomatic and orbital region; 2) the maxillomandibular regions; 3) the facial soft tissues; 4) the external ear; 5) the auditory canal; and 6) the middle-ear structures.*

Some clinicians have speculated that there is a reduced facial growth potential in patients with HFM which then leads to a progression of the primary malformation and secondary deformities of the maxillofacial skeleton. This assumption has led some to advocate early mandibular reconstruction to prevent progression. This author agrees with Rune and colleagues,[225,226] Polley and colleagues,[194] Meazzini et al.[136A] and Ongkosuwito and colleagues[161A] that the observed facial asymmetry in patients with HFM is not progressive with ongoing growth (see the section earlier in this chapter about facial growth potential with HFM) (Figs. 28-5 through 28-7).[203,209-212,233,234]

Zygomatic and Orbital Reconstruction

The benefits of thoughtfully timed and meticulously executed orbito–zygomatic reconstruction for the patient with HFM are often underappreciated.[209-213] When upper face (i.e., orbito–zygomatic) reconstruction is needed, it is carried out through a coronal scalp incision. Correction of the upper face skeletal deformity may require the intracranial disassembly and reconstruction of the dysmorphic ipsilateral anterior cranial vault; the squamous portion of the temporal bone; and the lateral orbit regions with complete construction of the zygomatic complex, including the glenoid fossa. The use of full- and split-thickness cranial bone grafts is required (Fig. 28-8).

*References 70, 76, 77, 92-95, 99, 109, 135, 137, 142, 145, 151, 157, 166-168, 170, 172, 236, 243, 253, 256, 266, 270-275, 277, 287



• **Figure 28-8** Intraoperative views of a 7-year-old child demonstrate the technique of zygomatic–orbital reconstruction as described in this chapter. When working through a coronal scalp incision, the zygomatic and orbital structures are exposed. A full-thickness cranial bone graft is harvested, and a zygomatic complex is crafted from this. The crafted zygoma is inset and stabilized with microplates and screws. The orbital defects are reconstructed as indicated with cranial bone graft, and the orbital rim is reshaped with a rotary drill. A lateral canthopexy is completed through the coronal incision and fixed to the new frontozygomatic suture region. **A,** View of the dissected rudimentary zygomatic remnant. A template of the proposed zygomatic complex had been made. **B,** The template is taken to the temporoparietal region of the skull, and its outline is marked out for craniotomy. Through a craniotomy, a full-thickness cranial bone graft is harvested for zygomatic complex reconstruction. **C,** The full-thickness cranial bone graft is fixed in place with microscrews. The lateral canthus is identified through the coronal incision, and a 28-gauge stainless steel wire is passed through it. The lateral canthus is pexied to the new frontozygomatic region. *From Posnick JC, Goldstein JA, Waitzman AA: Surgical correction of the Treacher-Collins malar deficiency: quantitative CT scan analysis of long-term results,* Plast Reconstr Surg *92:12-22, 1993.*

This author believes that the reconstruction of the cranial vault, malar, and orbital deficiencies before the age of 7 years is not indicated unless functional disability warrants it.[213] After the patient reaches this age, cranio–orbitozygomatic skeletal development is nearly mature. When indicated, an adult-sized anterior cranial vault, orbit, and zygomatic complex may be constructed and matched with the contralateral normal side with little concern about how future growth will alter the initial results achieved. Donor site skull reconstruction is also simpler after the age of 7 years than it is in younger children, because the presence of bicortical cranial bone will facilitate the splitting of the inner and outer tables for efficient reconstruction. Artificial bone material may also be used to reconstruct the graft donor site of the posterior skull (see Chapter 27).

Other surgeons have recommended variations in the methods and timing of the reconstruction of the upper face skeletal anomalies associated with HFM.[31] Posnick and colleagues confirmed that the construction of the complete zygomatic complex with full-thickness autogenous cranial bone maintains volume and shape better than onlay grafts, which have been universally disappointing. Over time, onlay autogenous bone grafts that have been placed in the craniofacial region (e.g., supraorbital ridge, zygoma, anterior maxilla, angle of mandible, chin) from all tried donor sites (e.g., skull, hip, rib) have demonstrated significant and unpredictable resorption. Furthermore, no commensurate growth (i.e., volume expansion) of the graft with the underlying bone (e.g., the zygoma) should be expected. Some feel that, even if the onlay graft partially resorbs, at least something has been gained; however, this philosophy should be abandoned, because surface irregularities result in secondary deformities that further detract from the baseline malformation.

Maxillomandibular Reconstruction

An essential consideration when choosing the timing and techniques for maxillomandibular reconstruction in a patient with HFM is an understanding of the presenting TMJ–mandibular anatomy.[2,6,7,20,25,30-32,43,46,59,96,97,180,209-212,226] The classification of the TMJ–mandibular malformation, which was described earlier in this chapter, is clinically relevant and facilitates communication.[94,157] In the patient with HFM with either a Type I or Type IIA malformation, the basic maxillomandibular skeletal asymmetry and dysmorphology that requires reconstruction include the following: 1) degrees of altered facial height (i.e., decreased posterior facial height on the ipsilateral side); 2) diminished horizontal projection (i.e., deficiency more prominent on the ipsilateral side); and 3) decreased facial width (i.e. deficiency on the ipsilateral side). These malformations frequently result in canting (i.e., roll orientation) of the pyriform apertures, the maxilla, and the gonial angles; the shifting of the maxillary, mandibular, and chin midlines off of the facial midline (i.e. yaw orientation); clockwise rotation of the occlusal plane (i.e., pitch orientation); and an asymmetric class II malocclusion often with anterior open-bite malocclusion. According to the classification described, a patient who presents with a Type IIB malformation requires the *construction of a neocondyle*. For the individual with a Type III malformation in addition to this, there is a need for the *construction of a neo–glenoid fossa*.

The most favorable long-term function (i.e., speech, swallowing, chewing, breathing) and enhanced facial aesthetics are generally achieved in patients who undergo definitive reconstructive surgery of the maxilla, mandible, and chin regions at any time after jaw maturity (i.e., at 13 to 15 years of age for the HFM patient); this should be carried out in combination with effective orthodontic treatment after the permanent teeth have erupted and before the patient graduates from high school. As with other dentofacial deformities, extractions may be required to orthodontically relieve dental root crowding and to normalize the inclination of the anterior teeth in preparation for the surgical repositioning of the jaws.

Type I and IIA mandibular malformations are best reconstructed after all of the permanent teeth have erupted and orthodontic goals have been reached (see Fig. 28-4, *A, B,* and *C*). Surgical objectives can be met by making use of sagittal split ramus osteotomies of the mandible in combination with Le Fort I osteotomy (often in segments) and osseous genioplasty. This combination represents standard techniques, and it does not require bone grafts. The success of reconstruction is dependent on approximating mirror-image symmetry and Euclidian proportions of the skeleton through orthognathic procedures.

For **Type IIB** mandibular malformations, costochondral graft reconstruction of the deficient condyle ascending ramus at the time of skeletal maturity remains this surgeon's preferred approach in most cases (see Fig. 28-4, *D*), despite its limitations (see the section to follow concerning condylar reconstruction with the use of costochondral graft). A sagittal split ramus osteotomy is completed on the contralateral side to derotate the distal mandible. This is combined with a Le Fort I osteotomy (often in segments) and an osseous genioplasty.

A ramus osteotomy of the contralateral side of the mandible is completed first to control repositioning of the distal mandible. The distal mandible is then secured to the maxilla via intermaxillary fixation through a custom designed acrylic splint to create the preferred reorientation of the lower jaw (see Chapter 14). It may be necessary to resect the ipsilateral coronoid process before repositioning the distal mandible. The contralateral ramus osteotomy is then rigidly fixed with bicortical screws. The ipsilateral proximal mandible is reconstructed with the autogenous costochondral graft. Harvesting the rib graft from the contralateral chest wall provides the best contour for mandibular reconstruction. The fixation of the rib graft to the native distal mandible is accomplished with a titanium miniplate and 2.0-mm or 2.3-mm screws. The fixation plate extends from the graft forward along the

inferior border of the body of the mandible (fig. 28-15). It is generally necessary to recontour (i.e., with a bur on a rotary drill) the outer cortex of the distal mandible before onlaying the graft. Graft placement and fixation is often carried out through an extraoral Risdon neck incision. The avoidance of intraoral incisions on the ipsilateral mandibular ramus region during this procedure may minimize the incidence of infection. The effective seating of the neocondyle in the glenoid fossa is a critical step for successful reconstruction. In some patients, the extent of associated soft-tissue and skeletal deficiency will favor use of a vascularized fibula composite flap over the use of a costochondral graft.

The **Type III** glenoid fossa–mandibular malformation requires the surgical construction of the congenitally missing parts (see Fig. 28-4, *E*). The mandibular reconstruction is generally carried out as previously described for the Type IIB deformity. When the glenoid fossa requires construction (i.e., with a Type III malformation), so will the zygomatic complex. The glenoid fossa–zygoma and orbital reconstruction are best carried out as a separate operation when the patient is at least 7 years old (see the previous section about orbito–zygomatic reconstruction) and before mandibular reconstruction. With regard to the mandibular reconstruction, the deficiency of both the condyle-ascending ramus and the associated soft tissues may benefit from a vascularized fibula composite flap rather than a costochondral graft.

Consideration of First-Stage Mandibular Reconstruction during the Mixed Dentition

Personal Perspective Concerning First-Stage Mandibular Reconstruction during Childhood

The maxillofacial malformations in patients with HFM may be severe enough that, for airway or psychosocial reasons, the clinician feels compelled to recommend first-stage mandibular reconstruction during the mixed detention (i.e., between the ages of 7 and 11 years). Reported options for first-stage mandibular reconstruction in patients with HFM generally include 1) a ramus/body osteotomy followed by DO carried out over time for Type I and Type IIA malformations or 2) costochondral grafting with the immediate construction of a neocondyle ascending ramus for Type IIB and Type III malformations. In either case, derotation of the mandible to position the chin in the facial midline with the creation of an ipsilateral posterior open bite is usually considered the objective. The reconstructive option selected (i.e., costochondral graft versus DO) should be based on the presenting malformation and then on the following: 1) the three-dimensional morphologic results believed to be achievable; 2) the anticipated ongoing growth potential after surgery; 3) the differences in perioperative morbidity among the techniques; and 4) the overall burden of treatment to the patient, the family, the clinicians, and the health care system.[209-212]

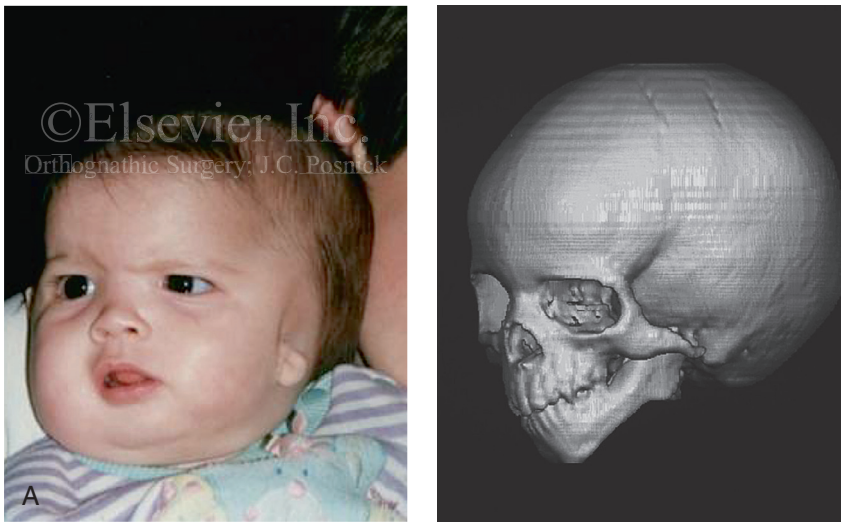
Interestingly, most published reports of first-stage mandibular reconstruction in patients with HFM do not fully clarify the details of the presenting dysmorphology of the native glenoid fossa and condylar components before surgery (Figure 28-9). As stated, for DO to be effective, a functional glenoid fossa and an adequate condyle must be present (i.e., in the presence of Type I or Type IIA malformation). Unfortunately, a high incidence of bony and fibrous ankylosis has been reported in conjunction with mandibular DO in HFM.[49] In addition, the consistent occurance of "undergrowth" after mandibular DO reconstruction carried out during the mixed dentition was confirmed by Meazzini and colleagues.[143] Those authors conducted a comparison study of long-term follow up until the completion of facial growth of two homogenous samples of children with HFM. The experimental group was treated with mandibular DO during the deciduous or early mixed dentition in an attempt to correct the mandibular deformity. The control group was not subjected to any treatment until adulthood. The experimental group included children (n = 14) who underwent mandibular ramus osteotomies with DO (mean age, 5.9 years) with a mean follow up of 11.2 years. With the use of quantitative measurements on serial panorex radiographs, the DO group was compared with the control group (n = 8). The study results document that facial proportions in patients with HFM are maintained throughout growth when no treatment is undertaken. Unfortunately, after ramus osteotomies with DO, the mandibular disproportions returned to their original level of asymmetry during growth. The authors concluded the following: 1) HFM does not progress with regard to the degree of facial asymmetry or deformity when the patient is left to mature naturally and 2) early intervention with mandibular ramus osteotomies and DO does not effectively reduce long-term facial asymmetry in the patient with HFM.

Ongoing problems when choosing the costochondral graft option to be carried out during the mixed dentition for the Type IIB or III malformation include overgrowth, undergrowth, ankylosis, and resorption of the mandible (Fig. 28-10). The overgrowth concerns are diminished but not eliminated when the costochondral graft procedure is carried out in a child who is in the late mixed dentition stage (i.e., 9 to 11 years old) and in whom only a minimum amount of cartilage (i.e., ≈2 mm) is left at the articulating surface of the graft (see section to follow concerning condylar reconstruction with costochondral graft).

If a first-stage mandibular procedure is carried out during the mixed dentition for the Type IIB or III malformation, then the need for definitive mandibular reconstruction in conjunction with a Le Fort I maxillary osteotomy and an osseous genioplasty at the time of skeletal maturity should be anticipated.[3,16,28,27] The idea of carrying out a mixed dentition mandibular reconstruction to avoid definitive

Text continued on p. 1121

• **Figure 28-9** A Hispanic girl who was born with a mild form of left-sided hemifacial microsomia. The mandibular malformation was Type I. At another institution, she underwent a mandibular distraction procedure when she was 4 years old with an external device; the goals were to "derotate" the mandible, to shift the chin to the midline, and to create a posterior open bite. An acrylic splint was then used in an attempt to hypererupt the maxillary molars over time. The patient is shown at the time of procedure and then 7 years later after her referral to this surgeon. Unfortunately, the procedure carried out did not improve the occlusion or correct the facial asymmetry. During the process, the left mandibular permanent molars (i.e., the first, second, and third molars) were injured, and secondary deformities of the mandible also resulted. **A,** The child is shown when she was 4 years old, during the mandibular distraction. **B,** She is also shown at 7 years of age note the distortion and deformity of the mandible and the facial scarring. **C,** Residual malocclusion as a result of the external distraction. **D,** Panorex radiograph taken when the child was 11 years old (i.e., 7 years after the distraction) that illustrates the injuries to the first, second, and third permanent molars on the distracted side.



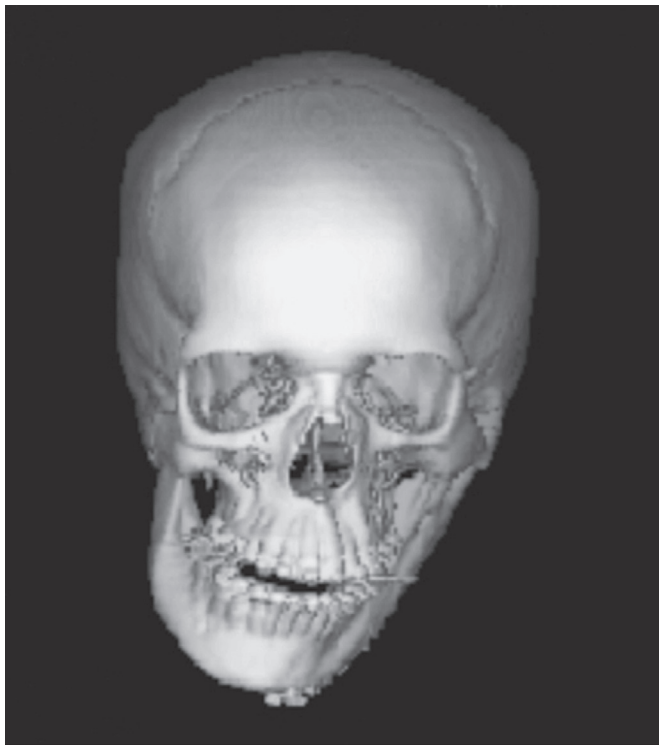
• **Figure 28-10** A newborn with left-sided hemifacial microsomia. She had a good airway as well as swallowing and chewing abilities. She was treated at another institution until she was referred to this surgeon when she was 15 years old. **A, B,** and **C,** Facial and computed tomography (CT) scan views taken during infancy that indicate microtia with an absent external auditory canal, hypoplasia and clefting through the zygomatic arch, and mandibular malformation (Type IIB) with a shortened posterior facial height and a rudimentary condyle.

3 years of age

5 years of age

• **Figure 28-10, cont'd D,** Frontal and CT scan views taken when the patient was 3 years old indicate no progression of the malformation. **E,** The same child when she was 5 years old just before undergoing a mandibular procedure at another institution. At surgery the mandible was derotated to shift the chin to the midline with the creation of a posterior open bite on the ipsilateral side. A costochondral graft was harvested and used to reconstruct the left condyle ascending ramus. An acrylic splint was then worn for several years in an attempt to "grow" the left maxilla and relieve the posterior open bite. The external ear was later reconstructed, but with suboptimal results.

Continued



15 years of age

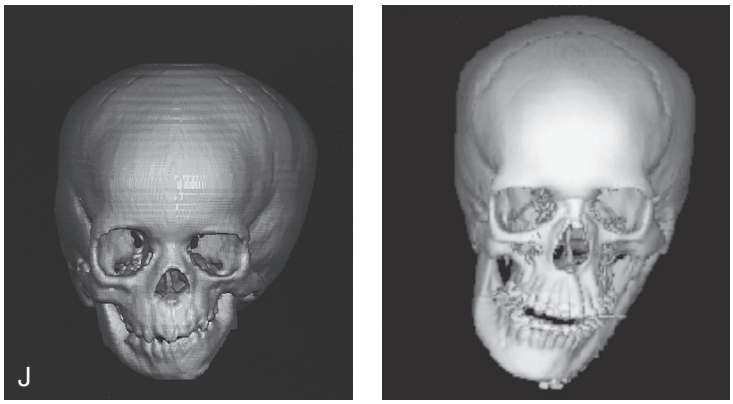
15 years of age

• **Figure 28-10, cont'd F, G, H,** and **I,** Facial and CT scan views of the patient when she was 15 years old, at the time of referral to this surgeon (i.e., 10 years after costochondral grafting) and without other interventions. There has been extensive overgrowth of the costochondral graft previously placed on the left side of the mandible. This has negatively altered (distorted) the growth of the whole mandible and the maxilla, and it has also resulted in limited mandibular mobility. There is gross distortion of the entire face, with negative effects on the airway, speech articulation, chewing ability, lip closure/posture, and self-esteem.

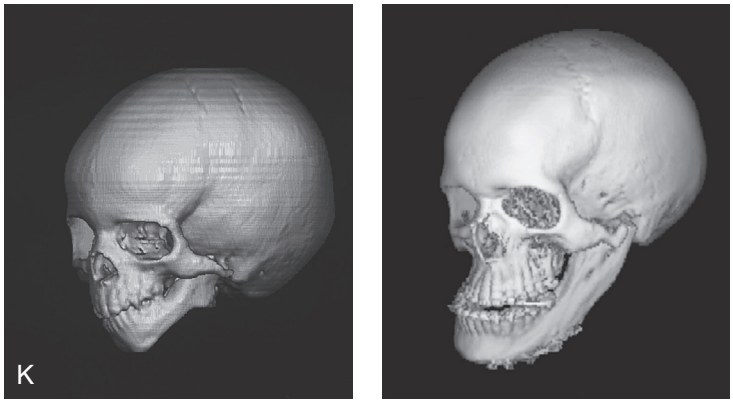
©Elsevier Inc.
Orthognathic Surgery; J.C. Posnick

• **Figure 28-10, cont'd**

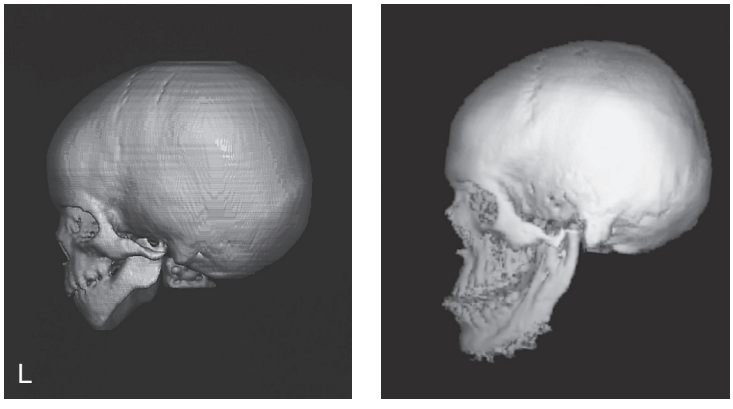
Continued



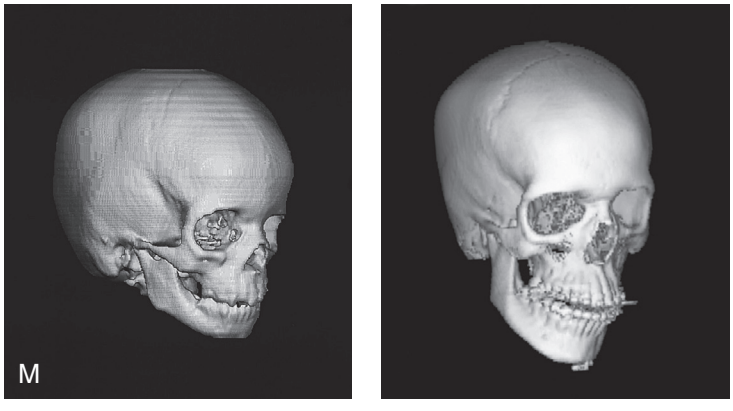
3 years 15 years



3 years 15 years



3 years 15 years



3 years 15 years

• **Figure 28-10, cont'd J, K, L,** and **M,** Comparable CT scan views are shown when the patient was 3 years old (i.e., before rib grafting) and at the age of 15 years (i.e., 11 years after rib grafting).

maxillomandibular orthognathic reconstruction during adulthood is generally unrealistic.

The rationale for postponing the correction of the dentofacial deformity until the time of "early" skeletal maturity is the same as that for the correction of the jaw deformity associated with a repaired cleft palate and most other routine dentofacial deformities (e.g., long face growth pattern, primary mandibular deficiency, maxillary deficiency in combination with relative mandibular excess). This rationale includes the consideration of the following: 1) avoiding injury to the developing permanent dentition and the inferior alveolar nerves 2) avoiding soft-tissue scarring 3) avoiding the loss of ramus marrow space that will complicate the success of necessary redo ramus osteotomies 4) minimizing negative psychosocial memories of earlier jaw surgery that may prevent the patient and family from later pursuing definitive reconstruction and 5) avoiding the iatrogenic three-dimensional deformation of the mandible that limits long-term success. In addition, any orthodontics carried out during the mixed dentition will not be reflective of the permanent dentition; a final course of braces would also be required.

Patients with HFM who have a significant degree of vertical, horizontal, and transverse maxillary deformity will also require Le Fort I maxillary osteotomy in segments. No lesser approach will be sufficient. The surgical objectives of the upper jaw surgery may include the following:

1. *Cant correction of the maxilla* with the asymmetric intrusion of the anterior maxilla to establish a more normal upper lip–to–tooth relationship in repose and during smiling as well as to improve lip competence (i.e., roll orientation)
2. *Correction of the maxillary dental midline to the facial midline* with control of the yaw orientation to achieve an improved cheek region appearance and a better angle of mandibular symmetry
3. Either extrusion or intrusion of the posterior maxilla on each side, depending on the extent of *counterclockwise rotation* required to achieve adequate horizontal projection of the mandible and chin (i.e., pitch orientation)
4. *Widening of the maxilla* (i.e., segmentation) to correct crossbites and to add facial fullness
5. *Horizontal advancement* to improve midface projection and to open the upper airway
6. Bone *grafting of the zygomatic buttress* on the ipsilateral side to improve facial symmetry
7. Provision of access for *intranasal procedures* (e.g., septoplasty, inferior turbinate reduction, recontouring of the pyriform rims and the nasal floor).

Safe and effective first-stage Le Fort I osteotomies carried out during the mixed dentition are not practical as a result of the location of the developing teeth and the expected inhibition of any postoperative horizontal maxillary growth.

Osteodistraction Reconstruction of the Mandible during Childhood

Since 1992, DO has been used as a technique in an attempt to correct mandibular morphology in young patients with HFM.* In 2002, a literature overview was published by **Mommaerts and colleagues** to address the long-term results of early DO among patients with HFM.[148] It was concluded that no convincing evidence supported the benefits of DO over other techniques for the treatment of HFM, and DO was not found to offer advantages with regard to the limiting of complications. In 2009, **Nagy and colleagues** published a critical and exhaustive literature review in search of evidence of long-term stability after early distraction osteogenesis of the mandible in patients with HFM.[163] Eighty-nine relevant articles were reviewed. Only 13 studies were found to have sufficient scientific merit to be included. The authors concluded the following:

1. The majority of the studies reported unstable results after DO with regard to facial symmetry that especially affected ramus height.
2. Clinicians frequently hypothesized that the volume of the soft tissues of the affected side would increase after DO in patients with HFM. Interestingly, the only published study that used a volumetric evaluation showed no postsurgical improvement of the soft-tissue deficiency on the affected side after DO reconstruction.
3. Even after accepting the shortcomings of certain study designs and evaluation methods, none of the published studies demonstrated the convincing long-term stability of the mandibular dimensions after DO mandibular elongation.
4. Relapse in regeneration after DO was seen in the majority of the studies that used reliable evaluation methods.
5. Repeated DO was reported to be necessary when pursuing attempts to maintain facial symmetry during growth in the patient with HFM. Therefore, any gains in mandibular dimensions after early DO were not stable, whether as a result of relapse or growth impairment.
6. In published reports, most children with HFM were still rated as "unattractive" after surgery and showed no "improvement in self-esteem." Children with HFM have an elevated risk for childhood psychosocial difficulties.
7. If early DO is the treatment that is selected to be carried out during childhood for HFM, the patient

*References 1, 4-6, 21, 23, 34, 39, 55, 58, 63, 68, 71, 72, 74, 79, 80, 82, 87, 100, 109-111, 113-116, 132, 136, 138-140, 144, 146-148, 150, 171, 173, 179, 182, 200, 202, 221, 222, 225, 232, 237, 241, 242, 246, 250, 252, 254, 257, 262, 265, 278, 279, 283, 284, 286

and family must accept that the procedure will have to be repeated later during the patient's life.

Costochondral Graft Reconstruction of the Mandible during Childhood

There are several basic theories regarding the mechanism of how mandibular growth occurs:

1. One theory of mandibular growth involves *chondroblastic proliferation* (i.e., cartilage formation with subsequent osseous replacement). Linear growth is primarily thought to be the result of condylar cartilage proliferation pushing the mandible forward and downward.
2. A second theory is the *functional matrix theory,* which explains mandibular growth primarily as a result of facial soft-tissue growth with subsequent projection (or pulling) of the mandible downward and forward in response to the forces of function. The condyle (and the condylar cartilage) is thought to merely secondarily react to the stimulating functional matrix forces that draw the mandible downward and forward. This is the theory behind the use of a Herbst appliance to "grow" a small mandible.
3. A third line of thinking is that both theories have merit. This explanation holds that a condylar growth center (i.e., chondroblastic proliferation) is present but that it is at least somewhat influenced by the physiologic function (i.e., functional matrix) of the mandible.

When the condyle and the components of the ascending ramus are hypoplastic or absent in patients with conditions such as HFM, surgeons have selected varied donor sources to replace the missing parts.* Autogenous replacements for the condyle have included 1) the metatarsal bones 2) the proximal head of the fibula 3) the costochondral junction of the rib (CCJ/rib) and 4) the sternoclavicular joint.

In general, the CCJ/rib bone is an expendable donor site and often an ideal morphologic contour match, with adequate strength at the mandibular recipient site. For these reasons, the CCJ/rib bone has been commonly used as a substitute for the "condylar growth center" and for the replacement of components of the ascending ramus. Unfortunately, in the skeletally immature patient, clinical observation after transplantation confirms unpredictable growth of the graft as compared with what would be considered "normal" growth or growth that is equivalent to the contralateral "normal" side of the mandible.

Clinical experience indicates that the CCJ/rib graft that is placed in the child or young teenager is prone to excessive growth. Although some would argue that overgrowth of the mandible and the need for additional surgical correction is a relatively minor consequence, experienced clinicians have found the secondary distortions in the maxillomandibular complex difficult to correct in a satisfactory way as compared with what could be achieved by single-stage reconstruction at the time of early skeletal maturity (Fig. 28-11). Unless pressing functional issues occur during childhood (e.g., obstructive sleep apnea), the advantages of a single-stage approach at jaw growth maturity to achieve favorable long-term function and aesthetic enhancement are difficult to ignore (Figs. 28-12 through 28-16).

Scientists began to focus on the biodynamics and growth of the transplanted CCJ/rib during the early 1990s. Researchers have addressed questions about differences in the biologic nature of the cartilage of the CCJ/rib; the epiphyseal growth plate; the mandibular condyle; and the sternoclavicular joint. It is now clear that the cartilage associated with the CCJ/rib and the epiphyseal plate of a long bone are derived from the primary cartilaginous template of the skeleton. These cartilages are relatively resistant to extrinsic competitive growth-related factors. Alternatively, the mandibular condyle and sternoclavicular cartilages arise from their own blastemas in association with the periosteum and then form as secondary cartilages.

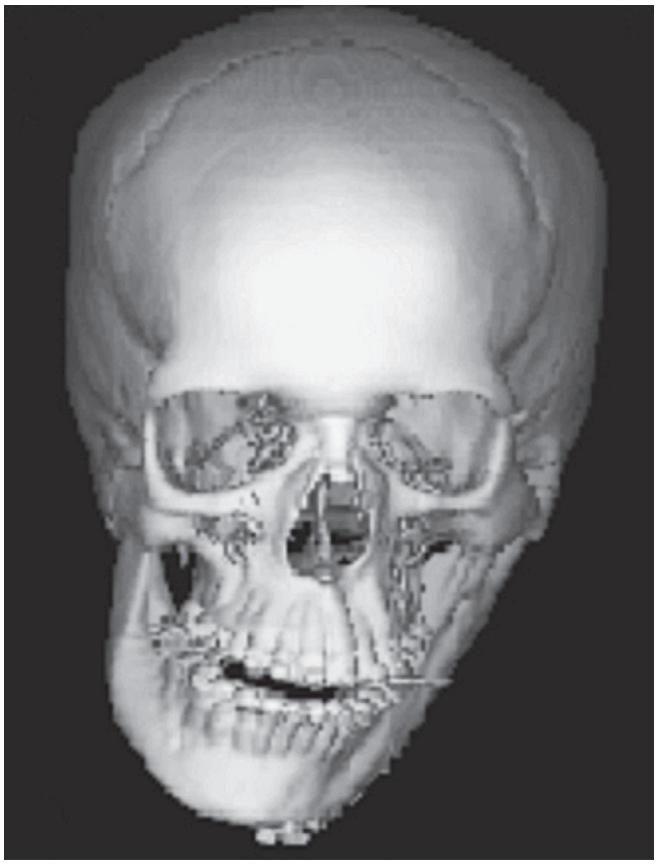
Peltomaki and colleagues used a rat model to analyze the dynamic histology that occurred with the growth of the CCJ/rib.[183-192] The observed histologic findings are dependent on the amount of cartilage that remains on the autogenous CCJ at the time of transplant. One of the initial findings of these authors was that "costochondral grafts do not adapt to the new environment, but rather preserve their inherent growth potential." In subsequent studies, Peltomaki and colleagues continued to explore the idea of whether the intrinsic growth of the CCJ/rib may also be influenced by the interaction of hormonal factors and masticatory movements.[183-192] It is now clear that variations in mandibular growth after the use of a CCJ/rib graft in both humans and experimental animal model simulations likely vary in accordance with the amount of cartilage used in the graft.

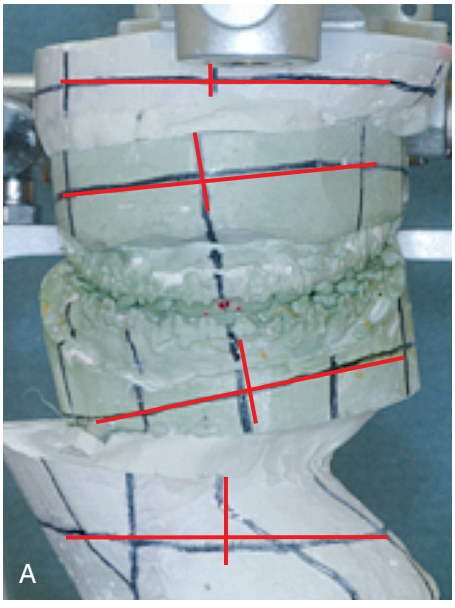
Peltomaki notes that, in the CCJ/rib the distance from the bone cartilage junction to the germinative zone is approximately 0.4 mm to 0.5 mm. It is evident that the germinative cells will invariably be included in a CCJ/rib graft. It is also evident that, in clinical practice, considerable variation in the amount of germinative cells maintained will occur. The reported clinical differences in mandibular growth after the replacement of the condylar process with a CCJ/rib graft is likely the result of variations in the amount of cartilage included in each graft. Peltomaki believes that the number of germinative cells has a direct impact on rib elongation (i.e., overgrowth). Clinicians should be cognitive of the fact that the amount of cartilage included in the graft is of significance with respect to the

Text continued on p. 1149

*References 2, 22, 37, 44, 45, 48, 60, 73, 118, 120, 126-128, 156, 158, 159, 160-162, 169, 177, 178, 183-192, 194, 195, 201, 209-211, 219, 224, 235, 285

©Elsevier Inc.
Orthognathic Surgery; J.C. Posnick





• **Figure 28-11** The 15-year-old girl who was born with hemifacial microsomia on the left side of the face and who was shown in Fig. 28-10 is shown at the time of referral to this surgeon. It has now been 10 years since she underwent left mandibular reconstruction with a costochondral graft. The mandibular opening was limited to approximately 23 mm but without bony ankylosis. The physical findings were the result of both the original malformation and the surgical intervention earlier during her life. She then underwent a combined orthodontic and surgical approach with this surgeon. **A,** Facial and computed tomography scan views taken when the patient was 15 years old indicate the extent of deformity and asymmetry. Articulated dental cast indicates analytic model planning to derotate the maxillomandibular complex (i.e., roll orientation correction).

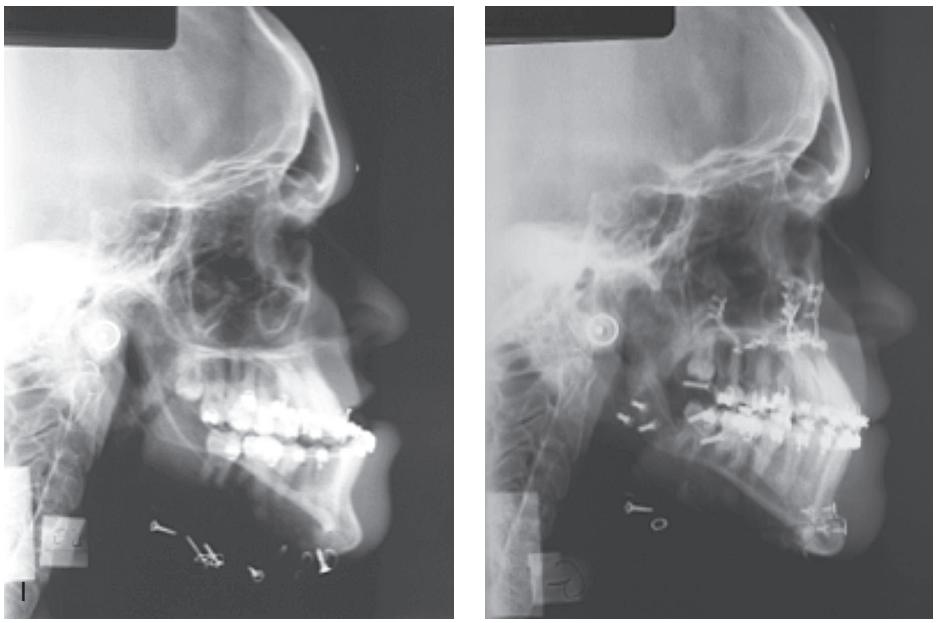
Continued

• **Figure 28-11, cont'd B,** Facial views in repose before and after reconstruction. **C,** Frontal views with smile before and after reconstruction.

• **Figure 28-11, cont'd D,** Right oblique facial views before and after reconstruction. **E,** Right profile views before and after reconstruction.

Continued

• **Figure 28-11, cont'd F,** Left oblique facial views before and after reconstruction. **G,** Left profile views before and after reconstruction.



• **Figure 28-11, cont'd H,** Occlusal views before and after reconstruction. **I,** Lateral cephalometric radiographs before and after reconstruction.

<1 year

5 years

10 years

14 years

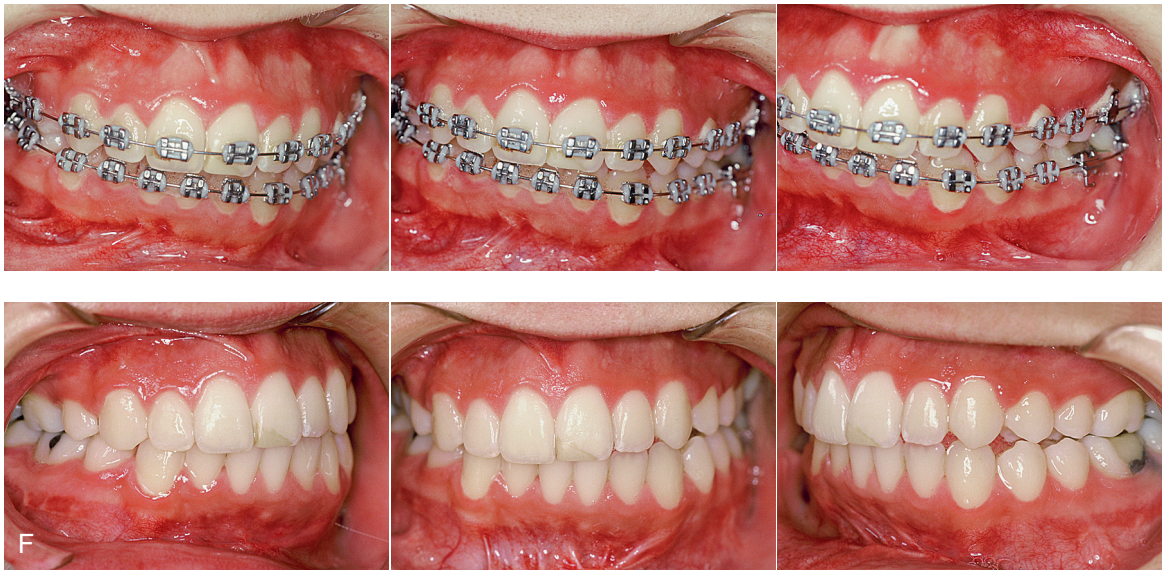
• **Figure 28-12** A child who was born with right-sided hemifacial microsomia (Type IIA mandible) was followed up longitudinally without skeletal interventions until jaw maturity. No progression of the malformation occurred with growth. The patient had nasal obstruction and forced mouth breathing, and she developed a long face growth pattern. There is clockwise rotation of the maxillomandibular complex with a retrognathic profile (i.e., pitch orientation). The chin is also vertically long and retrusive. There is severe canting of the maxillomandibular complex (i.e., roll orientation). The patient was referred to this surgeon and agreed to further orthodontic treatment and orthognathic surgery. Her procedures included maxillary Le Fort I osteotomy (vertical intrusion, counterclockwise rotation, and correction of cant); bilateral sagittal split ramus osteotomies (correction of asymmetry, counterclockwise rotation, and horizontal advancement); osseous genioplasty (vertical reduction and horizontal advancement); and septoplasty, inferior turbinate reduction, and nasal floor recontouring. **A,** Frontal views from 6 months to 14 years of age.

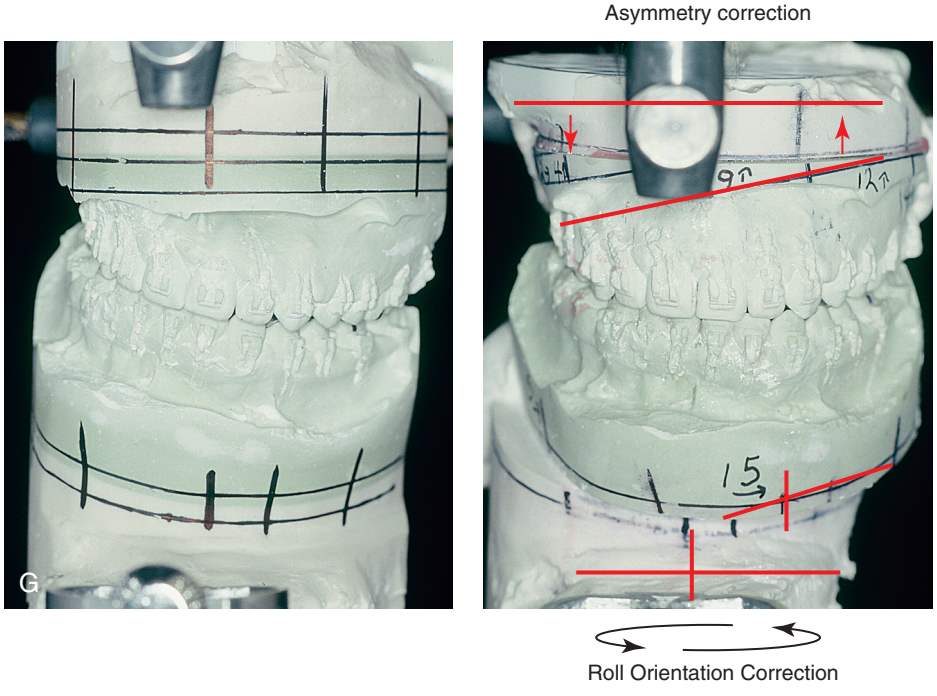
• **Figure 28-12, cont'd B,** Frontal views in repose before and after reconstruction. **C,** Frontal views with smile before and after reconstruction.

NOTE: The suboptimal external ear reconstruction performed by another surgeon.

Continued

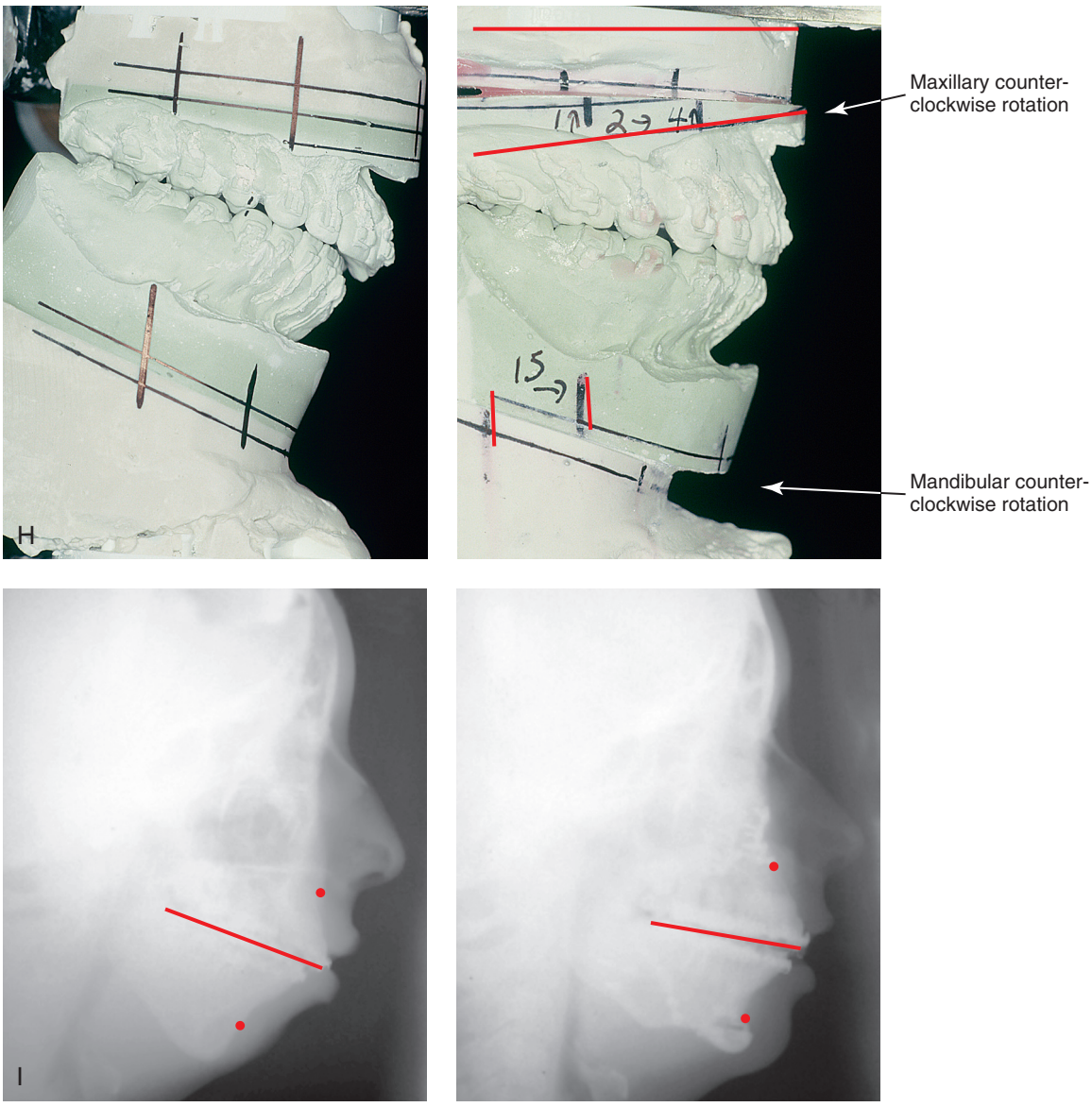
• **Figure 28-12, cont'd** **D,** Profile views before and after reconstruction. **E,** Worm's-eye views before and after reconstruction.





• **Figure 28-12, cont'd** **F,** Occlusal views before and after reconstruction. **G** and **H,** Articulated dental casts indicate analytic model planning.

Continued



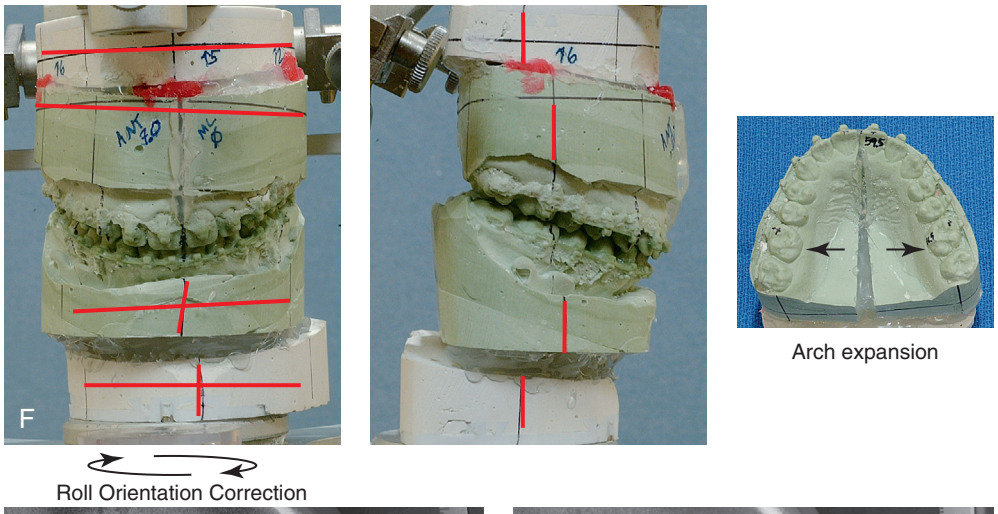
• **Figure 28-12, cont'd I,** Lateral cephalometric radiographs before and after reconstruction. *C,D, From Posnick JC: Hemifacial microsomia: evaluation and staging of reconstruction,* J Oral Maxillofac Surg *56:646, 1998.*

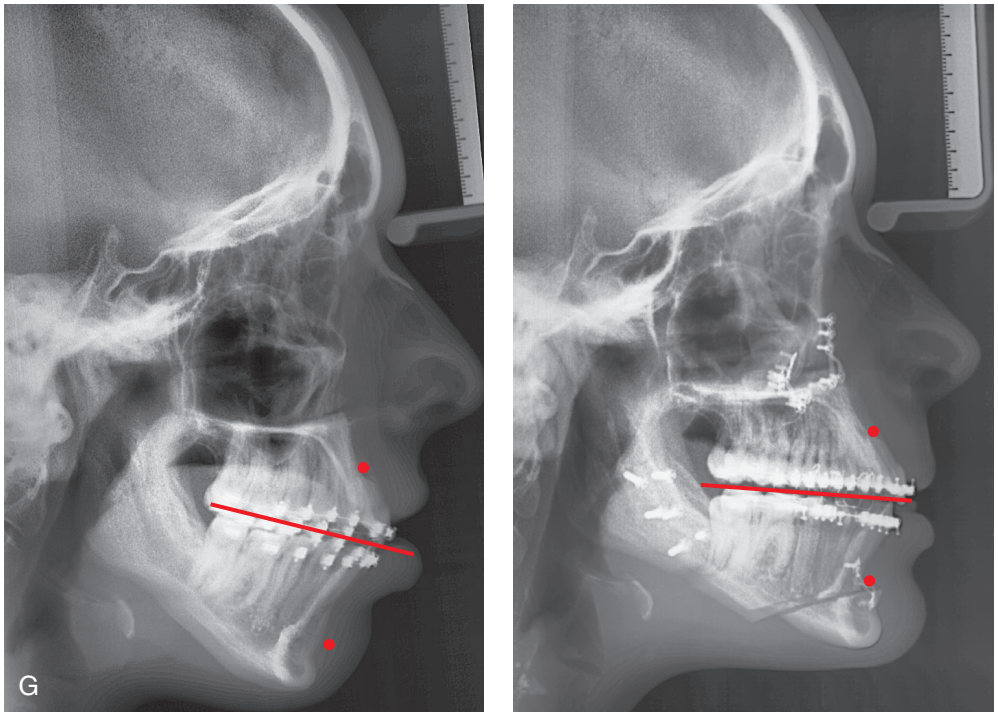
©Elsevier Inc.
Orthognathic Surgery; J.C. Posnick

• **Figure 28-13** A teenage girl who was born with a mild form of left-sided hemifacial microsomia. She presented to this surgeon as a teenager with a lifelong history of difficulty breathing through the nose, a long lower anterior facial height (i.e., gummy smile and lip incompetence), facial asymmetry, and retrusive jaws in profile. Her external ears were also malformed. She agreed to a combined orthodontic and surgical approach. The patient's procedures included maxillary Le Fort I osteotomy in segments (horizontal advancement, vertical intrusion, cant correction, and transverse widening); bilateral sagittal split ramus osteotomies (correction of asymmetry and horizontal advancement); osseous genioplasty (vertical shortening and horizontal advancement); and septoplasty, inferior turbinate reduction, and recontouring of the nasal floor. **A,** Frontal views in repose before and after reconstruction. **B,** Frontal views with smile before and after reconstruction.

Continued

• **Figure 28-13, cont'd C,** Oblique facial views before and after reconstruction. **D,** Profile views before and after reconstruction.

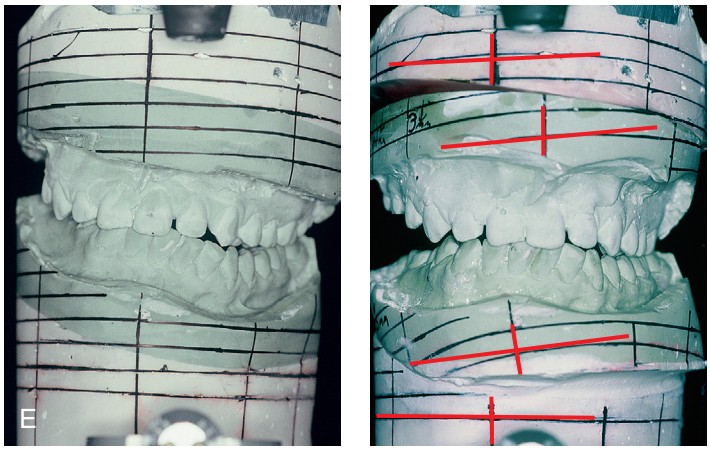




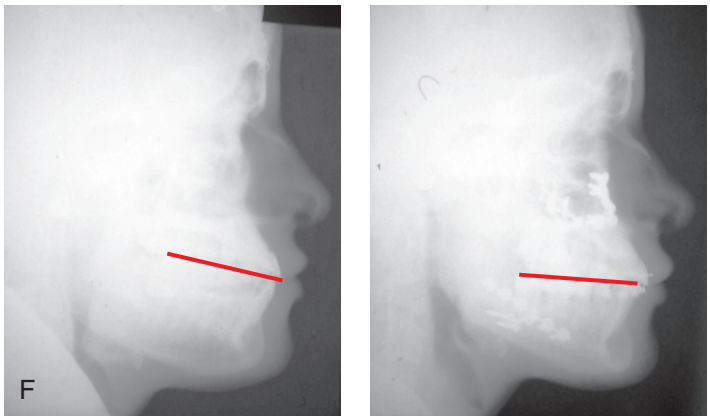
• **Figure 28-13, cont'd** **E,** Occlusal views with orthodontics in progress and after treatment. **F,** Articulated dental casts that indicate analytic model planning. **G,** Lateral cephalometric radiographs before and 1 year after reconstruction.

NOTE: The planned correction of pitch, yaw and roll orientation to enhance facial aesthetics.

• **Figure 28-14** A girl who was born with a severe form of hemifacial microsomia (right side) that included extensive involvement of the upper facial skeleton (including microophthalmia, lower facial skeleton issues (Type IIA mandibular malformation), and the external ear. She had undergone multiple intracranial cranio–orbital procedures, with limited success. She was referred to this surgeon as a teenager, and she underwent two additional reconstructive procedures. The first included cranio–orbito–zygomatic reconstruction through a coronal scalp incision (i.e., an intracranial approach) that required osteotomies of the cranial vault, the orbit, and the zygoma; this included autogenous cranial bone grafting. The second required orthognathic surgery, including a Le Fort I osteotomy (cant correction and horizontal advancement), bilateral sagittal split ramus osteotomies (asymmetry correction and horizontal advancement), and an osseous genioplasty (horizontal advancement) in combination with orthodontic treatment. The patient is shown before and after the two described procedures. **A,** Frontal views in repose before and after reconstruction. **B,** Worm's-eye view before surgery and frontal view with smile after reconstruction. **C,** Right and left profile views after reconstruction.

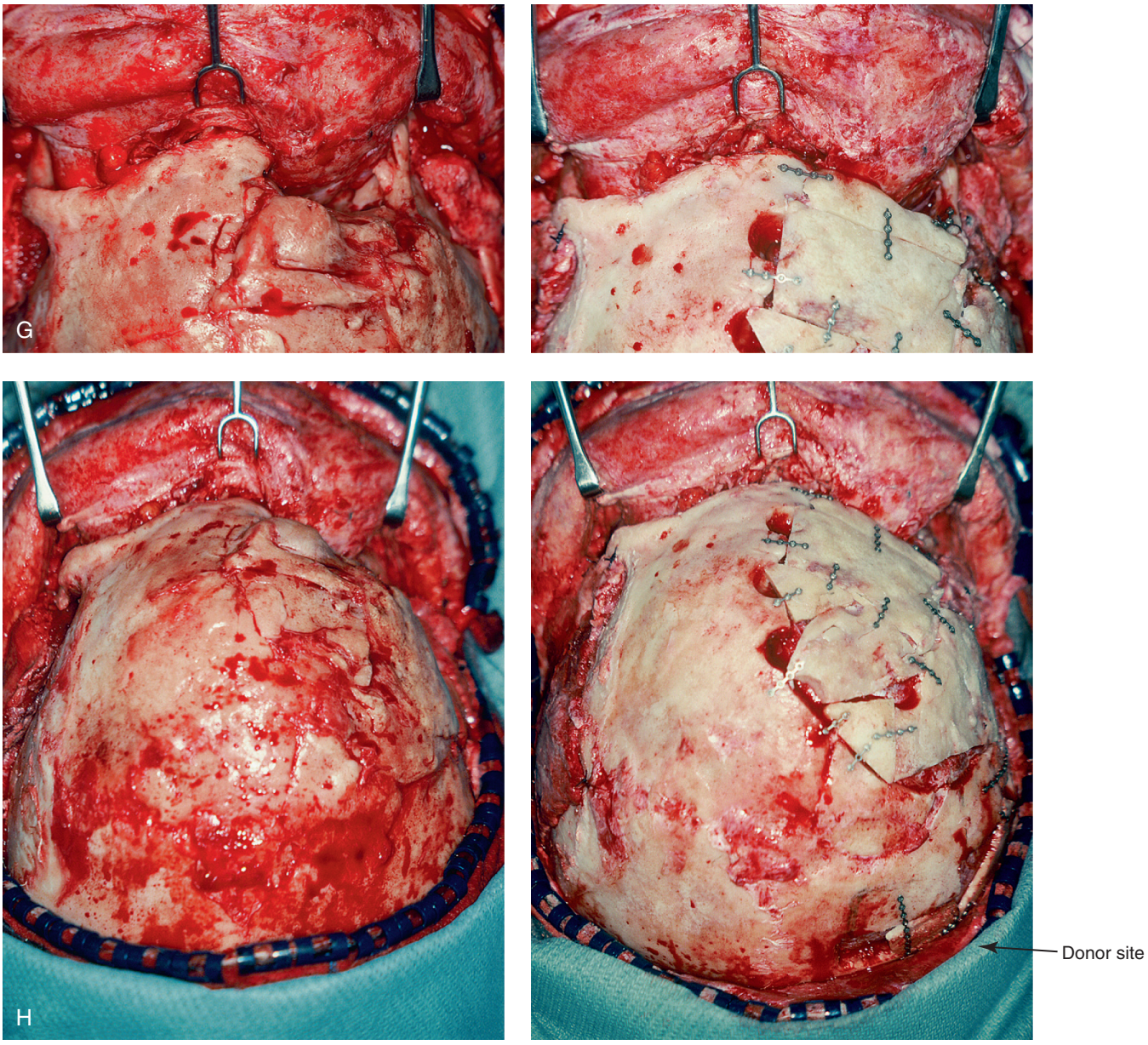


Roll Orientation Correction

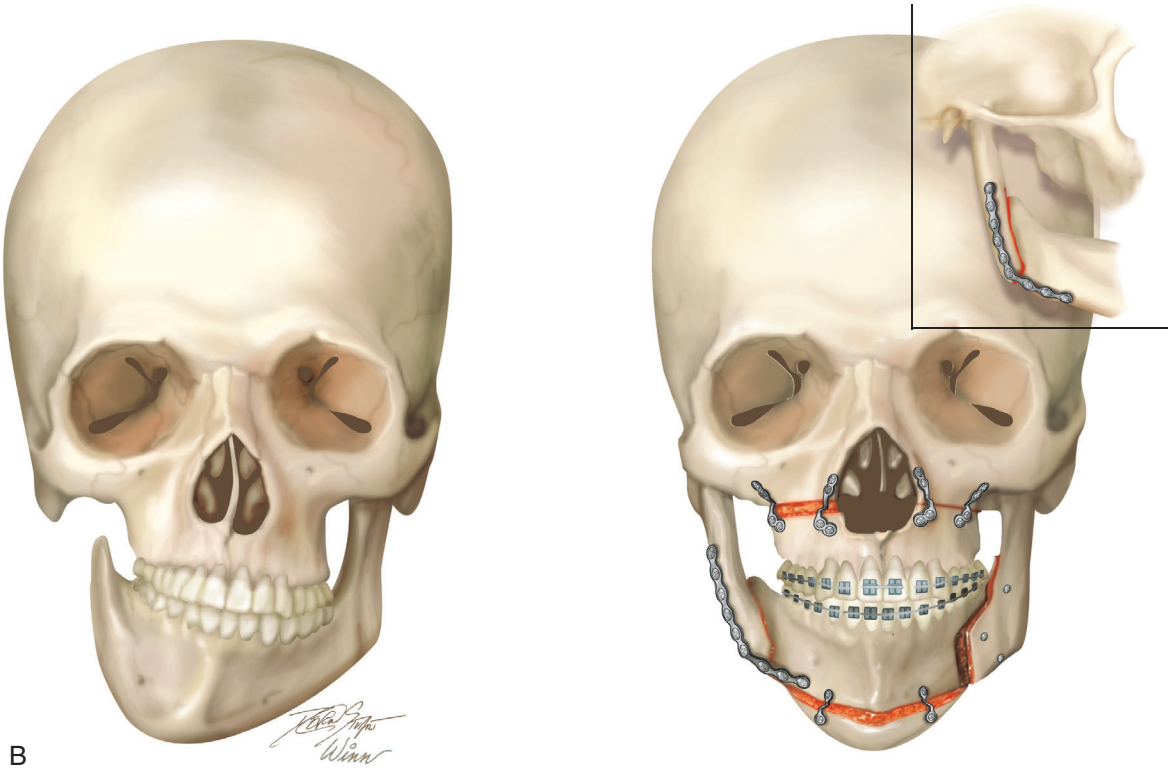


• **Figure 28-14, cont'd** **D,** Occlusal views before and after reconstruction. **E,** Articulated dental casts that indicate model planning. **F,** Lateral cephalometric radiographs before and after reconstruction.

Continued



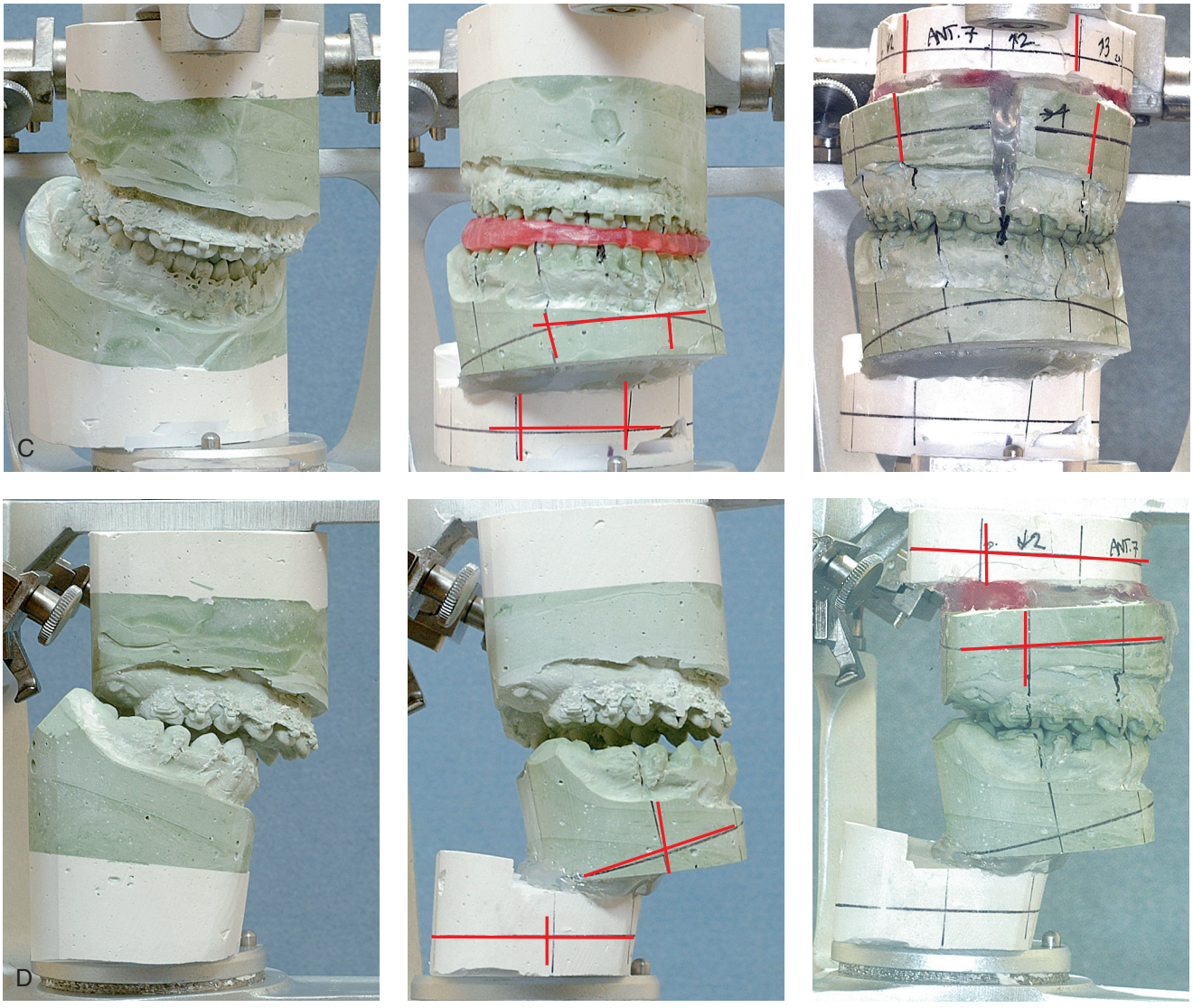
• **Figure 28-14, cont'd G,** Intraoperative views of upper orbits before and after redo cranio-orbit zygomatic reconstruction. **H,** Intraoperative views of cranial vault before and after redo cranio-orbito-zygomatic reconstruction. *A,B,D, From Posnick JC: Hemifacial microsomia: evaluation and staging of reconstruction,* J Oral Maxillofac Surg *56:648, 1998.*



• **Figure 28-15** A boy who was born with hemifacial microsomia involving the right side of face. There was also a Type IIB mandibular malformation. The patient was followed longitudinally without treatment intervention until he approached skeletal maturity. Facial views demonstrate no progression of the malformation. After the patient was in the adult dentition and his orthodontic decompensation was complete, he underwent single-stage reconstruction that included Le Fort I osteotomy in segments (cant correction, horizontal advancement arch expansion, and vertical adjustment); left-sided sagittal split ramus osteotomy; right mandibular reconstruction with a costochondral graft (horizontal advancement and asymmetry correction); osseous genioplasty (asymmetry correction and horizontal advancement); and septoplasty, inferior turbinate reduction, and bilateral otoplasty (i.e., ear set-back). **A,** Frontal facial views in repose from the ages of 11 to 14 without treatment intervention indicate no progression of deformity. **B,** Illustrations of the presenting skeletal deformities and of the reconstruction that was carried out. *Part B modified from an original illustration by Bill Winn.*

Continued

"Mandible-first" model planning



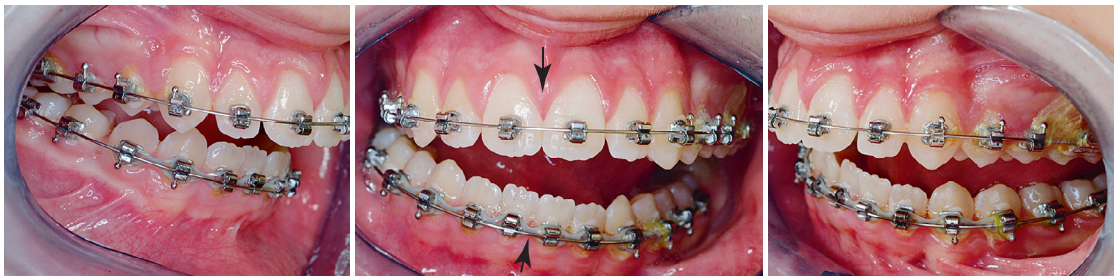
• **Figure 28-15, cont'd** **C** and **D,** Articulated dental casts that indicate analytic model planning with the use of the "mandible first" technique (see Chapter 14).

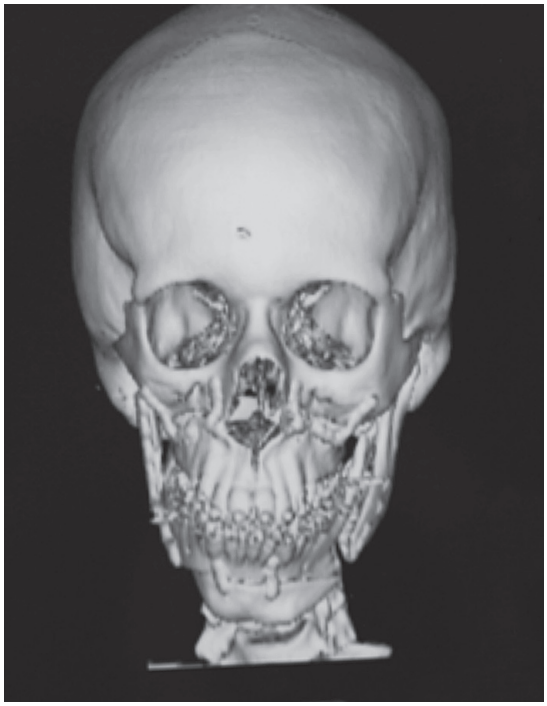
©Elsevier Inc.
Orthognathic Surgery; J.C. Posnick

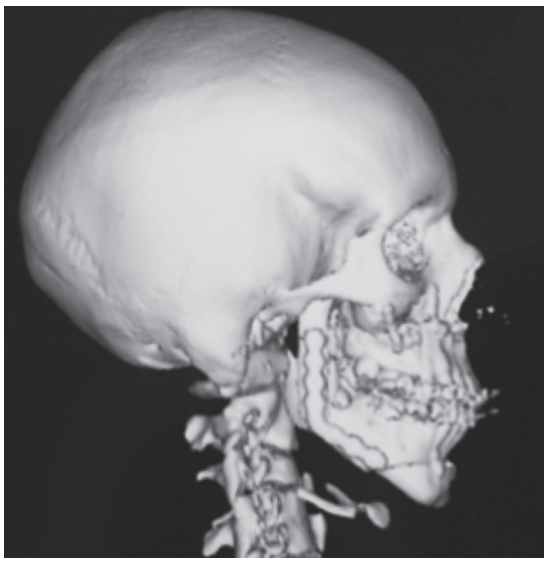
• **Figure 28-15, cont'd E,** Frontal views in repose before and after reconstruction. **F,** Frontal views with smile before and after reconstruction.

Continued

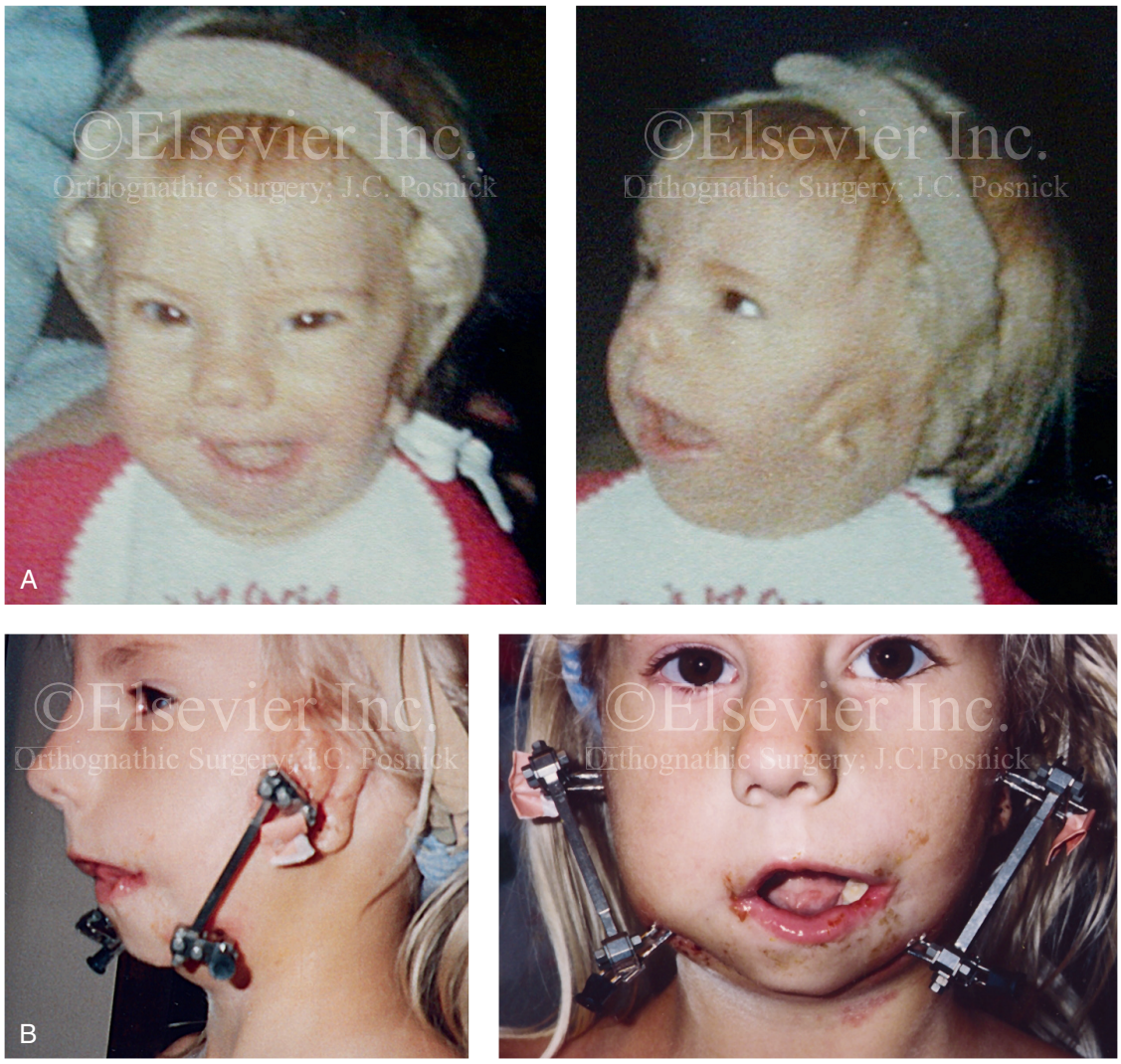
• **Figure 28-15, cont'd G,** Left oblique facial views before and after reconstruction. **H,** Right oblique facial views before and after reconstruction.





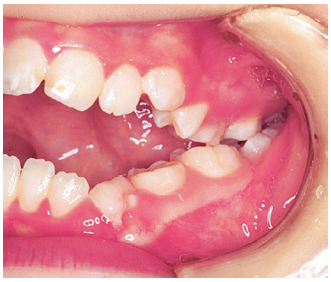


• **Figure 28-15, cont'd I,** Occlusal views with orthodontics in progress and after reconstruction. **J,** Frontal facial and computed tomography scan views after reconstruction. **K,** Profile computed tomography scan views before and after reconstruction that indicate mandibular malformation and costochondral graft reconstruction.



• **Figure 28-16** A newborn with a bilateral form of hemifacial microsomia that is more severe on the left side than the right and that involves the facial soft tissues and the jaws. The upper facial skeleton and the adnexal structures were essentially normal. The patient had an adequate airway and feeding ability at the time of birth and throughout childhood. At another institution when she was 7 years old, she underwent bilateral external distraction of the mandible. The procedure was unsuccessful for improving the mandibular dysmorphology or the occlusion. When she was 11 years of age, at another institution, the patient underwent a second mandibular distraction procedure with an internal device. This was also unsuccessful for improving the mandibular malformation or malocclusion. The patient had also undergone bilateral external ear reconstruction with a suboptimal result, and she wore external hearing aids. She presented to this surgeon when she was 15 years old with orthodontics in progress including 4 bicuspid extractions. She had a lifelong history of obstructed nasal breathing and deformities of the maxilla and the mandible. There was a marked Class II anterior open-bite malocclusion. An assessment was carried out that included an evaluation of the cervical spine, a complete computed tomography scan, and evaluation by a speech pathologist and an otolaryngologist. Reconstruction was executed and included septoplasty and inferior turbinate reduction; Le Fort I osteotomy (horizontal advancement, vertical adjustment, and counterclockwise rotation); bilateral ramus osteotomies (horizontal advancement and counterclockwise rotation) with interpositional grafting; and osseous genioplasty (vertical shortening and horizontal advancement). **A,** Facial views at 1 year of age, before any intervention. **B,** Facial views at 7 years of age, with bilateral mandibular distractors in place.

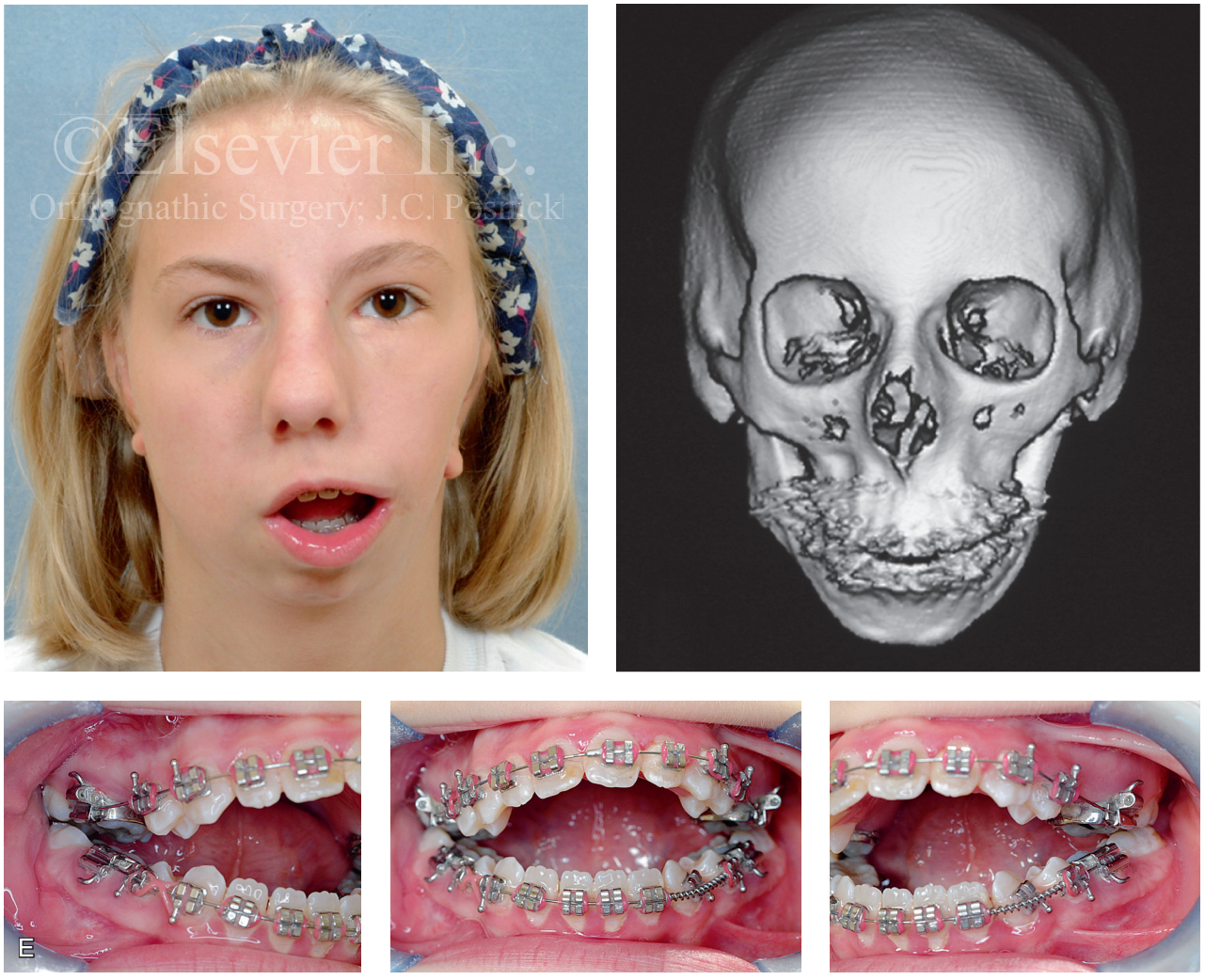
©Elsevier Inc.
Orthognathic Surgery: J.C. Posnick



8 years of age

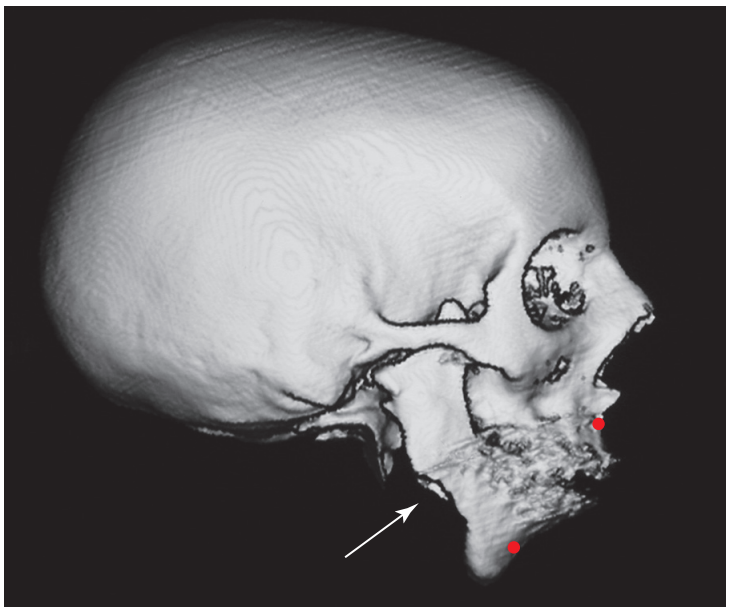
• **Figure 28-16, cont'd C,** Facial views at 8 years of age (i.e., 1 year after mandibular distraction) that indicate residual malocclusion and deformities of the maxilla and the mandible. **D,** Facial view at 11 years of age with bilateral mandibular distractions in place.

Continued

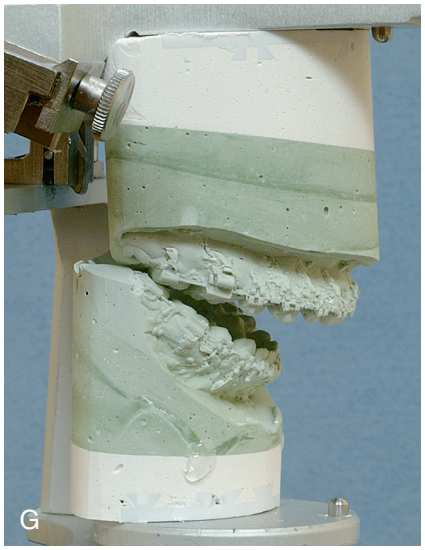


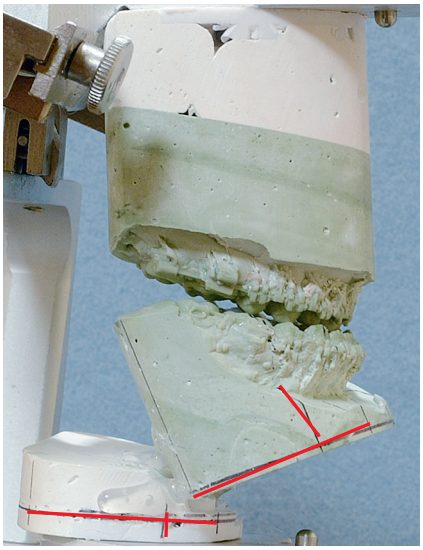
©Elsevier Inc.
Orthognathic Surgery; J.C. Posnick

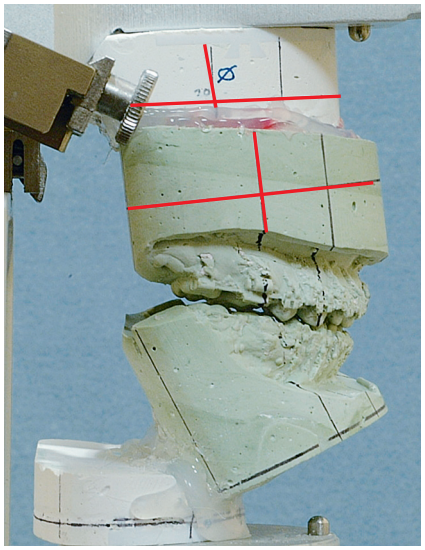
• **Figure 28-16, cont'd E,** Facial view, computed tomography scan, and occlusal views at 15 years of age at the time of referral to this surgeon.



Distraction osteogenesis altered ramus of mandible







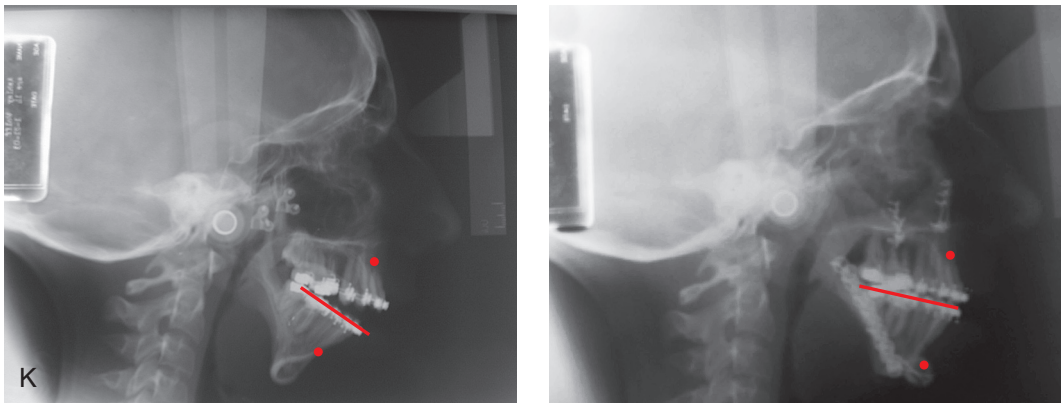
• **Figure 28-16, cont'd** **F,** Profile and computed tomography scan views at time of referral to this surgeon. **G,** Articulated dental casts that indicate analytic model planning with the "mandible first" technique (see Chapter 14).

Continued

• **Figure 28-16, cont'd H,** Facial views with smile before and after reconstruction. **I,** Oblique facial views before and after reconstruction.

Prior to definitive jaw surgery after 4 bicuspid extractions

After treatment



• **Figure 28-16, cont'd J,** Occlusal views with orthodontics in progress and after reconstruction. **K,** Lateral cephalometric radiographs before and after reconstruction.

NOTE: In this patient with HFM, the lack of effectiveness of the two mandibular distraction procedures carried out in the mixed dentition is documented.

future growth of the reconstructed condyle–ramus unit. It is currently impossible to estimate the optimal amount of cartilage to be preserved in each individual case. Nevertheless, clinical and experimental studies support the notion that, by including only a "small amount" of cartilage in the CCJ/rib graft, less postoperative growth (i.e., overgrowth) can be expected.

Peltomaki examined the growth of the mandible after CCJ/rib graft condyle replacement in marmoset monkeys. Interestingly, in adult animals, the amount of cartilage did not make any difference with regard to the postoperative mandibular measurements. In the growing animal, a postoperative gradual deviation of the midline to the unoperated side occurred. The greater the amount of cartilage left in the graft at the time of transplantation, the greater the amount of midline deviation observed over time.

For a series of human patients, Peltomaki reviewed the histology of surgically removed overgrown CCJ/rib grafts that were initially placed during childhood. On the basis of the data, he believes that the unpredictable growth of the constructed condyle–ascending ramus is a common occurrence. Clinical and histologic examination revealed that the clinical type of overgrowth (i.e., linear versus exuberant) was not related to any specific microarchitecture, which in itself showed considerable variation. This suggested that other local factors (e.g., mandibular movement, loading of the reconstructed condyle) may also have an effect on the subsequent and eventual growth and overgrowth.

In general, for a first-stage mandibular procedure that is carried out during childhood to be considered successful in a patient with HFM, the growth of the reconstructed side of the mandible should equal or at least be close to that of the contralateral normal mandible. Unfortunately, despite decades of surgical experience with the use of the CCJ/rib graft as a reconstructive option for the missing condyle–ascending ramus in the growing patient with HFM, this option has not passed the test. Overgrowth is a frequent and serious consequence. The mechanism that causes this postsurgical growth disturbance is now clear. Predictable methods of overcoming this complication while still allowing the CCJ/rib graft to be effective have not been realized.

Osteodistraction of the Residual Mandibular Deformity after Costochondral Graft Placement during Childhood

Wan and colleagues reported their findings for the reconstruction of Type IIB and III mandibular malformations in patients with HFM.[279] Their basic approach was to reconstruct the ipsilateral mandible with a CCJ/rib graft (N = 30 grafts, 27 patients) during the mixed dentition (mean age, 9.9 years ± 4.1 years). As a result of a high failure rate, this was followed by DO treatment of the residual deficient mandible. CCJ/rib graft failure that required regrafting occurred in 7 out of 30 cases (23%). Undergrowth that required DO during the mixed dentition occurred in 17 cases (57%). Overgrowth with severe distortion that required surgical correction occurred in 3 of the 30 cases (10%). For the majority of study patients who later required CCJ/rib graft DO, the complication rate was exceedingly high. In addition, all study patients (N = 27) required definitive orthognathic surgery at growth maturity, despite earlier procedures having been carried out.

Corcoran and colleagues presented a series of 8 patients with HFM who underwent mixed dentition costochondral reconstruction and who then submitted to DO.[33] They found a 62.5% complication rate. Stelnicki and colleagues similarly reported on 9 patients who underwent mixed dentition CCJ/rib graft reconstruction and then required distraction.[252A] They reported a 33% rate of fibrous nonunion as well as other significant complications and limited ultimate success. On the basis of current published studies, it must be concluded that first-stage mandibular reconstruction with the use of CCJ/rib grafts during the mixed dentition for patients with Type IIB and Type III HFM deformities is fraught with complications and achieves limited success. *The theory that a mandible reconstructed in this way can be reliably "distracted' later if it does not grow normally has proved to be false.*

Facial Soft-Tissue Reconstruction

A variety of well-intentioned surgical attempts to correct the soft-tissue deficiencies of the *eyelid adnexal regions* in patients with HFM have been carried out. The transposition of a pedicled upper eyelid skin–muscle flap to the lower eyelid's deficient region is not technically difficult, but the resulting adnexal scarring inevitably results in an operated look that generally detracts from any positive advantages of the procedure. For this reason, it may be best for the patient and the family to consult an experienced pediatric oculoplastic surgeon when considering even minor eyelid surgical procedures for the patient with HFM. Decisions about the removal of epibulbar or bulbar dermoid cysts from the adnexal region are based on eye function and eyelid aesthetics.

The inferior displacement of the *lateral canthus* in a patient with HFM is a reflection of both orbital dystopia and hypoplasia of the lateral canthi and other components of the eyelid structures. Correction of the orbital dystopia through osteotomies and bone grafting and the completion of a direct lateral canthopexy are helpful and should be carried out when indicated, but these procedures will not fully correct hypoplasia of the lateral canthal complex.

When *temporal fossa* hollowing is clinically noticeable in the patient with HFM, it is a reflection of multilevel hypoplasia that may include that of the skin, the temporoparietal fascia, the temporalis muscle, and the squamous portion of the temporal bone. Attempts to fill the soft-tissue defect with local pericranial or temporoparietal flaps are generally not effective, because these tissues are part of the deficiency. The intrinsic hypoplasia of these flaps (i.e., of the temporoparietal fascia and the temporalis muscle) also explains why they have limited utility for adnexal region subcutaneous augmentation.

For many patients, the extent of soft-tissue hypoplasia in the *preauricular–cheek region* will be distinctly noticeable at conversational distance, even after effective skeletal reconstruction.[61,86,89,91,117,123,124,149,204,205,244,245,258,268,269] This results from multilevel hypoplasia of the subcutaneous tissue; the fat; the muscles of mastication and facial expression; and the parotid gland. For these patients, a well-designed soft-tissue "free" vascularized composite flap should be considered. When meticulously designed and expertly executed, the transfer of soft-tissue flaps from other body regions (with microvascular reanastomosis) may be the alternative of choice. Parascapular free flaps have become the workhorse for the reestablishment of facial soft-tissue volume since this process was first described by Dos Santos.[40] Siebert and colleagues found that the parascapular flap allows for the reasonable correction of contour defects and yields improved facial aesthetic results in selected patients with HMF while minimizing scarring by providing vascularized soft tissue in the subcutaneous plane.[244,245] Fortunately, hypoplasia of the centrally located soft tissues of the face (i.e., the nose, the medial aspects of the upper and lower lips, and the submental region) is not seen in patients with HFM. The soft-tissue reconstruction requires an experienced microvascular surgeon working closely with a maxillofacial and a auricular surgeon to coordinate the flap inset; to minimize recipient-site morbidity; and to coordinate

skeletal and soft-tissue reconstruction. The timing of soft-tissue preauricular–cheek region reconstruction is generally planned to follow the skeletal reconstruction.

Advocates of the DO technique for mandibular reconstruction in patients with HFM had hoped that the soft tissues (i.e., the skin, subcutaneous tissue, and muscle) would increase in bulk when this method is used. However, this hoped-for advantage has not been documented by clinical studies. Of importance is that effective skeletal reconstruction by whatever means will favorably influence the aesthetic appearance of the soft-tissue envelope; however, it will not directly increase the soft-tissue volume.

Dermal fat grafts, *autogenous fat injections,* and other forms of soft-tissue augmentation have gained in popularity. The results of autogenous fat injection are variable and considered technique sensitive, but are generally not harmful and clearly promising. **Tanna and colleagues** investigated the use of serial autologous fat grafting to restore soft-tissue contour in patients with HFM.[259] Patients with moderate to severe HFM were divided into two groups. Group I included those who were undergoing microvascular free-flap reconstruction (i.e., inframammary extended circumflex scapular flaps, n = 10). Group II included patients who were undergoing multiple staged autogenous fat grafting (n = 21). The two patient groups had similar OMENS scores (2.4 and 2.3, respectively) and similar pre-reconstruction facial symmetry scores (74% and 75%, respectively). During the final evaluation, facial symmetry scores were 121% for the microvascular free-flap group and 99% for the fat-grafting group. Furthermore, no statistically significant difference between the microvascular and fat graft groups with regard to either patient or physician rating of overall satisfaction was noted.[67,107,259]

External Ear Reconstruction

In the hands of a few experts, surgical reconstruction of the external ear can be satisfactorily achieved with a staged approach.[8,10-15,54,260,261] The methods described by Brent continue to represent the reference standard for auricular reconstruction.[10-15] The successful placement of a well-sculpted autogenous cartilage framework is the foundation for a sound auricle repair. Brent prefers to wait until the patient is at least 6 years old, when rib cartilage is adequate for the reconstruction in most children. The synchondrotic region of the sixth and seventh ribs will then provide an ample cartilage block to use to form a framework for the ear. With the use of a prefabricated template of the contralateral ear and preoperatively determined measurements of the face, the ear's position and size are chosen, and a small preauricular incision is made. Other stages of auricular construction include lobule transposition, the detachment of the ear with a skin graft, hairline management, and tragus construction. Other options for external ear reconstruction include the use of alloplastic and homologous frameworks. Alloplastic materials currently in use include silicone or Medpor (Porex Surgical, Inc, College Park, Ga). These foreign substances are more susceptible to infection, soft-tissue wound dehiscence, and minor trauma, even decades after reconstruction. Techniques of tissue engineering with bovine cartilage cells continue to be tested; these cells can be grown in the laboratory and seeded on a synthetic biodegradable ear template that is then implanted beneath the skin of an immunocompetent mouse. Unfortunately, until tissue engineering evolves beyond the currently encountered immunogenic problems, sculpted autogenous rib cartilage will remain the material of choice for the surgical repair of the ear.

Before completing microtia reconstruction, a decision must be made about whether middle-ear surgery will eventually be carried out. Auricular construction should precede any middle-ear surgery; after an atretic ear canal is opened, the chances of obtaining a satisfactory auricular reconstruction are severely compromised as a result of scarring of the soft-tissue envelope. Consideration should also be given to the need for condyle-ascending ramus reconstruction (i.e. Type IIB and Type III malformations). If CCJ/rib graft harvesting will also be required then the location of each donor site becomes even more important.

External Auditory Canal and Middle-Ear Reconstruction

Occasionally, neurosensory hearing loss is present in the patient with HFM, but hearing loss is generally attributable to external auditory canal stenosis or atresia, hypoplasia of the middle-ear cavity, or ankylotic or missing ossicles.[17,18,223,280] Ankylotic or non-functioning ossicles limit hearing conduction to the same extent that would occur if the ossicles were absent. Generally, attempts to reconstruct the external auditory canal and the bones of the middle ear for patients with HFM are not carried out. As long as adequate hearing is present in the contralateral ear, clinical problems generally relate only to the patient's ability to locate the origin of the sound. For most patients, attempts to restore middle-ear function involve residual conductive hearing loss to the extent that "stereo" hearing is rarely achieved.

Bone-Anchored Hearing Aid Option

A *bone-anchored hearing aid* (BAHA) consists of a permanent titanium fixture that is surgically implanted into the skull bone behind the ear and a small detachable sound processor that clips onto the fixture. BAHAs are suitable for people with conductive or mixed hearing loss who cannot adequately benefit from or who prefer not to use conventional hearing aids.

Colquitt and colleagues completed a systematic review and economic evaluation of BAHAs for people who are bilaterally deaf. [29] Previous prospective studies of adults or children with bilateral hearing loss were eligible for review. Twelve studies were included; there were seven cohort

pre-post studies and five cross-sectional audiologic comparison studies. Comparisons were made between BAHAs and the following: 1) conventional hearing aids, which are also known as air-conduction hearing aids (ACHAs) 2) bone-conduction hearing aids (BCHAs) and 3) unaided hearing and ear surgery. The study also looked at 4) unilateral versus bilateral BAHAs. Outcomes reviewed included hearing measures, validated measures of quality of life, adverse events, and measures of cost-effectiveness.

There appeared to be some audiologic benefits of BAHAs for this group of patients including 1) improvements in speech as compared with BCHAs and 2) understanding and noise as compared with ACHAs (however, ACHAs may produce better audiologic results for other outcomes). In addition, 3) hearing was found to be improved with the use of BAHAs as compared with unaided hearing and 4) studies that compared unilateral with bilateral BAHAs suggested the benefits of bilateral BAHAs in many situations.

However, prospective case series of BAHAs reported rates of 6.1% to 19.4% for the loss of the implants over time. In addition, the financial analysis suggests that BAHAs may not be a cost-effective option in terms of hearing gain and probability of needing alternative aids. Interestingly, the greater the benefit to the individual from aided hearing and the greater the difference in the proportion of people using the hearing aid for more than 8 hours per day, the more likely the BAHAs were a cost-effective option.

Janssen and colleagues completed a systematic review of bilateral BAHAs for bilateral permanent conductive hearing loss.[88] This was a review of the literature from between 1977 and 2011, and it included studies in which subjects of any age had permanent conductive hearing loss and bilateral implanted BAHAs. Eleven studies met the criteria for data analysis. Bilateral BAHAs were found to provide audiologic benefit as compared with unilateral BAHAs (i.e., improved thresholds for tones, speech in quiet and in noise, improved localization/lateralization, and patient's perceived subjective benefit). Disadvantages of bilateral BAHAs included 1) listening in noise in some condition 2) presumed additional cost and 3) presumed increasing adverse event risk. The authors concluded that bilateral BAHAs provided additional objective and subjective benefit as compared with unilateral BAHAs.

Conclusions

Hemifacial Microsomia generally occurs sporadically and without a familial pattern. It is characterized by variable deficiencies of the skeletal and soft-tissue structures within the nasal placode and the first and second branchial arches, primarily on one side. Considerations during infancy and early childhood center on airway, feeding, hearing, vision, and the need for psychosocial family support. The degree of malformation present at the time of birth in the patient with HFM is believed to be relatively stable and non-progressive with age.

The current approach to the correction of the malformations associated with HFM is to stage the reconstruction to coincide with facial growth patterns, visceral functions, and psychosocial development. A precise morphologic analysis of each patient's anomalies followed by a broad-based reconstructive plan serves to clarify for the clinicians and the family the objectives of each stage and method of treatment. As we continue to define our rationale for the timing, methods, and extent of interventions and then to objectively evaluate the functional, morphologic, and psychosocial outcomes, we will improve the outlook for individuals who are affected by HFM.

References

1. Altug-Atac AT, Grayson BH, McCarthy JG: Comparison of skeletal and soft tissue changes following unilateral mandibular distraction osteogenesis. *Plast Reconstr Surg* 121:1751, 2008.
2. Anderson PJ, McLean NR, David DJ: Modified costochondral graft osteotomy in hemifacial microsomia. *Br J Plast Surg* 56(4):414–415, 2003.
3. Aoe T, Kohchi T, Mizuguchi T: Respiratory induced cyanosis plethysmography and pulse oximeter in the assessment of upper airway patency in a child with Goldenhar syndrome. *Can J Anesth* 37:369, 1990.
4. Baas EM, Horsthuis RBG, Lange JD: Subjective alveolar nerve function after bilateral sagittal split osteotomy or distraction osteogenesis of mandible. *J Oral Maxillofac Surg* 70:910–918, 2012.
5. Baek SH, Kim S: The determinants of successful distraction osteogenesis of the mandible in hemifacial microsomia from longitudinal results. *J Craniofac Surg* 16(4):549–558, 2005.
6. Batra P, Ryan FS, Witherow H, Calvert ML: Long-term results of mandibular distraction. *J Indian Soc Pedod Prev Dent* 24:30, 2006.
7. Beichman K: Response of muscles to altered skeletal morphology and functional rehabilitation of severely malformed mandibles in hemifacial microsomia [thesis], San Francisco, Calif, 1990, University of California School of Dentistry.
8. Bennun RD, Mulliken JB, Kaban LB, Murray JE: Microtia: A microform of hemifacial microsomia. *Plast Reconstr Surg* 76(6):859–865, 1985.
9. Bergmann C, Zerres K, Peschgens T, et al: Overlap between VACTERL and hemifacial microsomia illustrating a spectrum of malformations seen in axial mesodermal dysplasia complex (AMDC). *Am J Med Genet A* 121A(2):151–155, 2003.
10. Brent B: The correction of microtia with autogenous cartilage grafts: I. The classic deformity. *Plast Reconstr Surg* 66:11, 1980.
11. Brent B: The correction of microtia with autogenous cartilage grafts: II. Typical and complex deformities. *Plast Reconstr Surg* 66:13, 1980.
12. Brent B: Auricular repair using autogenous rib cartilage grafts: Two decades of experience with 600 cases. *Plast Reconstr Surg* 90:355, 1992.
13. Brent B: Advances in ear reconstruction with autogenous rib cartilage grafts: Personal experience with 1200 cases. *Plast Reconstr Surg* 104:319; 1999.
14. Brent B: The team approach to treating the microtia atresia patient. *Otolaryngol Clin North Am* 33:1353, 2000.
15. Brent B: Microtia repair with rib cartilage grafts: a review of personal experience with 1000 cases. *Clin Plast Surg* 29:257, 2002.
16. Burstein FD, Cohen SR, Scott PH, et al: Surgical therapy for severe refractory sleep apnea in infants and children: Application of the airway zone concept. *Plast Reconstr Surg* 96:34, 1995.

17. Caldarelli DD, Hutchinson JC, Gould HJ: Hemifacial microsomia: Priorities and sequence of comprehensive otologic management. *Cleft Palate J* 17:111, 1980.
18. Caldarelli DD, Hutchinson JG, Jr, Pruzansky S, et al: A comparison of microtia and temporal bone anomalies in hemifacial microsomia and mandibulofacial dysostosis. *Cleft Palate J* 17:103, 1980.
19. Canton E: Arrest of development of the left ramus of the lower jaw, combined with malformation of the external ear. *Trans Pathol Soc Lond* 12:237, 1861.
20. Carlotti AE, Jr: A variable treatment alternative for hemifacial microsomia: Case report. *J Maxillofac Surg* 9:176, 1981.
21. Carls FR, Sailer HF: Seven years clinical experience with mandibular distraction in children. *Craniomaxillofac Surg* 26:197, 1998.
22. Carlson DS: Growth of a costochondral graft in the rat temporomandibular joint [discussion]. *J Oral Maxillofac Surg* 50:857, 1992.
23. Cascone P, Gennaro P, Spuntarelli G, Lannetti G: Mandibular distraction: Evolution of treatment protocols in hemifacial microsomia. *J Craniofac Surg* 16(4):563–571, 2005.
24. Cavaliere CM, Buchman SR: Mandibular distraction in the absence of an ascending ramus and condyle. *J Craniofac Surg* 13(4):527–532, 2002.
25. Choung PH, Nam IW, Kim KS: Vascularized cranial bone grafts for mandibular and maxillary reconstruction: The parietal osteofascial flap. *J Craniomaxillofac Surg* 19:85, 1989.
26. Cohen MM, Jr: Variability versus "incidental findings" in the first and second branchial arch syndrome: Unilateral variants with anophthalmia. *Birth Defects Orig Artic Ser* 7:103, 1989.
27. Cohen SR, Holmes Re, Machado L, Magit A: Surgical strategies in the treatment of complex obstructive sleep apnea in children. *Paediatr Respir Rev* 3:25, 2002.
28. Cohen SR, Levitt CA, Simms C, Burstein FD: Airway disorders in hemifacial microsomia. *Plast Reconstr Surg* 103(1):27–33, 1999.
29. Colquitt JL, Jones J, Harris P, Loveman E, et al: Bone anchored hearing aids (BAHAs) for people who are bilaterally deaf; a systematic review and economic evaluation. *Health Technol Assess* 15:1–200, iii–iv, 2011.
30. Converse JM, Coccaro PJ, Becker M, Wood-Smith D: On hemifacial microsomia: The first and second branchial arch syndrome. *Plast Reconstr Surg* 51(3):268–279, 1973.
31. Converse JM, Horowitz SL, Coccaro PJ, et al: The corrective treatment of the skeletal asymmetry in hemifacial microsomia. *Plast Reconstr Surg* 52:221, 1973.
32. Converse JM, Shapiro HH: Treatment of developmental malformations of the jaws. *Plast Reconstr Surg* 19:173, 1952.
33. Corcoran J, Hubli EH, Salyer KE: Distraction osteogenesis of costochondral neomandibles: A clinical experience. *Plast Reconstr Surg* 100:311–315, discussion 316–317, 1997.
34. Cousley RR: A comparison of two classification system for hemifacial microsomia. *Br J Oral Maxillofac Surg* 31(2):78–82, 1993.
35. Cousley RR, Calvert ML: Current concepts in the understanding and management of hemifacial microsomia. *Br J Plast Surg* 50(7):536–551, 1997.
36. Cranin AN, Gallo L: Hemifacial microsomia with an edentulous mandible: Forme fruste or a new syndrome? *Oral Surg Oral Med Oral Pathol* 70(1):29–33, 1990.
37. Daniels S, Ellis E, III, Carlson DS: Histological analysis of costochondral and sternoclavicular grafts in the TMJ of the juvenile monkey. *J Oral Maxillofac Surg* 45:675–682, 1987.
38. David DJ, Mahatumarat C, Cooter RD: Hemifacial microsomia: A multisystem classification. *Plast Reconstr Surg* 80:525, 1987.
39. Diner PA, Tomat C, Soupre V, et al: Intraoral mandibular distraction: Indications, technique and long-term results. *Ann Acad Med Singapore* 25:634, 1999.
40. Dos Santos LF: The vascular anatomy and dissection of the free scapular flap. *Plast Reconstr Surg* 73(4):599–603, 1982
41. Duncan PA, Shapiro LR: Interrelationships of the hemifacial microsomia-VATER, VATER and sirenomelia phenotypes. *Am J Med Genet* 47(1):75–84, 1993.
42. Dyggve HV, Mikkelsen M: Partial deletion of the short arms of a chromosome of the 4–5 group (Denver). *Arch Dis Child* 40:82, 1965.
43. Edgerton MT, Marsh JL: Surgical treatment of hemifacial microsomia: First and second branchial arch syndrome. *Plast Reconstr Surg* 59:653, 1977.
44. Ellis E, III, Carlson DS: Histological comparison of the costochondral, sternoclavicular, and temporomandibular joints during growth in *Macaca mulatta*. *J Oral Maxillofac Surg* 44:312–321, 1986.
45. Ellis E, III, Carlson DS, Schneiderman ED: Growth of the mandible following replacement of the mandibular condyle: An experimental investigation in *Macaca mulatta*. *J Oral Maxillofac Surg* 60:1461–1470, 2002.
46. Ellis E, III, Johnson DG, Hayward JR: Use of orthognathic surgery simulating instrument in the presurgical evaluation of facial asymmetry. *J Oral Maxillofac Surg* 42:805, 1984.
47. Engiz O, Balci S, Unsal M, et al: 31 cases with oculoauriculovertebral dysplasia (Goldenhar syndrome): Clinical, neuroradiologic, audiologic and cytogenetic findings. *Genet Couns* 18(3):277–288, 2007.
48. Entin MA: Reconstruction in congenital deformity of the temporomandibular component. *Plast Reconstr Surg* 21:461, 1958.
49. Fan K, Andrews BT, Liao E, et al: Protection of the temporomandibular joint during syndromic neonatal mandibular distraction using condylar unloading. *Plast Reconstr Surg* 129:1151–1161, 2012.
50. Fan WS, Mulliken JB, Padwa BL: An association between hemifacial microsomia and facial clefting. *J Oral Maxillofac Surg* 63(3):330–334, 2005.
51. Farkas LG, James JS: Anthropometry of the face in lateral facial dysplasia: The unilateral form. *Cleft Palate J* 14:193, 1977.
52. Figueroa AA, Friede H: Craniovertebral malformations in hemifacial microsomia. *J Craniofac Genet Dev Biol Suppl* 1:167–178, 1985.
53. Figueroa AA, Pruzansky S: The external ear, mandible and other components of hemifacial microsomia. *J Maxillofac Surg* 10:200, 1982.
54. Firmin F: Microtia: Reconstruction by Brent's technique. *Ann Chir Plast Esthet* 37:119, 1992.
55. Fisher E, Staffenberg DA, McCarthy JG, et al: Histopathologic and biochemical changes in the muscles affected by distraction osteogenesis of the mandible. *Plast Reconstr Surg* 99:366, 1997.
56. Funayama E, Igawa HH, Nishizawa N, et al: Velopharyngeal insufficiency in hemifacial microsomia: Analysis of correlated factors. *Otolaryngol Head Neck Surg* 136(1):33–37, 2007.
57. Gallagher DM, Hyler RL, Epker BN: Hemifacial microsomia: An anesthetic airway problem. *Oral Surg Oral Med Oral Pathol* 49:2, 1980.
58. Garcia-Cimbrelo E, Olsen B, Ruiz-Yague M, et al: Ilizarov technique: Results and difficulties. *Clin Orthop* 283:116, 1992.
59. Gateno J, Xia JJ, Teichgraeber JF, et al: Clinical feasibility of computer-aided surgical simulation (CASS) in the treatment of complex cranio-maxillofacial deformities. *J Oral Maxillofac Surg* 65(4):728–734, 2007.
60. Glahn M, Winther JE: Metatarsal transplants as replacement for lost mandibular condyle (3-year follow-up). *Scand J Plast Reconstr Surg* 1:97, 1967.
61. Goldsmith D, Sharzer L, Berkman MD: Microvascular groin flaps in the treatment of hemifacial microsomia. *Cleft Palate Craniofac J* 29:44, 1992.
62. Gorlin RJ, Jue KL, Jacobsen U, et al: Oculoauriculovertebral dysplasia. *J Pediatr* 63:991, 1963.
63. Gosain AK: Distraction osteogenesis of the craniofacial skeleton. *Plast Reconstr Surg* 107:278, 2001.
64. Gosain AK, McCarthy JG, Pinto RS: Cervicovertebral anomalies and basilar impression in Goldenhar syndrome. *Plast Reconstr Surg* 93:498, 1994.
65. Gougoutas AJ, Singh DJ, Low DW, Bartlett SP: Hemifacial microsomia: Clinical features and pictographic representations of the

OMENS classification system. 120(7): 112–120, 2007.
66. Grabb WC: The first and second branchial arch syndrome. *Plast Reconstr Surg* 36:485, 1965.
67. Grahovac TL, Rubin JP: Discussion: An analysis of the experiences of 62 patients with moderate complications after full face fat injection for augmentation. *Plast Reconstr Surg* 129:1369, 2012
68. Grayson BH, McCormick S, Santiago PE, McCarthy JG: Vector of device placement and trajectory of mandibular distraction. *J Craniofac Surg* 8:473, 1997.
69. Greenberg F, et al: *Chromosome abnormalities associated with facio-auriculo-vertebral dysplasia*. Clinical Genetics Conference: Neural crest and craniofacial disorders. March of Dimes Birth Defects Meeting, Minneapolis, July 19–22, 1987.
70. Gripp L, Husgen W, Luhr HG, et al: Hemifacial microsomia: Extraoral appliance for the early treatment of an infant. *J Orofac Orthop* 58(6):352–360, 1997.
71. Gursoy S, Hukki J, Hurmerinta K: Five-year follow-up of mandibular distraction osteogenesis on the dentofacial structures of syndromic children. *Orthod Craniofac Res* 11:57, 2008.
72. Guyette TW, Polley JW, Figueroa AA, Cohen M: Mandibular distraction osteogenesis: Effects on articulation and velopharyngeal function. *J Craniofac Surg* 7:186, 1996.
73. Guyuron B, Lasa CI, Jr: Unpredictable growth pattern of costochondral grafts. *Plast Reconstr Surg* 90:880, 1992.
74. Hagino H, Sawaki Y, Ueda M: The fate of developing teeth in mandibular lengthening by distraction: An experimental study. *J Craniomaxillofac Surg* 29:94, 2001.
75. Hartsfield JK: Review of the etiologic heterogeneity of the oculo-auriculo-vertebral spectrum (hemifacial microsomia). *Orthod Craniofac Res* 10(3):121–128, 2007.
76. Harvold E: Centric relation: A study of pressure and tension systems in bone modeling and mandibular positioning. *Dent Clin North Am* 19:473, 1975.
77. Harvold E, Chierici G, Vargervik K, editors: *Treatment of hemifacial microsomia*, New York, 1983, Alan R. Liss.
78. Hathout EH, Elmendorf E, Bartley J: Hemifacial microsomia and abnormal chromosome 22. *Am J Med Genet* 76(1):71–73, 1998.
79. Hennig TB, Ellis E, III, Carlson DS: Growth of the mandible following replacement of the mandibular condyle with the sternal end of the clavicle: An experimental investigation in *Macaca mulatta*. *J Oral Maxillofac Surg* 50:1196, 1992.
80. Hollier LH, Kim JH, Grayson B, McCarthy JG: Mandibular growth after distraction in patients under 48 months of age. *Plast Reconstr Surg* 103:1361, 1999.
81. Horgan JE, Padwa BL, LaBrie RA, Mulliken JB: OMENS-Plus: Analysis of craniofacial and extracraniofacial anomalies in hemifacial microsomia. *Cleft Palate Craniofac J* 32(5):405–412, 1995.
82. Huisinga-Fisher CE, Vaandrager JM, Prahl-Anderson B: Longitudinal results of mandibular distraction osteogenesis in hemifacial microsomia. *J Craniofac Surg* 14(6):924–933, 2003.
83. Huisinga-Fisher CE, Vaandager JM, Prahl-Anderson B, van Ginkel FC: Masticatory muscle right-left differences in controls and hemifacial microsomia patients. *J Craniofac Surg* 15(1):42–46, 2004.
84. Huisinga-Fisher CE, Zonneveld FW, Vaandrager JM, Prahl-Anderson B: Relationship in hypoplasia between the masticatory muscle and the craniofacial skeleton in hemifacial microsomia, as determined by 3-D CT imaging. *J Craniofac Surg* 12(1):31–40, 2001.
85. Hwang K, Chung RS: Masks depicting hemifacial microsomia and cleft lip. *J Craniofac Surg* 13(5):721–723, 2002.
86. Iñigo F, Jimenez-Murat Y, Arroyo O, et al: Restoration of facial contour in Romberg's disease and hemifacial microsomia: Experience with 118 cases. *Microsurgery* 20(4):167–172, 2000.
87. Jansma J, Bierman MW, Becking AG: Intraoral distraction osteogenesis to lengthen the ascending ramus: Experience with seven patients. *Br J Oral Maxillofac Surg* 45:526, 2004.
88. Janssen RM, Hong P, Chadha NK: Bilateral bone-anchored hearing aids for bilateral permanent conductive hearing loss: A systematic review. *Otolaryngol Head Neck Surg* 147:412–422, 2012.
89. Ji Y, Li T, Shamburger S, et al: Microsurgical anterolateral thigh fasciocutaneous flap for facial contour correction in patients with hemifacial microsomia. *Microsurgery* 22(1):34–38, 2002.
90. Johnston MC, Bronsky PT: Animal models for human craniofacial malformations. *J Craniofac Genet Dev Biol* 11:277, 1991.
91. Jurkiewicz MJ, Nahai F: The omentum: Its use as a free vascularized graft for reconstruction of the head and neck. *Ann Plast Surg* 9:756, 1982.
92. Kaban LB, Moses MH, Mulliken JB: Correction of hemifacial microsomia in the growing child: A follow-up study. *Cleft Palate J* 23(Suppl 1):50, 1986.
93. Kaban LB, Moses MH, Mulliken JB: Surgical correction of hemifacial microsomia in the growing child. *Plast Reconstr Surg* 82:9, 1988.
94. Kaban LB, Mulliken JB, Murray JE: Three-dimensional approach to analysis and treatment of hemifacial microsomia. *Cleft Palate J* 18:90, 1981.
95. Kaban LB, Padwa BL, Mulliken JB: Surgical correction of mandibular hypoplasia in hemifacial microsomia: The case for treatment in early childhood. *J Oral Maxillofac Surg* 56(5):628–638, 1998.
96. Kamiji T, Ohmori K, Takada H: Clinical experiences with patients with facial bone deformities associated with hemifacial microsomia. *J Craniofac Surg* 2:181, 1992.
97. Kan EY, Doyle A, de Chalain TB: Morphological variability of inferior alveolar nerve in low-grade craniofacial microsomia. *J Craniofac Surg* 13(1):53–58, 2002.
98. Kane AA, Lo LL, Christensen D, et al: Relationship between bone and muscles of mastication in hemifacial microsomia. *Plast Reconstr Surg* 99:990, 1997.
99. Kaplan RG: Induced condylar growth in a patient with hemifacial microsomia. *Angle Orthod* 59(2):85–90, 1989.
100. Karp NS, Thorne CHM, McCarthy JG, et al: Bone lengthening in the craniofacial skeleton. *Ann Plast Surg* 24:231, 1990.
101. Kaye CI, Rollnick BR, Pruzansky S: Malformations of the auricle: Isolated and in syndromes: IV. Cumulative pedigree data. *Birth Defects Orig Artic Ser* 15:163, 1979.
102. Kearns G, Kaban LB, Padwa B, et al: Progression of facial asymmetry in patients with hemifacial microsomia. *J Oral Maxillofac Surg* 55(Suppl):48, 1997.
103. Kearns GJ, Padwa BL, Mulliken JB, Kaban LB: Progression of facial asymmetry in hemifacial microsomia. *Plast Reconstr Surg* 105(2):493–498, 2000.
104. Keegan CE, Mulliken JB, Wu BL, Korf BR: Townes-Brocks syndrome versus expanded spectrum hemifacial microsomia: Review of eight patients and further evidence of a "hot spot" for mutation in the SALL1 gene. *Genet Med* 3(4):310–313, 2001.
105. Kelberman D, Tyson J, Chandler DC, et al: Hemifacial microsomia: Progress in understanding the genetic basis of a complex malformation syndrome. *Hum Genet* 109(6):638–645, 2001.
106. Keogh IJ, Troulis MJ, Monroy AA, et al: Isolated microtia as a marker for unsuspected hemifacial microsomia. *Arch Otolaryngol Head Neck Surg* 133(10):997–1001, 2007.
107. Kim SM, Kim YS, Hong JW, et al: An analysis of the experiences of 62 patients with moderate complications after full face fat injection for augmentation. *Plast Reconstr Surg* 129:1359, 2012.
108. Kitai N, Murakami S, Takashima M, et al: Evaluation of temporomandibular joint in patients with hemifacial microsomia. *Cleft Palate Craniofac J* 41(2):157–162, 2004.
109. Knowles CC: Cephalometric treatment, planning and analysis of maxillary growth following bone grafting to the ramus in hemifacial microsomia. *Dent Pract Dent Rec* 17:28, 1966.
110. Ko EW, Hung KF, Huang CS, Chen PK: Correction of facial asymmetry with multiplanar mandible distraction: A one-year follow-up study. *Cleft Palate Craniofac J* 41(1):5–12, 2004.
111. Kofod T, Norhold SE, Pedersen TK, Jensen J: Unilateral mandibular ramus elongation

by intraoral distraction osteogenesis. *J Craniofac Surg* 16:247, 2005.
112. Krucylak CP, Schreiner MS: Orotracheal intubation of an infant with hemifacial microsomia using a modified lighted stylet. *Anesthesiology* 77:826, 1992.
113. Kulewicz M, Cudzilo D, Hortis-Dzierzbicka M, et al: Distraction osteogenesis in the treatment of hemifacial microsomia. *Med Wieku Rozwoj* 8:761, 2004.
114. Kunz C, Brauchli L, Moehle T, et al: Theoretical considerations for the surgical correction of mandibular deformity in hemifacial microsomia patients using multifocal distraction osteogenesis. *J Oral Maxillofac Surg* 61(3):364–368, 2003.
115. Kusnoto B, Figueroa AA, Polley JW: A longitudinal three-dimension evaluation of the growth pattern in hemifacial microsomia treated by mandibular distraction osteogenesis: A preliminary report. *J Craniofac Surg* 10:480, 1999.
116. Lai G, Zhiyong Z, Zang M, et al: Restoration of facial symmetry in hemifacial microsomia with mandibular outer cortex bone grafting combined with distraction osteogenesis. *Plast Reconstr Surg* 127:1997–2004, 2011.
117. LaRossa D, Whitaker L, Dabb R, et al: The use of microvascular free flaps for soft tissue augmentation of the face in children with hemifacial microsomia. *Cleft Palate J* 17:138, 1980.
118. Lauritzen C, Munro IR, Ross RB: Classification and treatment of hemifacial microsomia. *Scand J Plast Reconstr Surg* 19:33, 1985.
119. Lawson K, Waterhouse N, Gault DT, et al: Is hemifacial microsomia linked to multiple maternities? *Br J Plast Surg* 55(6):474–478, 2002.
120. Link JO, Hoffman DC, Laskin DM: Hyperplasia of a costochondral graft in an adult. *J Oral Maxillofac Surg* 51:1392–1394, 1993.
121. Longacre JJ, deStefano GA, Holmstrand K: The early versus the late reconstruction of congenital hypoplasias of the facial skeleton and skull. *Plast Reconstr Surg* 27:489, 1961.
122. Longacre JJ, deStefano GA, Holmstrand KE: The surgical management of the first and second branchial arch syndromes. *Plast Reconstr Surg* 31:507, 1963.
123. Longaker MT, Siebert JW: Microsurgical correction of bilateral facial contour deformities. *Plast Reconstr Surg* 98:951, 1996.
124. Longaker MT, Siebert JW: Microsurgical correction of facial contour in congenital craniofacial malformations: The marriage of the hard and soft tissue. *Plast Reconstr Surg* 98:942, 1996.
125. Luce EA, McGibbon B, Hoopes JE: Velopharyngeal insufficiency in hemifacial microsomia. *Plast Reconstr Surg* 30(4):602–606, 1977.
126. MacIntosh RB: A current spectrum of costochondral grafting. In Bell WH, editor: *Surgical correction of dentofacial deformities,* Vol III, Philadelphia, 1985, WB Saunders, pp 355–410.
127. MacIntosh RB: Costochondral grafting. In Bell WH, editor: *Modern practice in orthognathic and reconstructive surgery,* Vol II, Philadelphia, 1992, WB Saunders, pp 873–949.
128. MacIntosh RB, Henry FA: A spectrum of application of autogenous costochondral grafts. *J Maxillofac Surg* 5:257–267, 1977.
129. MacQuillian A, Biarda FU, Grobbelaar A: The incidence of anterior belly of digastrics agenesis in patients with hemifacial microsomia. *Plast Reconstr Surg* 126:1285–1290, 2010.
130. MacQuillan A, Vesely M, Harrison D, Grobbelaar A: Reanimation options in patients with hemifacial microsomia and marginal mandibular nerve palsy. *Plast Reconstr Surg* 112(7):1962–1963, 2003.
131. Maris CL, Endriga MC, Omnell ML, Speltz ML: Psychosocial adjustment in twin pairs with and without hemifacial microsomia. *Cleft Palate Craniofac J* 36:43, 1999.
132. Marquez IM, Fish LC, Stella JP: Two-year follow-up of distraction osteogenesis: Its effect on mandibular ramus height in hemifacial microsomia. *Am J Orthod Dentofacial Orthop* 117:130, 2000.
133. Marsh JL, Baca D, Vannier MW: Facial musculoskeletal asymmetry in hemifacial microsomia. *Cleft Palate J* 26:292, 1989.
134. Maruko E, Hayes C, Evans CA, et al: Hypodontia in hemifacial microsomia. *Cleft Palate Craniofac J* 38(1):15–19, 2001.
135. Mathog RH, Leonard MS: Surgical correction of Goldenhar syndrome. *Laryngoscope* 90:1137–1147, 1980.
136. Matsumoto K, Nakanishi H, Koizumi Y, et al: Occlusal difficulties after simultaneous mandibular and maxillary distraction in an adult case of hemifacial microsomia. *J Craniofac Surg* 15(3):464–468, 2004.
137. McCarthy JG, Grayson BH, Coccaro PJ, et al: Craniofacial microsomia. In McCarthy J, editor: *Plastic Surgery,* Vol 4, Philadelphia, 1990, WB Saunders, pp 3054–3100.
138. McCarthy JG, Schreiber J, Karp N, et al: Lengthening of the human mandible by gradual distraction. *Plast Reconstr Surg* 89:1, 1992.
139. McCarthy JG, Stelnicki EJ, Grayson BH: Distraction osteogenesis of the mandible: A ten-year experience. *Semin Orthod* 5:3, 1999.
140. McCarthy JG, Stelnicki EJ, Mehrara BJ, Longaker MT: Distraction osteogenesis of the craniofacial skeleton. *Plast Reconstr Surg* 107:1812, 2001.
141. McNamara JA, Carlson DS, Ribbens KA: *The effect of surgical intervention on craniofacial growth*, Ann Arbor, Mich, 1982, University of Michigan, Center for Human Growth and Development.
142. Meazzini MC, Caprioglio A, Garattini L, Poggio CE: Hemimandibular hypoplasia successfully treated with functional appliances: Is it truly hemifacial microsomia? *Cleft Palate Craniofac J* 45(1):50–56, 2008.
143. Meazzini MC, Mazzoleni F, Bozzetti A, Brusati R: Comparison of mandibular vertical growth in hemifacial microsomia patients treated with early distraction or not treated: Follow up till the completion of growth. *J Craniomaxillofac Surg* 40:105–111, 2012.
144. Meazzini MC, Mazzoleni F, Gabriele C, Bozzetti A: Mandibular distraction osteogenesis in hemifacial microsmia: Long-term follow-up. *J Craniomaxillofacial Surg* 33(6):370–376, 2005.
145. Melsen B, Bjerregaard J, Bundgaard M: The effect of treatment with functional appliance on a pathologic growth pattern of the condyle. *Am J Orthod Dentofacial Orthop* 90:503, 1986.
146. Molina F: Mandibular distraction: Surgical refinements and long-term results. *Clin Plast Surg* 31:443, 2004.
147. Molina F, Ortiz-Monasterio F: Mandibular elongation and remodeling by distraction: A farewell to major osteotomies. *Plast Reconstr Surg* 96:825, 1995.
148. Mommaerts MY, Nagy K: Is early osteodistraction a solution for the ascending ramus compartment in hemifacial microsomia? A literature study. *J Craniomaxillofac Surg* 30(4):201–207, 2002.
149. Mordick TG, Larossa D, Whitaker L: Soft tissue reconstruction of the face: A comparison of dermal–fat grafting and vascularized tissue transfer. *Ann Plast Surg* 29:390, 1992.
150. Morovic CG, Monasterio L: Distraction osteogenesis for obstructive apneas in patients with congenital craniofacial malformations. *Plast Reconstr Surg* 105:2321, 2000.
151. Moses JJ, Lo HH: The use of asymmetric yaw in the correction of lateral facial defects in hemifacial microsomia deformities: A case report. *Int J Adult Orthodon Orthognath Surg* 7(4):229–234, 1992.
152. Moss ML: The functional matrix. In Kraus BS, Riedel RA, editors: *Vistas of orthodontics*, Philadelphia, 1962, Lea and Febiger, pp 85–98.
153. Moss ML: The primacy of functional matrices in orofacial growth. *Dent Pract* 19:65–73, 1968.
154. Moss ML, Salentijn L: The primary role of functional matrices in facial growth. *Am J Orthod* 55:566–577, 1969.
155. Moulin-Romsee C, Verdonck A, Schoenaers J, Carels C: Treatment of hemifacial microsomia in a growing child: The importance of co-operation between the orthodontist and the maxillofacial surgeon. *J Orthod* 31(3):190–200, 2004.
156. Mulliken JB, Ferraro NF, Vento AR: A retrospective analysis of growth of the constructed condyle–ramus in children with hemifacial microsomia. *Cleft Palate J* 26:312, 1989.

157. Mulliken JB, Kaban LB: Analysis and treatment of hemifacial microsomia in childhood. *Clin Plast Surg* 14:91, 1987.
158. Munro IR: One-stage reconstruction of the temporomandibular joint in hemifacial microsomia. *Plast Reconstr Surg* 66:699, 1980.
159. Munro IR, Phillips JH, Griffin G: Growth after construction of the temporomandibular joint in children with hemifacial microsomia. *Cleft Palate J* 26:303, 1989.
160. Murray JE, Kaban LB, Mulliken JB: Analysis and treatment of hemifacial microsomia. *Plast Reconstr Surg* 74:186, 1984.
161. Murray JE, Kaban LB, Mulliken JB, et al: Analysis and treatment of hemifacial microsomia. *J Craniofac Surg* 33:377, 1985.
161A. Ongkosuwito EM, van Vooren J, van Neck JW, et al: Changes of mandibular ramal height, during growth in unilateral hemifacial microsomia patients and unaffected controls. *J Craniomaxillofac Surg* 41:92–97, 2013.
162. Murray JE, Mulliken JB, Kaban IB, et al: Twenty-year experience in maxillofacial surgery: An evaluation of early surgery on growth function and body image. *Ann Surg* 190:320, 1979.
163. Nagy K, Kuijpers-Jagtman AM, Mommaerts MY: No evidence for long-term effectiveness of early osteodistraction in hemifacial microsomia. *Plast Reconstr Surg* 124:2061–2071, 2009.
164. Naora H, Kimura M, Otani H, et al: Transgenic mouse model of hemifacial microsomia: Cloning and characterization of insertional mutation region on chromosome 10. *Genomics* 23(3):515–519, 1994.
165. Nargozian C, Ririe DG, Bennun RD, Mulliken JB: Hemifacial microsomia: Anatomical prediction of difficult intubation. *Paediatr Anaesth* 9(5):393–398, 1999.
166. Obwegeser HL: Zur korrektur der dysostosis ostomandibularis. *Schweiz Monatschr Zahnhlkd* 80:331–340, 1970.
167. Obwegeser HL: Correction of skeletal anomalies of otomandibular dysostosis. *J Maxillofac Surg* 2:73–92, 1974.
168. Obwegeser HL, Lello GE, Sailer HF: Otomandibular dysostosis. In Bell WH, editor: *Surgical correction of dentofacial deformities. New concepts,* Vol III, Philadelphia, 1985, WB Saunders, 14:639–661.
169. Oeltomaki T, Vahatalo K, Ronning O: The effects of a unilateral costochondral graft on the growth of the marmoset mandible. *J Oral Maxillofac Surg* 60(11):1307–1314; discussion 1314–1315, 2002.
170. Ortiz-Monasterio F: Early mandibular and maxillary osteotomies for the correction of hemifacial microsomia: A preliminary report. *Clin Plast Surg* 9:509, 1982.
171. Ortiz-Monasterio F, Molina F, Andrade L, et al: Simultaneous mandibular and maxillary distraction in hemifacial microsomia in adults: Avoiding occlusal disorders. *Plast Reconstr Surg* 100:852, 1997.
172. Ousterhout DK, Vargervik K: Surgical treatment of the jaw deformities in hemifacial microsomia. *Aust N Z J Surg* 57(2):77–87, 1987.
173. Ow AT, Cheung LK: Meta-analysis of mandibular distraction osteogenesis: Clinical applications and functional outcomes. *Plast Reconstr Surg* 121:54, 2008.
174. Padwa BL, Bruneteau RJ, Mulliken JB: Association between "plagiocephaly" and hemifacial microsomia. *Am J Med Genet* 47(8):1202–1207, 1993.
175. Padwa BL, Evans CA, Pillemer FC: Psychosocial adjustment in children with hemifacial microsomia and other craniofacial deformities. *Cleft Palate Craniofac J* 28:354, 1991.
176. Padwa BL, Kaiser MO, Kaban LB: Occlusal cant in the facial plane as a reflection of facial asymmetry. *J Oral Maxillofac Surg* 55:811, 1997.
177. Padwa BL, Mulliken JB, Maghen A, Kaban LB: Midfacial growth after costochondral graft construction of the mandibular ramus in hemifacial microsomia. *J Oral Maxillofac Surg* 56:122, 1998.
178. Padwa BL, Mulliken JB, Maghen BA, et al: Midfacial growth after costochondral graft reconstruction of the mandibular ramus in hemifacial microsomia. *J Oral Maxillofac Surg* 55:1144, 1997.
179. Padwa BL, Zaragoza SM, Sonis AL: Proximal segment displacement in mandibular distraction osteogenesis. *J Craniofac Surg* 13(2):293–296; discussion 297, 2002.
180. Paeng JY, Lee JH, Kim MJ: Condyle as the point of rotation for 3-D planning of distraction osteogenesis for hemifacial microsomia. *J Craniomaxillofac Surg* 35(2):91–102.
181. Pashayan H, Pinsky L, Fraser FC: Hemifacial microsomia-oculo-auriculo-vertebral dysplasia: A patient with overlapping features. *J Med Genet* 7(2):185–188, 1970.
182. Patterson AR, Brady G, Loukota RA: Distraction of the mandibular ramus in hemifacial microsomia with a defect of the glenoid fossa. *Br J Oral Maxillofac Surg* 45(7):599–600, 2007.
183. Peltomaki T: Growth of a costochondral graft in the rat temporomandibular joint. *J Oral Maxillofac Surg* 50(8):851–857; discussion 857–858, 1992.
184. Peltomaki T: Histologic structure of human costochondral junction. *Plast Reconstr Surg* 94(5):585–588, 1994.
185. Peltomaki T, Isotupa K: The costochondral graft: A solution or a source of facial asymmetry in growing children: A case report. *Proc Finn Dent Soc* 87(1):167–176, 1991.
186. Peltomaki T, Hakkinen L: Growth of the ribs at the costochondral junction in the rat. *J Anat* 181:259–264, 1992.
187. Peltomaki T, Kylamarkula S, Vinkka-Puhakka H, et al: Tissue-separating capacity of growth cartilages. *Eur J Orthod* 19(5):473–481, 1997.
188. Peltomaki T, Quevedo LA, Jeldes G, Ronning O: Histology of surgically removed overgrown osteochondral rib grafts. *J Craniomaxillofac Surg* 30(6):355–360, 2002.
189. Peltomaki T, Ronning O: Interrelationship between size and tissue-separating potential of costochondral transplants. *Eur J Orthod* 13(6):459–465, 1991.
190. Peltomaki T, Ronning O: Costochondral graft as replacement of a dysplastic mandibular condyle [comment]. *Plast Reconstr Surg* 92(5):981–983, 1993.
191. Peltomaki T, Ronning O: Growth of costochondral fragments transplanted from mature to young isogeneic rats. *Cleft Palate Craniofac J* 30(2):159–163, 1993.
192. Peltomaki T, Vahatalo K, Ronning O: The effect of a unilateral costochondral graft on the growth of the marmoset mandible. *J Oral Maxillofac Surg* 60(11):1307–1314; discussion 1314–1315, 2002.
193. Perkins JA, Sie KC, Milczuk H, et al: Airway management in children with craniofacial anomalies. *Cleft Palate Craniofac J* 34:135, 1997.
194. Perrott DH: Clinical and computed tomographic findings in costochondral grafts replacing the mandibular condyle [discussion]. *J Oral Maxillofac Surg* 54:1400, 1996.
195. Perrot DH, Umeda H, Kaban LB: Costochondral graft construction/reconstruction of the ramus/condyle unit: Long-term follow-up. *Int J Oral Maxillofac Surg* 23:321–328, 1994.
196. Pertschuk MJ, Whitaker LA: Psychosocial adjustment and craniofacial malformations in childhood. *Plast Reconstr Surg* 75:177, 1985.
197. Phillips J, Whitaker LA: The social effects of craniofacial deformity and its correction. *Cleft Palate J* 16:7, 1979.
198. Pilai RR, Singh IJ: Hemifacial microsomia with Goldenhar syndrome: Report case. *Dent Dig* 76(9):382–385, 1970.
199. Pillemer FG, Cook KV: The psychosocial adjustment of pediatric craniofacial patients after surgery. *Cleft Palate J* 26:201, 1989.
200. Politi M, Sembronio S, Robiony M, Costa F: The floating bone technique of the vertical ramus in hemifacial microsomia: Case report. *Int J Adult Orthodon Orthognath Surg* 17(3):223–229, 2002.
201. Politis C, Fossion E, Bossuyt M: The use of costochondral grafts in arthroplasty of the temporomandibular joint. *J Craniomaxillofac Surg* 15:345–354, 1987.
202. Polley JW, Figueroa AA: Distraction osteogenesis: Its application in severe mandibular deformities in hemifacial microsomia. *J Craniofac Surg* 8(5):422–430, 1997.
203. Polley JW, Figueroa AA, Jein-Wein Liou E, et al: Longitudinal analysis of mandibular

asymmetry in hemifacial microsomia. *Plast Reconstr Surg* 99:328, 1997.
204. Poole MD: A composite flap for early treatment of hemifacial microsomia. *Br J Plast Surg* 42(2):163–172, 1989.
205. Poole MD: Hemifacial microsomia. *World J Surg* 13:396, 1989.
206. Poon CC, Meata JG, Heggie AA: Hemifacial microsomia: Use of the OMENS-Plus classification at the Royal Children's Hospital of Melbourne. *Plast Reconstr Surg* 111(3):1011–1018, 2003.
207. Pope AW, Speltz ML: Research on psychosocial issues of children with craniofacial anomalies: Progress and challenges. *Cleft Palate Craniofac J* 34:371, 1997.
208. Pope AW, Ward J: Self-perceived facial appearance and psychosocial adjustment in preadolescents with craniofacial anomalies. *Cleft Palate Craniofac J* 34:396, 1997.
209. Posnick JC: Discussion: Midface growth after costochondral graft reconstruction of the mandibular ramus in hemifacial microsomia. *J Oral Maxillofac Surg* 56:127–128, 1998.
210. Posnick JC: Surgical correction of mandibular hypoplasia in hemifacial microsomia: A personal perspective. *J Oral Maxillofac Surg* 56(5):639–650, 1998.
211. Posnick JC: Hemifacial microsomia: Evaluation and treatment. In Posnick JC, editor: *Craniofacial and Maxillofacial Surgery in Children and Young Adults*, Vol 20, Philadelphia, 2000, WB Saunders Co, pp 419–445.
212. Posnick JC, Fantuzzo J, Orchin J: Deliberate operative rotation of the maxillo-mandibular complex to alter the A-point to B-point relationship for enhanced facial esthetics. *J Oral Maxillofac Surg* 64:1687–1695, 2006.
213. Posnick JC, Goldstein JA, Waitzman A: Surgical correction of the Treacher Collins malar deficiency: Quantitative CT scan analysis of long-term results. *Plast Reconstr Surg* 92:12, 1993.
214. Poswillo DE: The pathogenesis of the first and second branchial arch syndrome. *Oral Surg* 35:302–328, 1973.
215. Poswillo DE: Otomandibular deformity: Pathogenesis as a guide to reconstruction. *J Maxillofac Surg* 2:64–72, 1974.
216. Poswillo DE: Hemorrhage in development of the face. *Birth Defects Orig Artic Ser* 11(7):61–81, 1975.
217. Poswillo DE: The embryological basis of craniofacial dysplasia. *Postgrad Med J* 53:517, 1977.
218. Poswillo DE: Biologic approach to temporomandibular reconstruction. In Whitaker LA, Randall P, editors: *Symposium on reconstruction of jaw deformity*, St. Louis, 1978, Mosby, p 139–145.
219. Preston CB, Losken HW, Evans WG: Restitution of facial form in a patient with hemifacial microsomia: A case report. *Angle Orthod* 55:197, 1985.
220. Pruzansky S: Not all dwarfed mandibles are alike. *Birth Defects* 1:120, 1969.
221. Rachmiel A, Aizeenbud D, Eleftheriou S, et al: Extraoral vs. intraoral distraction osteogenesis in the treatment of hemifacial microsomia. *Ann Plast Surg* 45(4):386–394, 2000.
222. Rachmiel A, Manor R, Peled M, Laufer D: Intraoral distraction osteogenesis of the mandible in hemifacial microsomia. *J Oral Maxillofac Surg* 59(7):728–733, 2001.
223. Rahbar R, Robson CD, Mulliken JB, et al: Craniofacial, temporal bone, and audiologic abnormalities in the spectrum of hemifacial microsomia. *Arch Otolaryngol Head Neck Surg* 127(3):265–271, 2001.
224. Raustia A, Pernu H, Pyhtinen J, Oikarinen K: Clinical and computed tomographic findings in costochondral grafts replacing the mandibular condyle. *J Oral Maxillofac Surg* 54:1393–1400, 1996.
225. Regev E, Jensen JN, McCarthy JG, et al: Removal of mandibular tooth follicles before distraction osteogenesis. *Plast Reconstr Surg* 113:1910, 2004.
226. Robinson M, Stoughton D: Surgical-orthodontic treatment of a case of hemifacial microsomia. *Am J Orthod* 57(3):287–292, 1970.
227. Rodgers SF, Eppley BL, Nelson CL, Sadove AM: Hemifacial microsomia: Assessment of classification system. *J Craniofac Surg* 2(3):114–126, 1991.
228. Rogers GF, Mulliken JB: Repair of transverse facial cleft in hemifacial microsomia: Long-term anthropometric evaluation of commissural symmetry. *Plast Reconstr Surg* 120(3):728–737, 2007.
229. Rollinck BR: Oculoauriculovertebral anomaly: Variability and causal heterogeneity. *Am J Med Genet* 4(Suppl):41, 1988.
230. Rollnick BR, Kaye CI: Hemifacial microsomia and variants: Pedigree data. *Am J Med Genet* 15:233, 1983.
231. Ross RB: Lateral facial dysplasia (first and second branchial arch syndrome, hemifacial microsomia). *Birth Defects* 11:51, 1975.
232. Rubio-Bueno P, Padron A, Villa E, Diaz-Gonzales FJ: Distraction osteogenesis of the ascending ramus for mandibular hypoplasia using extraoral or intraoral devices: A report of 8 cases. *J Oral Maxillofac Surg* 58:593, 2000.
233. Rune B, Sarnas KV, Selvik G, et al: Roentgen stereometry with the aid of metallic implants in hemifacial microsomia. *Am J Orthod* 84:231, 1983.
234. Rune B, Selvik G, Sarnas KV, et al: Growth in hemifacial microsomia studied with the aid of roentgen stereophotogrammetry and metallic implants. *Cleft Palate J* 18:128, 1981.
235. Samman N, Cheung LK, Tiderman H: Overgrowth of a costochondral graft in an adult male. *Int J Oral Maxillofac Surg* 24:333–335, 1995.
236. Sandhu S, Kaur T: Hemifacial microsomia: A case report and review. *Ind J Dent Res* 13(2):82–86, 2002.
237. Santoh K, Suzuki H, Uemura T, Hosaka Y: Maxillo-mandibular distraction osteogenesis for hemifacial microsomia in children. *Ann Plast Surg* 49(6):572–578; discussion 578–579, 2002.
238. Sarnas KV, Pancherz H, Rune B, et al: Hemifacial microsomia treated with the Herbst appliance: Report of a case analyzed by means of roentgen stereometry and metallic implants. *Am J Orthod* 82:68, 1982.
239. Sarnas KV, Rune B, Aberg M: Maxillary and mandibular displacement in hemifacial microsomia: A longitudinal, roentgen stereometric study of 21 patients with the aid of metallic implants. *Cleft Palate Craniofac J* 41(3):290–303, 2004.
240. Sarnas KV, Rune B, Selvik G, Jacobsson S: Hemifacial microsomia. *Plast Reconstr Surg* 75(6):928–929, 1985.
241. Scolozzi P, Herzog G, Jaques B: Simultaneous maxillo-mandibular distraction osteogenesis in hemifacial microsomia: A new technique using two distractors. *Plast Reconstr Surg* 117(5):1530–1541; discussion 1542, 2006.
242. Shetye PR, Grayson BH, Mackool RJ, McCarthy JG: Long-term stability and growth following unilateral mandibular distraction in growing children with craniofacial microsomia. *Plast Reconstr Surg* 118:985, 2006.
243. Sidiropoulou S, Antoniades K, Kolokithas G: Orthopedically induced condylar growth in a patient with hemifacial microsomia. *Cleft Palate Craniofac J* 40(6):645–650, 2003.
244. Siebert JW, Goesel A, Longaker MT: Microsurgical correction of facial asymmetry in 60 consecutive cases. *Plast Reconstr Surg* 97:354, 1996.
245. Siebert JW, Longaker MT: Microsurgical correction of facial asymmetry in hemifacial microsomia: Operative techniques. *Plast Reconstr Surg* 1:93, 1994.
246. Silla Freitas R, Tolazzi ARD, Alonso N, Cruz GA: Evaluation of molar teeth and buds in patients submitted to mandibular distraction: Long-term results. *Plast Reconstr Surg* 121:1335–1342, 2010.
247. Singer SL, Haan E, Slee J, Goldblatt J: Familial hemifacial microsomia due to autosomal dominant inheritance: Case reports. *Aust Dent J* 39(5):287–291, 1994.
248. Singh A, Malhotra G, Singh GP, et al: Goldenhar syndrome: A case report. *Acta Chir Plast* 36:111, 1994.
249. Singh DJ, Bartlett SP: Congenital mandibular hypoplasia: Analysis and classification. *J Craniofac Surg* 16:291, 2005.
250. Snyder CC, Levine GA, Swanson HM, et al: Mandibular lengthening by gradual distraction. *Plast Reconstr Surg* 51:506, 1973.
251. Steinbacher D, Gougoutas A, Bartlett SP: An analysis of mandibular volume in hemifacial microsomia. *Plast Reconstr Surg* 127:2407, 2011.

252. Steinberg B, Fattahi T: Distraction osteogenesis in management of pediatric airway: Evidence to support its use. *J Oral Maxillofac Surg* 63:1206, 2005.

252A. Stelnicki EJ, Hollier L, Lee C, Lin WY, Grayson B, McCarthy JG: Distraction osteogenesis of costochondral bone grafts in the mandible. *Plast Reconstr Surg* 109: 925–933, 2002; discussion 934–935.

253. Stringer DE, Steed DL, Johnson RP, et al: Correction of hemifacial microsomia. *J Oral Surg* 39:35, 1981.

254. Stucki-Mccormick SU: Reconstruction of mandibular condyle using transport distraction osteogenesis. *J Craniofac Surg* 8:48, 1997.

255. Sulik KK: Craniofacial defects from genetic and teratogen-induced deficiencies in presomite embryos. *Birth Defects* 20:79, 1984.

256. Swanson LT, Murray JE: Mandibular reconstruction in hemifacial microsomia. In Tanzer RC, Edgerton MT, editors: *Symposium on reconstruction of the auricle*, St. Louis, 1974, CV Mosby, p 270.

257. Takashima M, Kitai N, Mori Y, et al: Mandibular distraction osteogenesis using an intraoral device and bite plate for a case of hemifacial microsomia. *Cleft Palate Craniofac J* 40(4):437–445, 2003.

258. Takushima A, Harii K, Asato H, Yamada A: Neurovascular free-muscle transfer to treat facial paralysis associated with hemifacial microsomia. *Plast Reconstr Surg* 109(4):1219–1227, 2002.

259. Tanna N, Wan DC, Kawamoto HK, Bradley JP: Craniofacial microsomia soft-tissue reconstruction comparison: Inframammary extended circumflex scapular flap versus serial fat grafting. *Plast Reconstr Surg* 127: 802, 2011

260. Tanzer RC: Total reconstruction of the external ear. *Plast Reconstr Surg* 23:1, 1959.

261. Tanzer RC: Total reconstruction of the auricle: The evolution of a plan of treatment. *Plast Reconstr Surg* 47:523, 1971.

262. Tharanon W, Sinn DP: Mandibular distraction osteogenesis with multidirectional extraoral distraction device in hemifacial microsomia patients: Three-dimensional treatment planning, prediction tracings, and case outcomes. *J Craniofac Surg* 10(3):202–213, 1999.

263. Thomas P: Goldenhar syndrome and hemifacial microsomia: Observations on three patients. *Eur J Pediatr* 133(3):287–292, 1980.

264. Tiner BD, Quaroni AL: Facial asymmetries in hemifacial microsomia, Goldenhar syndrome, and Treacher Collins syndrome. *Atlas Oral Maxillofac Surg Clin North Am* 4(1):37–52, 1996.

265. Trahar M, Sheffield R, Kawamoto H, et al: Cephalometric evaluation of the craniofacial complex in patients treated with an intraoral distraction osteogenesis device: A preliminary report. *Am J Orthod Dentofacial Orthop* 124:639, 2003.

266. Troulis MJ, Everett P, Seldin EB, et al: Development of a three-dimensional treatment planning system based on computed tomographic data. *Int J Oral Maxillofac Surg* 31(4):349–357, 2002.

267. Tsirikos AL, McMaster MJ: Goldenhar-associated conditions (hemifacial microsomia) and congenital deformities of the spine. *Spine* 31(13):E400–E407, 2006.

268. Tweed AE, Manktelow RT, Zuker RM: Facial contour reconstruction with free flaps. *Ann Plast Surg* 12:313, 1984.

269. Upton J, Mulliken JB, Hicks PD, et al: Restoration of facial contour using free vascularized omental transfer. *Plast Reconstr Surg* 66:500, 1980.

270. Vargervik K: Sequence and timing of treatment phases in hemifacial microsomias. In Harvold EP, editor: *Treatment of hemifacial microsomia*, New York, 1983, Alan R. Liss, pp 133–137.

271. Vargervik K: Treatment of hemifacial microsomia in patients without a functioning temporomandibular articulation. In Harvold EP, editor: *Treatment of hemifacial microsomia*, New York, 1983, Alan R. Liss, pp 207–242.

272. Vargervik K: Discussion of relationship between bone and muscles of mastication in hemifacial microsomia. *Plast Reconstr Surg* 99:990, 1997.

273. Vargervik K: Mandibular malformations: Growth characteristics and management in hemifacial microsomia and Nager syndrome. *Acta Odontol Scand* 56(6):331–338, 1998.

274. Vargervik K, Miller AJ: Neuromuscular patterns in hemifacial microsomia. *Am J Orthod* 86:33, 1984.

275. Vargervik K, Ousterhout DK, Farias M: Factors affecting long-term results in hemifacial microsomia. *Cleft Palate J* 23(Suppl I):53, 1986.

276. Vento AR, LaBrie RA, Mulliken JB: The OMENS classification of hemifacial microsomia. *Cleft Palate Craniofac J* 28:68–76, 1991.

277. Vilkki SK, Hukki J, Nietosvaara Y, et al: Microvascular temporomandibular joint and mandibular ramus reconstruction in hemifacial microsomia. *J Craniofac Surg* 13(6):809–815, 2002.

278. Vu HL, Panchal J, Levine N: Combined simultaneous distraction osteogenesis of the maxilla and mandible using a single distraction device in hemifacial microsomia. *J Craniofac Surg* 12(3):253–258, 2001.

279. Wan D, Taub P, Allam KA, et al: Distraction osteogenesis of costocartilaginous rib grafts and treatment algorithm for severely hypoplastic mandibles. *Plast Reconstr Surg* 127:2005–2013, 2011.

280. Wan J, Meara JG, Kovanlikaya A, et al: Clinical, radiological, and audiological relationships in hemifacial microsomia. *Ann Plast Surg* 51(2):161–166, 2003.

281. Werler MM, Sheehan JE, Hayes C, et al: Vasoactive exposures, vascular events, and hemifacial microsomia. *Birth Defects Res A Clin Mol Teratol* 70(6):389–395, 2004.

282. Werler MM, Sheehan JE, Hayes C, et al: Demographic and reproductive factors associated with hemifacial microsomia. *Cleft Palate Craniofac J* 41(5):494–450, 2004.

283. Wiens JL, Forte RA, Weins JP: The use of distraction osteogenesis to treat hemifacial microsomia: A clinical report. *J Prosthet Dent* 89(1):11–14, 2003.

284. Wijbenga JG, Verlinden CR, Jansma J, et al: Long lasting neurosensory disturbance following advancement of the retrognathic mandible: Distraction osteogenesis versus bilateral sagittal split osteotomy. *Int J Oral Maxillofac Surg* 38:719, 2009.

285. Williamson EH: Mandibular growth following a costochondral transplant in the treatment of hemifacial microsomia. *Facial Orthop Temporomandibular Arthrol* 5:3, 1988.

286. Yasui N, Kojimoto H, Shimizu H, Shimomura Y: The effect of distraction upon bone, muscle, and periosteum. *Orthop Clin North Am* 22:563, 1991.

287. Zhou YQ, Mu XZ, Ren W, Yu ZY: Surgical treatment of hemifacial microsomia [Chinese]. *Zhonghua Wai Ke Za Zhi* 44(11):754–756, 2006.

29

Binder Syndrome: Evaluation and Treatment

JEFFREY C. POSNICK, DMD, MD

- Current Approach to Reconstruction
- Skeletal Stability after Orthognathic Reconstruction
- Controversies and Unresolved Issues
- Conclusions

In 1939, Noyes first described a patient whose face was characterized by a flat nasal tip and a retruded maxillary–nasal base.[32] He did not recognize this as an entity unique from other known forms of maxillary retrusion. It was not until 1962 that von Binder came to recognize the specific entity of nasomaxillary hypoplasia that is now called *Binder syndrome.*[53] He described physical findings of nasomaxillary hypoplasia, a convex lip, a vertical (short) nose, a flat frontonasal angle, an absent anterior nasal spine, limited nasal mucosa, and hypoplastic frontal sinuses. von Binder postulated that the hypoplasia was the result of a disturbance of the prosencephalic induction center at a critical phase during development. In 1989, Sheffield and colleagues reviewed 103 cases of chondrodysplasia punctata (CDP) seen in Melbourne, Australia, over a 20-year period.[48] They concluded that Binder syndrome should be classified as a mild form of CDP. In 1991, Sheffield and colleagues pointed out that most patients with Binder syndrome seek medical attention during adolescence.[47] By this age, the confirmatory diagnostic radiologic features of CDP have disappeared, so the diagnosis of CDP is often not considered. Older patients may show terminal phalangeal hypoplasia of the hand and variable anomalies of the vertebrae (i.e., vertebral clefting). Associated malformations of the cervical spine primarily affect the atlas and the axis without known clinical sequelae.[35,43] Familial recurrence has been reported, and inheritance may occur as an autosomal recessive trait with incomplete penetrance.[34] The syndrome may also be of a threshold character with a genetically multifactorial background.[5,14,45]

The physical findings of Binder syndrome result from hypoplasia of the anterior nasal floor (fossa praenasalis), and the anterior maxilla including the pyriform rim region.[26,33] When viewing the nose–upper lip complex from the worm's-eye perspective, typical variations from normal are described as a retracted columella–lip junction, a lack of normal triangular flare at the nasal base, a perpendicular alar–cheek junction, a convex flat nasal tip with a wide and shallow philtrum, crescent-shaped nostrils without a distinct sill, and a stretched and shallow cupid's bow.[27,41] Striking profile characteristics of the nose include vertical shortening of the columella, a lack of tip projection, perialar flattening, and an acute nasolabial angle.[11,28] The dentition and occlusion in an untreated individual will typically demonstrate the proclination of the maxillary incisors, "peg" laterals, and a canine Angle class III negative overjet tendency.[21]

Anthropometric explanations for these findings were first suggested by Zuckerkandl in 1882, when he described an anomaly in the anterior nasal floor in which the normal crest that separates the nasal floor from the anterior surface of the maxilla was absent.[56] Instead, a small pit—the fossa praenasalis—constituted the pyriform aperture.[18,24] Other investigators have pointed out that the premaxilla of normal Caucasian individuals is incorporated into the upper arch.[2,3] This results in a prominence or projection of the base of the nose. By contrast, when the premaxilla (i.e., the primary palate) is not incorporated into the arch (i.e., in higher primates, certain racial groups, persons with Binder syndrome, and individuals with bilateral clefted alveolar ridges and lips), there will be flattening of the premaxillary region and of the base of the nose.[6] This is more commonly seen among African and Asian people.[10]

In an attempt to reconstruct the skeletal deformities associated with Binder syndrome, clinicians have suggested a spectrum of procedures, including Le Fort I osteotomy, Le Fort II osteotomy, Le Fort III osteotomy, and a combination of Le Fort I and II osteotomies.[4,8,12,17,19,20,22,23,31,36,39,42,44,49,51,55] Augmentation of the infraorbital rims, the pyriform rims (paranasal), and the anterior maxilla with the use of a spectrum of materials have all been tried.[7,25,40,50] Suggested nasal reconstructive

options have included autogenous, homogenous, and allogenic bone and cartilage grafts that extend up the columella (i.e., from the base of the maxilla to the nasal tip) and over the nasal dorsum (i.e., from the radix to the tip).[1,9,13,15,19,29,30,38,39,46,52] Suggested soft-tissue procedures include septal cartilage and mucosal flaps, upper lip to nasal skin flaps to lengthen the columella, and a variety of subcutaneous augmentation procedures and fillers.[37,54] Compensating orthodontic or dental restorative work may also be undertaken.

With Binder syndrome, a degree of hypoplasia of the premaxilla and of the quadrangular cartilage of the nasal septum is a consistent finding. This author agrees with Holmstrom and colleagues that, with this anomaly, there is a local shortage of bone in the premaxillary region and an absence of cartilage in the anterior septum.[18-20] However, as Tessier pointed out, there is no significant shortage of soft tissue available.[50-51] Furthermore, modification of the uninvolved orbits, zygomas, and upper nasal dorsum (i.e., the nasal bones) is rarely indicated or advantageous. The observed facial features in an individual with Binder syndrome are dependent on the degree of hypoplasia of the anterior nasal floor (fossa paranasalis) present at the time of birth. The deformities are believed to be non-progressive with age.

Current Approach to Reconstruction

The approach to the correction of the Binder syndrome deformity is to plan for a staged reconstruction to coincide with facial and dental growth patterns and psychosocial needs.[16,39] An analysis of each patient's morphology is followed by a review of the reconstructive options with the patient and family. The most gratifying long-term functions (i.e., occlusion and breathing) and facial aesthetics are generally achieved when carrying out the reconstruction after the completion of growth and before the individual's graduation from high school (Fig. 29-1 through 29-5). Orthodontic treatment should be coordinated with consideration of orthognathic correction and premaxillary augmentation to be followed by definitive nasal reconstruction. The ideal orthodontic treatment often includes maxillary first bicuspid extractions with retraction and alignment of the anterior teeth to produce ideal incisor inclination within solid basal bone. Unfortunately, many individuals with Binder syndrome will have already been treated with an orthodontic camouflage approach. During the process, the maxillary incisors are tipped facially, and the mandibular incisors are often retroclined. This will typically achieve successful neutralization of the occlusion (i.e., the correction of overjet), but it will leave the patient looking as if he or she has a maxillary deficiency. Attempts at surgical augmentation after orthodontic camouflage are generally suboptimal. When indicated, definitive orthognathic reconstruction is planned to idealize the vertical, transverse, and horizontal midface proportions. A Le Fort I osteotomy (horizontal advancement and a variable degree of vertical lengthening) is frequently combined with an osseous genioplasty (vertical shortening and horizontal advancement). Sagittal split ramus osteotomies are often needed to avoid the limitations inherent to mandibular autorotation. Further reconstruction involves the application of a crafted bone graft to the deficient premaxillary and pyriform rim regions. This is best accomplished simultaneously with the orthognathic procedures.

Text continued on p. 1177

• **Figure 29-1** A 17-year-old boy, who was born with Binder syndrome was referred by his orthodontist for surgical evaluation. When he was 7 years old he underwent nasal augmentation with Silastic implants with another surgeon. Orthodontic treatment including maxillary first-bicuspid extractions and orthognathic and nasal surgery was chosen. The patient's procedures included a Le Fort I osteotomy (horizontal advancement); the removal of nasal implants; osseous genioplasty (horizontal advancement); and nasal reconstruction (corticocancellous iliac graft). **A,** Facial views at 10 years old with Silastic implants in place.

• **Figure 29-1, cont'd** **B,** Frontal views with smile before and after reconstruction. **C** and **D,** Profile views before and after reconstruction.

Continued

• **Figure 29-1, cont'd E,** Worm's-eye views before and after reconstruction.

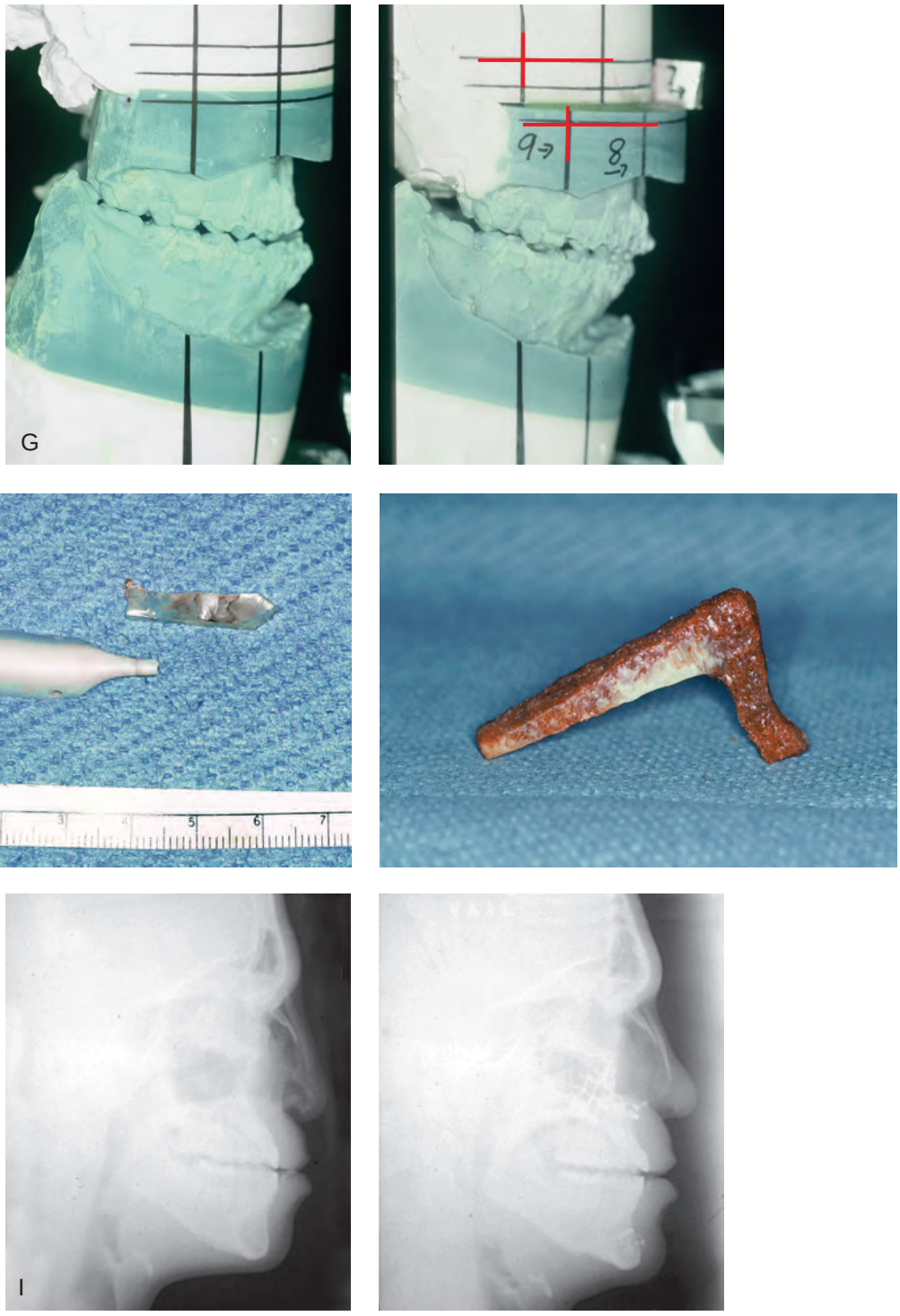
Prior to treatment

Prior to surgery after maxillary bicuspid extractions

After treatment

• **Figure 29-1, cont'd F,** Occlusal views before and after reconstruction.

Continued



• **Figure 29-1, cont'd** **G,** Articulated dental casts that indicate analytic model planning. **H,** Intraoperative views of removed Silastic nasal implants. The crafted iliac corticocancellous bone graft before inset is also shown. **I,** Lateral cephalometric radiographs before treatment and after reconstruction. *A (right), B, C, F, H, I, From Posnick JC, Tompson B: Binder syndrome: Staging of reconstruction and skeletal stability and relapse patters after Le Fort I osteotomy using miniplate fixation,* Plast Reconstr Surg *99:967, 1997.*

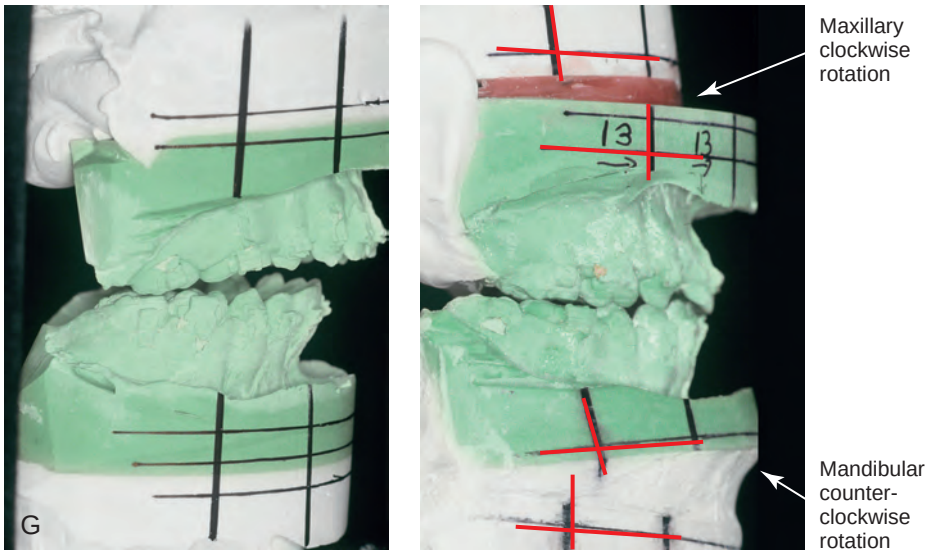
©Elsevier Inc.
Orthognathic Surgery; J.C. Posnick

• **Figure 29-2** A 16-year-old girl who was born with Binder syndrome was referred by her orthodontist for surgical evaluation. She agreed to orthodontic treatment that included maxillary first bicuspid extractions and orthognathic and nasal surgery. The patient's procedures included Le Fort I osteotomy (horizontal advancement); bilateral sagittal split ramus osteotomies; osseous genioplasty; and nasal reconstruction (autogenous rib graft). **A,** Frontal views in repose before and after reconstruction. **B,** Frontal views with smile before and after reconstruction. *Continued*

• **Figure 29-2, cont'd C,** Oblique facial views before and after reconstruction. **D,** Profile views before and after reconstruction.

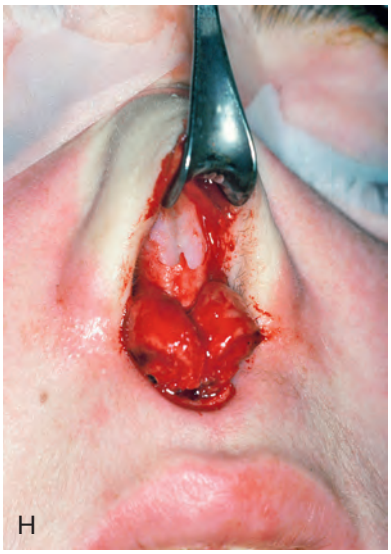
Prior to surgery after maxillary bicuspid extractions

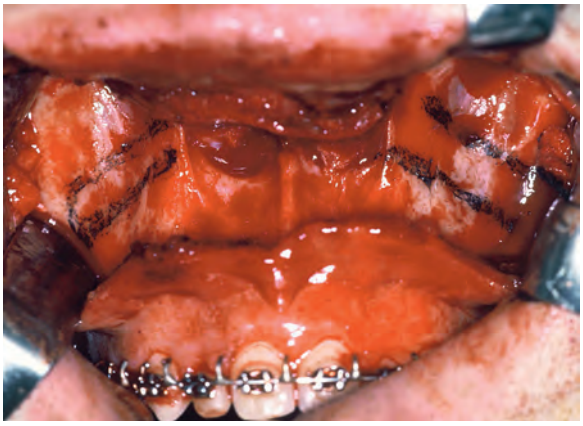
After treatment

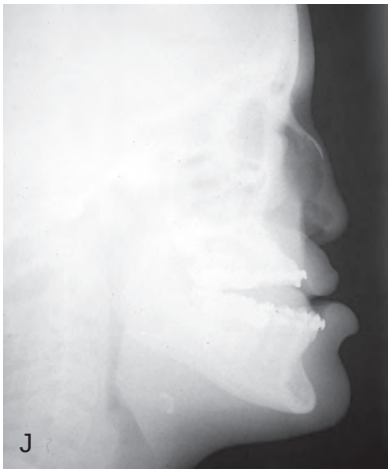


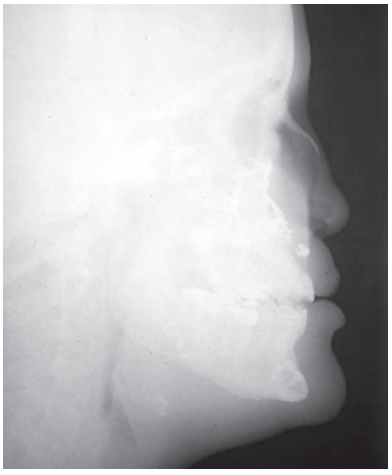
• **Figure 29-2, cont'd** **E,** Worm's-eye views before and after reconstruction. **F,** Occlusal views before and after reconstruction. **G,** Articulated dental casts that indicate analytic model planning.

Continued









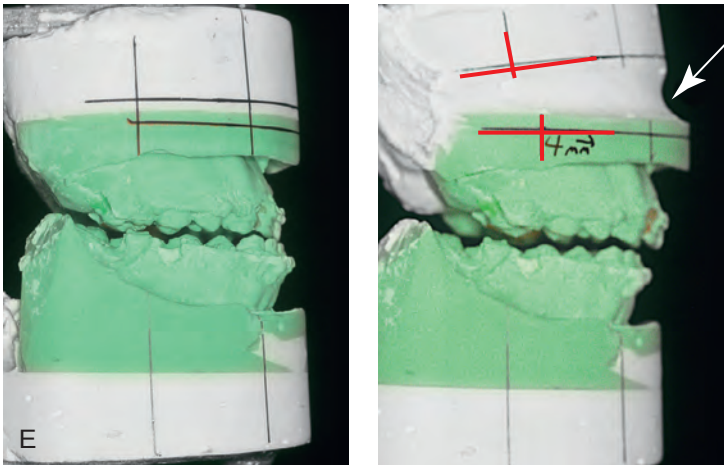
• **Figure 29-2, cont'd H,** Intraoperative views of open rhinoplasty exposure. The rib bone (dorsal strut) and rib cartilage (caudal strut) grafts are shown before inset. **I,** Computed tomography scan and intraoperative views that confirm premaxillary hypoplasia consistent with Binder syndrome. **J,** Lateral cephalometric radiographs before and after reconstruction. *B, F (top center and bottom center), H, From Posnick JC, Tompson B: Binder syndrome: Staging of reconstruction and skeletal stability and relapse patters after Le Fort I osteotomy using miniplate fixation,* Plast Reconstr Surg *99:965, 1997.*

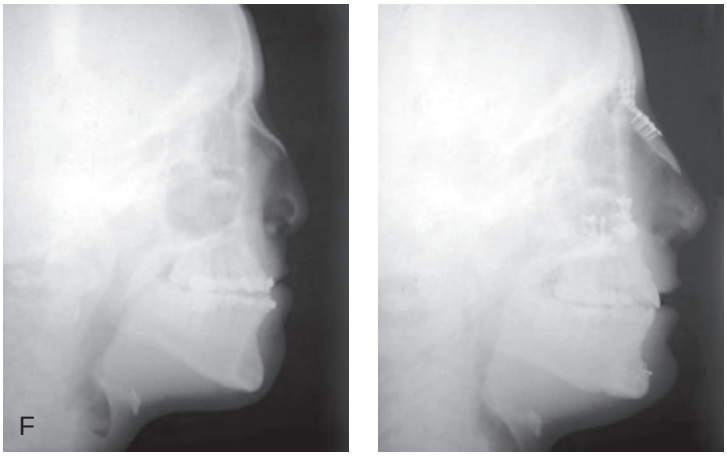
• **Figure 29-3** A 16-year-old girl who was born with Binder syndrome was referred by her orthodontist for surgical evaluation. She agreed to orthodontic treatment that included maxillary first bicuspid extractions and orthognathic surgery. The patient's procedures included Le Fort I osteotomy (horizontal advancement); osseous genioplasty (horizontal advancement); and nasal reconstruction. The nasal reconstruction (cranial graft) was complicated by pressure necrosis of the nasal tip skin, which necessitated the removal of the distal aspect of the graft. Six months later, through an open rhinoplasty approach, autogenous rib cartilage (a caudal strut) was used to revise the nasal tip. **A,** Frontal views in repose before and after reconstruction. **B,** Profile views before and after reconstruction. **C,** Worm's-eye views before and after reconstruction.

Continued

Prior to surgery after maxillary bicuspid extractions

After treatment



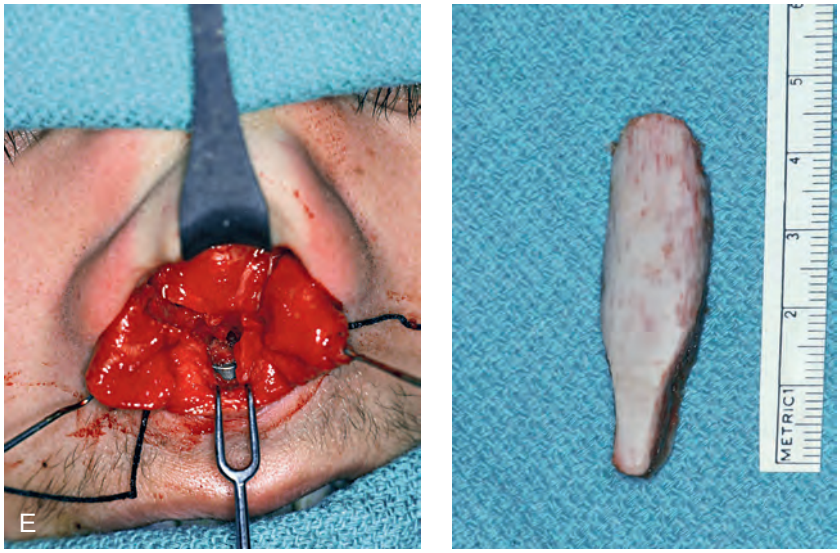


• **Figure 29-3, cont'd D,** Occlusal views during orthodontics and after reconstruction. **E,** Articulated dental casts that indicate analytic model planning. **F,** Lateral cephalometric radiographs before and after reconstruction.

• **Figure 29-4** A 17-year-old boy who was born with Binder syndrome was referred for surgical evaluation. He agreed to orthodontic treatment that included maxillary first bicuspid extractions and orthognathic and nasal surgery. The patient's procedures included Le Fort I osteotomy (horizontal advancement) and nasal reconstruction (autogenous rib graft). The nasal reconstruction was carried out through an open (columella splitting) technique. The skin of the columella was stretched but not directly lengthened. Autogenous rib bone (dorsal strut) and rib cartilage (caudal strut) were harvested, crafted, and then inset and secured in place. **A,** Frontal views in repose before and after reconstruction. **B,** Frontal views with smile before and after reconstruction.

Continued

• **Figure 29-4, cont'd C,** Close-up profile views before and after reconstruction. **D,** Worm's-eye views before and after reconstruction.

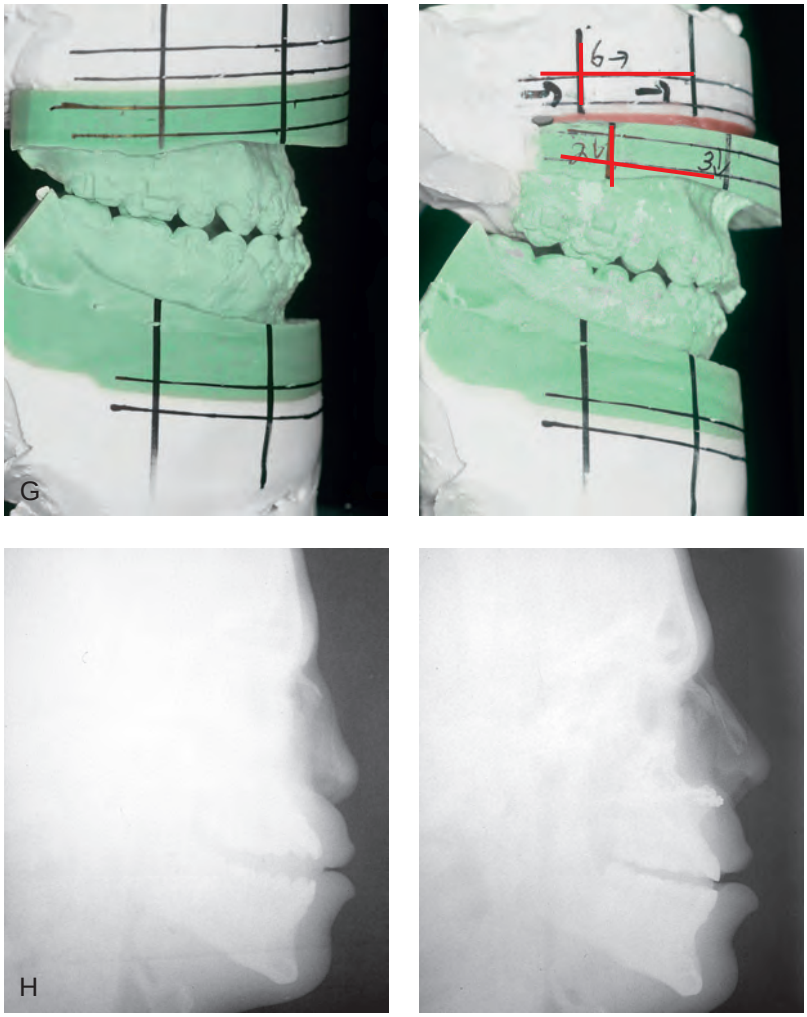


Prior to treatment

After treatment with maxillary bicuspid extractions

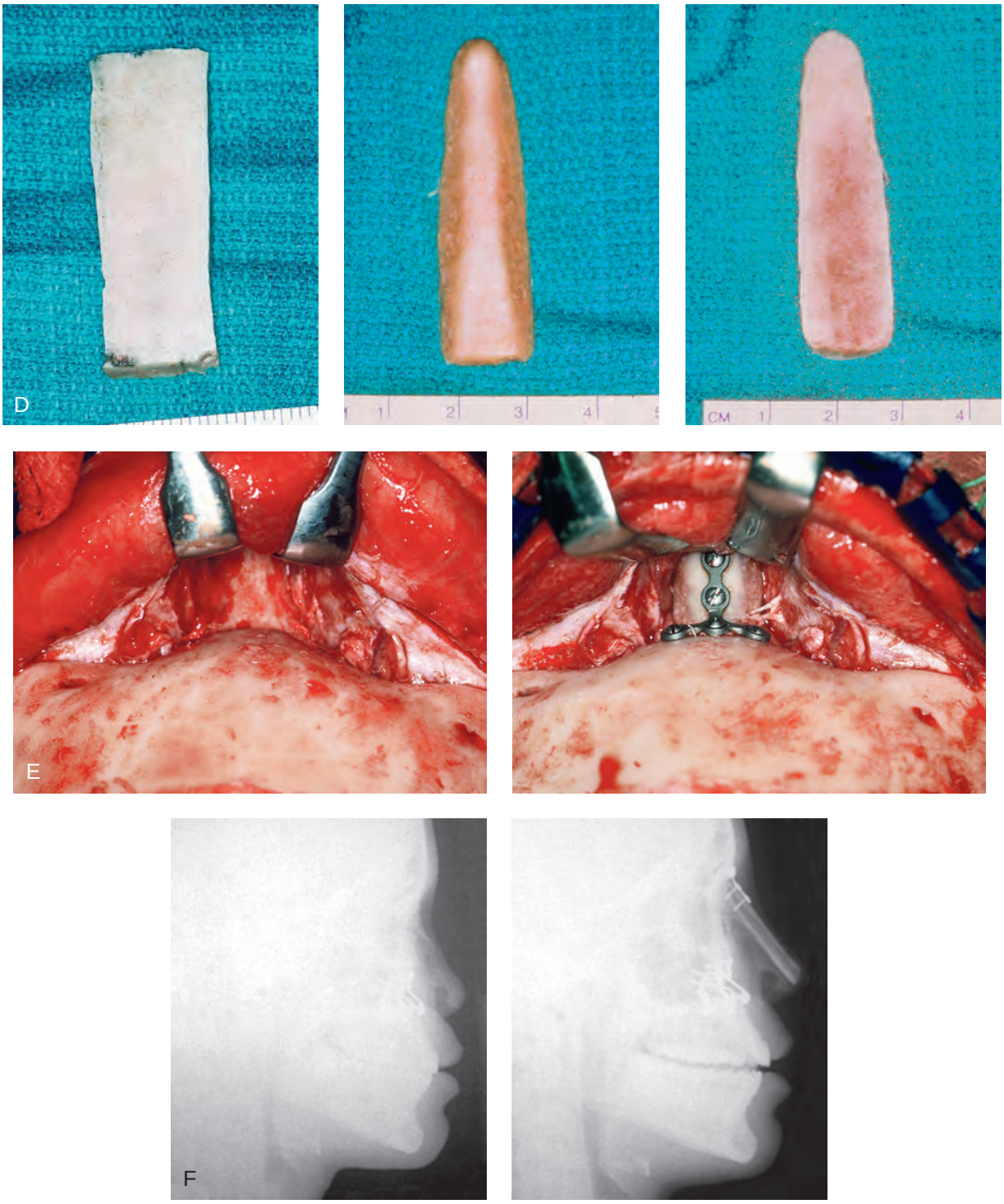
• **Figure 29-4, cont'd E,** Intraoperative views of open rhinoplasty exposure. A Kirschner wire has been inserted into the base of the maxilla. The Kirschner wire extends out of bone. The rib cartilage (caudal strut) will pierce the Kirschner wire and extend to the nasal tip. The crafted rib bone (dorsal strut) is shown before inset. It will join to the caudal graft to form the nasal tip. **F,** Occlusal views before and after reconstruction.

Continued



• **Figure 29-4, cont'd G,** Articulated dental casts that indicate analytic after model planning. **H,** Lateral cephalometric radiographs before and after reconstruction.

• **Figure 29-5** A 16-year-old girl who was born with Binder syndrome underwent Le Fort I osteotomy and septal cartilage graft to the dorsum of the nose with another surgeon. A residual nasal deformity remained. She was referred to this surgeon and underwent nasal reconstruction. Through a coronal scalp incision, a full-thickness cranial bone graft was harvested, crafted, and stabilized in place. The graft extended from the radix to the nasal tip. The lower lateral cartilages were then sutured over the top of the graft to form the new nasal tip. **A,** Close-up profile views before and after nasal reconstruction. **B,** Close-up frontal views before and after nasal reconstruction. **C,** Close-up worm's-eye views before and after nasal reconstruction. *Continued*



• **Figure 29-5, cont'd D,** Harvested full-thickness cranial bone before and after crafting for nasal reconstruction. **E,** Intraoperative close-up views of the nasofrontal process before and after the placement and stabilization of the cranial bone graft. **F,** Lateral cephalometric views before and after nasal reconstruction. *D, From Posnick JC, Seagle MB, Armstrong D: Nasal reconstruction with full-thickness cranial bone grafts and rigid internal skeletal fixation through a coronal incision,* Plast Reconstr Surg *86:894, 1990.*

Ideally, the nasal reconstruction is carried out after the orthognathic and anterior maxillary augmentation procedures (see Fig. 1-5).[38,39] This is accomplished by reconstructing the cartilaginous nasal deficiency. Stretching of the soft-tissue envelope over the reconstructed underlying nasal cartilage framework is accomplished at wound closure. When first-stage early nasal augmentation was carried out during childhood and before orthognathic correction, some profile improvements maybe achieved, but a relief of the stigma of Binder syndrome is not likely. Early nasal augmentation also imposes limitations on the child's physical activity to prevent graft fracture or displacement. These restrictions on the daily activities of childhood are likely to be more psychosocially traumatic than the original nasal deformity. For effective reconstruction, all aspects of the deformity should be addressed with a coordinated long-term approach in mind.

Skeletal Stability after Orthognathic Reconstruction

Few authors have looked at the early or late skeletal stability achieved when osteotomies are carried out to correct the Binder syndrome midface deficiency. In a previously published study, Posnick and colleagues prospectively assessed initial and long-term skeletal stability after orthognathic reconstruction including a Le Fort I osteotomy followed by nasal reconstruction in a consecutive series of skeletally mature Caucasian individuals born with Binder syndrome.[39]

Patients and Methods

The medical and cephalometric records and late posttreatment (surgical and orthodontic) clinical examination of a consecutive series of patients with Binder syndrome (n = 7) who underwent Le Fort I osteotomy by a single surgeon (Posnick) during a 6-year period were reviewed. The patients' ages at operation ranged from 16 to 20 years (mean, 17 years). For all study patients, the prospective protocol required obtaining a lateral cephalogram preoperatively, immediately (i.e., 3 to 7 days) after surgery, 6 to 8 weeks after surgery, and 1 year after surgery. At the time of final review, all patients had obtained their interval cephalograms and were available for a final clinical evaluation.

All patients underwent orthodontic treatment, orthognathic surgery, and nasal reconstruction. During the initial evaluation, all study patients were judged to have inadequate alveolar bone to retain the full complement of maxillary teeth and to maintain long-term periodontal health. Extraction of maxillary first bicuspids was carried out to remove dental compensations and to establish normal dentoalveolar arch form and incisor inclination. All patients underwent a standard one-piece Le Fort I osteotomy extended through the zygomatic buttress (below the malar eminence) and anteriorly through the hypoplastic pyriform aperture. All grafts to the upper jaw (five of seven patients) were autogenous corticocancellous iliac bone. After the titanium plates and screws were in place to stabilize the osteotomy, corticocancellous bone grafts were crafted to reconstruct the area of hypoplasia of the premaxilla. The grafts were fixed with additional plates and screws. Simultaneous sagittal split ramus mandibular osteotomies were carried out in three of seven patients. The majority of patients (six of seven patients) also underwent osseous genioplasty with horizontal advancement and varying degrees of vertical shortening. Six of seven patients underwent autogenous grafting of the nose (costochondral grafts in four patients, a cranial graft in one patient, and an iliac graft in one patient). One patient underwent a coronal scalp incision, the others (five of six patients) underwent an open rhinoplasty (i.e., columella skin-splitting incision) approach. Only one of the seven patients underwent simultaneous orthognathic surgery and nasal reconstruction.

Results

The serial cephalometric radiographs for each patient were analyzed, and the preoperative and immediate postoperative tracings were superimposed. The end result was a Cartesian coordinate system that illustrated the horizontal and vertical directional changes of the maxilla at intervals after surgery. In addition, the incisor overjet and overbite measurements from the 1-year postoperative cephalogram were documented and compared with the measurements taken at the final clinical assessment. All patients had a complete set of longitudinal clinical and cephalometric records, and all were available for late postoperative clinical reassessment at the close of the study. Clinical follow-up ranged from 1.5 to 5.5 years (mean, 3 years) at the completion of the study.

Perioperative morbidity was unremarkable when patients were reviewed for cardiopulmonary compromise, maxillofacial infection, hemorrhage, aseptic necrosis, loss of teeth, and need for root canal therapy. All patients maintained a positive overjet and overbite at the incisors as documented on the 1-year postoperative clinical and cephalometric examinations. The mean effective maxillary advancement achieved was 6.0 mm, with 5.9 mm maintained 1 year later. The mean anterior vertical change of the maxilla was 4.2 mm immediately after the operation and 3.1 mm after 1 year. The mean posterior vertical change of the maxilla was 2.8 mm immediately after the operation and 2.2 mm after 1 year. In review, the fixed and grafted Le Fort I osteotomies were able to maintain the horizontal advancement. Although a degree of anterior vertical relapse occurred, the long-term maintenance of satisfactory occlusion with a positive overjet and overbite was documented in all patients.

Controversies and Unresolved Issues

The precise genetic cause of Binder syndrome has not yet been confirmed. Gorlin and colleagues suggested that maxillonasal dysplasia was a non-specific abnormality

of the nasomaxillary complex.[14] Over time, it has become apparent that many patients with Binder syndrome ultimately have a form of chondrodysplasia punctate, although genetic heterogeneity exists. Children with the X-linked recessive for of chondrodysplasia punctate have molecular alterations in the arylsulfatase E gene. Because of the X-linked inheritance pattern, females with this form of chondrodysplasia punctate tend to be much more mildly affected than males. Molecular testing for alterations in arylsulfatase E is clinically available and should be tested for.

In retrospect, the features defined by von Binder were not altogether typical of those found in most patients who are currently categorized as having Binder syndrome. It is also known that frequent ethnic characteristics of both Asian and African individuals are a sloped backward and vertically short anterior maxilla, pyriform rims, and a deficient dorsum of the nose. This results in deficiency of alveolar bone to house the maxillary incisors. The anterior nasal spine is hypoplastic, but it is often present as a small ridge. A lack of height of the dorsum of the nose (i.e., bone and cartilage) with increased width is a frequent ethnic characteristic of Asian and African individuals but not a finding among patients with Binder syndrome. We believe that many patients who are believed to have Binder syndrome more accurately represent ethnic variation. This may well encompass many of the Asian patients described and treated by Goh and Chen in their review of nasal reconstruction for Binder syndrome.[13]

Holmstrom and others support an orthognathic approach and state that, in a series of Caucasian patients with Binder syndrome (54% of the study group), a majority "suffered a class III malocclusion."[18-20] Although an edge-to-edge incisor relationship may be possible with orthodontics only, it is generally accomplished with excessive maxillary incisor proclination. This camouflage approach does not address the flat midface appearance or the long-term orthodontic retention needs, and it may lead to periodontal sequelae. To correct the maxillary incisor inclination, an alveolar space analysis will confirm the advantage of extracting maxillary first bicuspids in many of these patients. With molar anchorage, the orthodontist is able to retract the anterior teeth into the space created by the bicuspid extractions, and ideal incisor positioning is accomplished. This approach will further unmask the maxillary hypoplasia and allow for an effective horizontal advancement at the Le Fort I level. When this is combined with a premaxillary region crafted bone graft reconstruction, maximum improvement in the perialar and nasal base morphology is also accomplished. An orthodontic camouflage approach should only be carried out with full disclosure to the patient and his or her family.

The aesthetic advantage of a vertical reduction and an advancement osseous genioplasty in the majority of individuals with Binder syndrome likely reflects the longstanding mouth-breathing pattern and its effects on anterior mandibular growth. In three of the seven patients in the study by Posnick and colleagues, bilateral sagittal split osteotomies were also necessary to correct the secondary mandibular dysmorphology or to prevent the inherent shortcomings of mandibular autorotation.[39] In none of the patients was the mandible "set back" as a camouflage for maxillary deficiency.[39]

Binder syndrome is not an orbital, zygomatic, nasal bone, or cranial vault malformation. Over time, it has been found that alteration of these regions is counterproductive. A Le Fort III osteotomy would have the disadvantage of flattening the nasofrontal angle, producing enophthalmos, and resulting in distortions (step-offs) of the lateral orbital rims. A Le Fort II osteotomy would also flatten the nasofrontal angle, produce enophthalmos, and result in distortions (step-offs) along the infraorbital rims. In addition, neither procedure would directly address the nasal septal cartilaginous vault malformation nor address the deficient premaxillary region.

In my experience, corticocancellous iliac bone offers a preferred volume, quality, and osteogenic potential to reconstruct the anterior alveolus–floor of the nose–anterior nasal spine region. Full-thickness cranial bone may lack the necessary volume (thickness) for premaxillary construction. The use of a rib graft is likely to be inadequate in both volume and quality. When the nasal reconstruction follows within 6 months after grafting and the new (bone-grafted) premaxilla is used as a platform, less resorption is expected. The use of artificial materials in this region circumvents the issue of resorption and donor-site morbidity, but the long-term concern of recipient-site infection and extrusion must be considered.

Effective facial rehabilitation for the patient with Binder syndrome must address the nasal deformity. Potential pitfalls to the nasal reconstruction merit discussion. The open rhinoplasty technique (i.e., the columella splitting incision) allows ideal access to the area of the nose that requires reconstruction. Whenever possible, the surgeon should avoid other external nasal incisions carried out either for exposure (e.g., coronal scalp flap, vertical incisions directly over the nose) or in an attempt to directly lengthen the columella (e.g., Cronin technique). In the Caucasian patient, if a graft is placed from the radix to the nasal tip, a technical error will be made unless the surgeon first reduces (i.e., burs down) the bony dorsum from the frontonasal suture to the caudal aspects of the nasal bones before the augmentation procedure. If this is not done, flattening of the nasofrontal angle occurs, and an unnatural facial appearance will result. Achieving a flexible (or at least "nonfirm") reconstructed nasal tip is preferred. A relatively soft nasal tip with enough strength for tip projection is best accomplished with the use of a rib cartilage graft. The lower lateral cartilages are sutured over the top of the graft. If a full-length dorsal bone graft is required (i.e., from the radix to the tip), then stable fixation is necessary; this may be accomplished in a number of ways (e.g., plates and screws, lag screws, Kirschner wires [K-wires]), depending on the clinical circumstances. Placing a protective external

nasal splint over the grafted region limits swelling and assists with immobilization and overall healing. If dorsal bone augmentation is carried out, union to the underlying nasal bones is essential to achieve long-term facial aesthetic and functional objectives.

Holmstrom reviewed the long-term results of nasal reconstruction in a series of patients with Binder syndrome and found overall good results with a variety of techniques.[18-20] A review of nasal reconstruction options is listed below. The option selected for a specific patient is dependent on the unique presenting dysmorphology, the potential risks, the patient-specific objectives, and the surgeon's comfort level with the particular technique.

1. A cranial graft placed through a coronal scalp incision offers ideal access to the nasofrontal region for contouring and graft stabilization (i.e., plates and screws), and it provides excellent bone volume and quality. The lower lateral cartilage is then sutured over the graft at the tip via a columella incision. This approach generally requires craniotomy and a full-thickness graft, depending on the volume of bone required. Unfortunately, it results in a firm nasal tip that has a tendency toward a degree of resorption and that is prone to fracture with trauma. This approach is preferred only for the occasional patient in whom significant reconstruction of the nasofrontal region and augmentation of the bony dorsum is required (see Fig. 29-5).
2. A costochondral (bone and cartilage) graft maybe placed through a columella splitting incision. This results in a relatively "non-firm" nasal tip with limited resorption or risk of warping over time. If the relatively thin and malleable rib graft is placed in a cantilever fashion (i.e., without a columella strut for stabilization), there is a tendency for non-union with the underlying nasal bones and dislocation or fracture after minimal trauma. If the graft extends from the radix, it may also flatten the nasofrontal angle in an unfavorable way. This approach is generally not a first choice (see Fig. 29-1).
3. If a costochondral graft is selected for dorsal reconstruction (as described previously), a separate columella (rib cartilage) strut graft maybe crafted and then secured with sutures to the dorsal costochondral graft at the new nasal tip. Temporary stabilization of the dorsal bone graft component may be accomplished with a transcutaneous K-wire. A second short buried K-wire is placed to stabilize the rib cartilage (caudal strut) at the base of the maxilla (see Fig. 29-2).
4. An anterior iliac (corticocancellous) graft offers another option, but this is only used when extensive bone volume reconstruction is required. It is placed as a crafted L-shaped strut that is stabilized at the base of the premaxilla with titanium plates and screws and over the mid dorsum with percutaneous K-wires. When iliac bone is used at the time of the Le Fort I osteotomy for augmentation of the premaxilla, additional graft may be harvested for simultaneous nasal reconstruction, as described previously. This surgeon has used this combined single-stage approach only in unique and specific circumstances, because very few deformities require upper dorsal reconstruction (see Fig. 29-1). This approach also raises the level of complexity of the procedure by involving the need for sophisticated intraoperative airway management.
5. An L-shaped rib cartilage graft reconstruction has become this author's preference whenever the Binder deformity allows. With this procedure, it is not necessary to harvest the L-shaped rib cartilage graft in one piece. Rather, two struts (dorsal and columella) are crafted separately and then inset and sutured together to form the new nasal tip. The columella strut graft extends from the base of the maxilla to the nasal tip. It is fixed in place at the base of the maxilla with a buried K-wire (no. 32 threaded) to prevent slippage or movement. The dorsal strut graft extends from the caudal edge of the nasal bones to the new nasal tip, where it is secured to the columella strut graft with the use of non-resorbable suture material. Warping of the cartilage graft can occur, but resorption is not a problem. A separate K-wire (no. 32 threaded) may be inserted through the spine of the dorsal graft before inset to limit the risk of warping. The lower lateral cartilages are sutured over the top of the grafts, and the end result is a flexible nasal tip. This rib cartilage reconstruction option is preferred whenever feasible (see Chapter 34).

For the individual with Binder syndrome, early reconstruction of the dysmorphic nose (i.e., between the ages of 5 and 12 years) to improve tip projection and to encourage self-esteem is technically possible, but the surgeon should proceed with caution. Disadvantages include the need for additional nasal procedures at the completion of growth and after definitive jaw reconstruction. The inherent perioperative risks and expense; the creation of scar tissue within the nasal soft-tissue envelope; and the need for the child to avoid normal school-aged physical activities to prevent graft dislodgement or fracture should also be considered (see Fig. 29-1, *A*).

Conclusions

The individual with Binder syndrome presents with hypoplasia of the anterior maxilla and the caudal aspect of the cartilaginous nose. No genetic marker yet exists for this condition, and diagnostic confusion with normal ethnic variations of premaxillary and nasal anatomy is frequent. A staged reconstructive approach is generally carried out during the teenage years, before the patient graduates from high school. In Caucasian patients, this often includes orthodontic treatment, orthognathic procedures, anterior

maxillary augmentation, and nasal reconstruction. The orthodontic treatment relieves dental compensation and often includes maxillary first bicuspid extractions. Orthognathic surgery includes a Le Fort I osteotomy to idealize any vertical, transverse, or horizontal facial disproportions with simultaneous augmentation of the deficient premaxillary region. An osseous genioplasty further improves profile aesthetics. Nasal reconstruction (often with autogenous rib cartilage) follows orthognathic surgery to project the nasal tip and to stretch the overlying soft-tissue envelope.

References

1. Ahn J, Honrado C, Horn C: Combined silicone and cartilage implants: Augmentation rhinoplasty in Asian patients. *Arch Facial Plast Surg* 6:120–123, 2004.
2. Ashley-Montagu MF: The premaxilla in the primates. *Q Rev Biol* 10:32, 1935.
3. Ashley-Montagu MF: The premaxilla in man. *J Am Dent Assoc* 23:2043, 1936.
4. Banks P, Tanner B: The mask rhinoplasty: A technique for the treatment of Binder syndrome and related disorders. *Plast Reconstr Surg* 92:1038, 1993.
5. Bütow KW, Jacobsohn PV, de Witt TW: Nasomaxillo-acrodysostosis. *S Afr Med J* 75:5, 1989.
6. Callender GW: The formation and early growth of the bones of the human face. IV. Philos. *Trans R Soc Lond Biol* 159:163, 1869.
7. Converse JM: Restoration of facial contour by bone grafts introduced through the oral cavity. *Plast Reconstr Surg* 6:295, 1950.
8. Converse JM, Horowitz SL, Valauri AJ, et al: The treatment of nasomaxillary hypoplasia: A new pyramidal naso-orbital maxillary osteotomy. *Plast Reconstr Surg* 45:527, 1970.
9. Draf W, Bockmuhl U, Hoffmann B: Nasal correction in maxillonasal dysplasia (Binder syndrome): A long-term follow-up study. *Br J Plast Surg* 56:199–204, 2003.
10. Dwight T: Fossa praenasalis. *Am J Med Sci* 103:156, 1892.
11. Freihofer HPM, Jr: The lip profile after correction of retromaxillism in cleft and non-cleft patients. *J Maxillofac Surg* 4:136, 1976.
12. Freihofer HPM, Jr: Changes in nasal profile after maxillary advancement in cleft and non-cleft patients. *J Maxillofac Surg* 5:20, 1977.
13. Goh RC, Chen YR: Surgical management of Binder's syndrome: Lessons learned. *Aesthetic Plast Surg* 34:722–730, 2010.
14. Gorlin RJ, Cohen MM, Jr, Levin LS: *Syndromes of the head and neck*, ed 3, New York, 1990, Oxford University Press, pp 813–814.
15. Gunter JP, Clark CP, Friedman RM: Internal stabilization of autogenous rib cartilage grafts in rhinoplasty: A barrier to cartilage warping. *Plast Reconstr Surg* 100:161–169, 1997.
16. Harper DC: Children's attitudes to physical differences among youth from Western and non-Western cultures. *Cleft Palate Craniofac J* 32:114, 1995.
17. Henderson D, Jackson IT: Nasomaxillary hypoplasia: The Le Fort II osteotomy. *Br J Oral Surg* 11:77, 1973.
18. Holmstrom H: Clinical and pathologic features of maxillonasal dysplasia (Binder syndrome): Significance of the prenasal fossa on etiology. *Plast Reconstr Surg* 78:559–567, 1986.
19. Holmstrom H: Surgical correction of the nose and midface in maxillofacial dysplasia (Binder syndrome). *Plast Reconstr Surg* 78:568, 1986.
20. Holmstrom H, Kahnberg KE: Surgical approach in severe cases of maxillonasal dysplasia (Binder syndrome). *Swed Dent J* 12:3, 1988.
21. Hopkin GB: Hypoplasia of the middle third of the face associated with congenital absence of the anterior nasal spine, depression of the nasal bones and Angle class III malocclusion. *Br J Plast Surg* 16:146, 1963.
22. Horswell BB, Holmes AD, Barnett JS, et al: Maxillonasal dysplasia (Binder syndrome): A critical review and case study. *J Oral Maxillofac Surg* 45:114–122, 1987.
23. Jackson IT, Moos KF, Sharpe DT: Total surgical management of Binder syndrome. *Ann Plast Surg* 7:25, 1981.
24. Kraus BS, Decker JD: The prenatal interrelationships of the maxilla and the premaxilla in the facial development of man. *Acta Anat (Basel)* 40:278, 1960.
25. Losken HW, Morris WM: Skull bone grafts in the treatment of maxillonasal dysostosis (Binder syndrome). *S Afr J Surg* 26:90–94, 1988.
26. Macalister: The apertura piriformis. *J Anat Physiol* 32:223, 1898.
27. McLaughlin CR: Absence of the septal cartilage with retarded nasal development. *Br J Plast Surg* 2:61, 1949.
28. McWilliam J, Linder-Aronson S: Hypoplasia of the middle third of the face: A morphological study. *Angle Orthod* 46:260, 1976.
29. Millard DR: Nasal reconstruction with full-thickness cranial bone grafts and rigid internal skeletal fixation through a coronal incision [discussion]. *Plast Reconstr Surg* 86:903, 1990.
30. Monasterio FO, Molina F, McClintock JS: Nasal correction in Binder syndrome: The evolution of a treatment plan. *Aesthetic Plast Surg* 21:299–308, 1997.
31. Munro IR, Sinclair WJ, Rudd NL: Maxillonasal dysplasia (Binder syndrome). *Plast Reconstr Surg* 63:657–663, 1979.
32. Noyes FB: Case report. *Angle Orthod* 9:160, 1939.
33. Olow-Nordenram M, Thilander B: The craniofacial morphology in persons with maxillonasal dysplasia (Binder syndrome). *Am J Orthod Dentofacial Orthop* 95:148, 1989.
34. Olow-Nordenram M, Valentin J: An etiologic study of maxillonasal dysplasia: Binder syndrome. *Scand J Dent Res* 96:69, 1988.
35. Olow-Nordenram MAK, Rådberg CT: Maxillo-nasal dysplasia (Binder syndrome) and associated malformations of the cervical spine. *Acta Radiol Diagn (Stockh)* 25:353, 1984.
36. Posnick JC: Binder syndrome: Evaluation and treatment. In Posnick JC, editor: *Craniofacial and maxillofacial surgery in children and young adults*, Philadelphia, 2000, WB Saunders Co, 21, pp 446–468.
37. Posnick JC, Goh RC, Chen YR: (Discussion of) Surgical management of Binder syndrome: Lessons learned. *Aesthetic Plast Surg* 34:731–733, 2010.
38. Posnick JC, Seagle MB, Armstrong D: Nasal reconstruction with full–thickness cranial bone grafts and rigid internal skeletal fixation through a coronal incision. *Plast Reconstr Surg* 86:894, 1990.
39. Posnick JC, Tompson B: Binder syndrome: Staging of reconstruction and skeletal stability and relapse patterns after Le Fort I osteotomy using miniplate fixation. *Plast Reconstr Surg* 99(4):961–973, 1997.
40. Psillakis JM, Lapa F, Spina V: Surgical correction of midfacial retrusion (nasomaxillary hypoplasia) in the presence of normal dental occlusion. *Plast Reconstr Surg* 51:67, 1973.
41. Quarrell CR: Absence of the septal cartilage with retarded nasal development. *Br J Plast Surg* 2:61, 1949.
42. Ragnell A: Nasomaxillary retroposition in children: Successive reconstruction of the nose: A preliminary report. *Nord Med* 77:847, 1967.
43. Resche F, Tessier P, Delaire J, et al: Craniospinal malformations associated with maxillofacial dysostosis (Binder syndrome). *Head Neck Surg* 3:123, 1990.
44. Resche F, Tulasne JF, Tessier P, et al: Malformations de la charniere cranio-rachidienne et du rachis cervical associes a la dysplasie maxillo-nasale de Binder. *Rev Stomatol Chir Maxillofac* 80:83, 1979.

45. Rival JM, Gherga-Negrea A, Mainard R, et al: Dysostose maxillo-nasale de Binder. *J Genet Hum* 22:263, 1974.
46. Rune B, Aberg M: Bone grafts to the nose in Binder syndrome (maxillonasal dysplasia): A follow-up of eleven patients with the use of profile roentgenograms. *Plast Reconstr Surg* 101:297–304; discussion 305-306, 1998.
47. Sheffield LH, Halliday JL, Jensen F: Maxillonasal dysplasia (Binder's syndrome) and chondrodysplasia punctata [letter]. *J Med Genet* 28:503–504, 1991.
48. Sheffield LJ, Halliday JL, Danks DM, et al: Clinical, radiological and biochemical classification of chondrodysplasia punctata [abstract]. *Am J Hum Genet* 45(Suppl):A64, 1989.
49. Steinhauser EW: Variations of Le Fort II osteotomies for correction of midfacial deformities. *J Maxillofac Surg* 8:258, 1980.
50. Tessier P: Aesthetic aspects of bone grafting to the face. *Clin Plast Surg* 8:279–301, 1981.
51. Tessier P, Tulasne JF, Delaire J, et al: Therapeutic aspects of maxillonasal dysostosis (Binder syndrome). *Head Neck Surg* 3:207, 1981.
52. Tham C, Lai YL, Weng CJ, et al: Silicone augmentation rhinoplasty in an Oriental population. *Ann Plast Surg* 54:1–5; discussion 6-7, 2005.
53. Von Binder KH: Dysostosis maxillo-nasalis, ein arhinencephaler missbildungscomplex. *Dtsch Zahnaerztl* 17:438, 1962.
54. Watanabe T, Matsuo K: Augmentation with cartilage grafts around the pyriform aperture to improve the midface and profile in Binder syndrome. *Ann Plast Surg* 36:206–211, 1996.
55. West RA: (Discussion of) Binder syndrome: Staging of reconstruction and skeletal stability and relapse patterns after Le Fort I osteotomy using miniplate fixation. *Plast Reconstr Surg* 99:961, 1997.
56. Zuckerkandl E: Fossae praenasales: Normale und pathologische. *Anat Nasenhohle* 1:48, 1882, 997.

30

Syndromes with Craniosynostosis: Evaluation and Treatment

JEFFREY C. POSNICK, DMD, MD

- General Considerations
- Mutations in Craniosynostosis and Craniosynostosis Syndromes
- Complex Craniosynostosis
- Neurologic Aspects of Craniosynostosis
- Effects of Midface Deficiency on the Upper Airway
- Morphologic Considerations
- Surgical Approach to Craniosynostosis Sydromes
- Staging of Reconstruction for Crouzon Syndrome
- Staging of Reconstruction for Apert Syndrome
- Conclusions

General Considerations

Craniosynostosis, which is the premature fusion of cranial sutures, affects approximately 1 in 2500 children. Patients may present with a wide range of phenotypic and functional deformities.[94] Virchow's law from 1851 states that the premature fusion of a cranial vault suture results in growth arrest perpendicular to the affected suture and compensatory growth parallel to the fused suture.[16,164,269] This principle is generally applicable to single sutures; multisutural synostosis—such as the combination of sagittal and lambdoid synostosis (coup de sabre) or a cloverleaf skull—can assume more complex patterns of cranial morphology.[43,46,47] In addition to the type of affected suture, the eventual head shape depends on the timing and order of cranial suture fusion. Craniosynostosis may be of prenatal or perinatal onset, or it may occur during infancy.[14,74] The earlier synostosis occurs, the more dramatic the effect on subsequent cranial growth and development; the later synostosis occurs, the less effect on cranial growth and development.[44]

Active pediatric neurosurgical services generally see a characteristic frequency, gender predilection, and phenotypic presentation for sutural involvement.[6,15,55,125,133,154,161,193,232,238,243,278,283] Sagittal synostosis occurs most commonly, with a higher incidence among boys than girls. Patients typically present with a boat-shaped skull (i.e., scaphocephaly) in combination with midline ridging in the posterior half of the skull. Metopic synostosis is currently the second most frequent form of synostosis. Presentations occur along a spectrum that ranges from metopic ridging to the presence of a triangular-shaped skull (i.e., trigonocephaly). It should be noted that metopic ridging is common during infancy and childhood and that it is not necessarily associated with a diagnosis of craniosynostosis. Coronal synostosis occurs next most frequently; girls slightly predominate or, in some surveys, boys and girls have equivalent frequency.[46] Unicoronal synostosis is marked by frontal plagiocephaly, whereas bicoronal synostosis is characterized by a short and wide skull (i.e., brachycephaly). Multiple synostoses of various types are less common than coronal synostosis. Lambdoid synostosis occurs with least frequency, and it is marked by occipital plagiocephaly.[44]

Craniosynostosis is etiologically heterogeneous and pathogenetically variable. Overall, 8% of cases have a familial component, and this is generally marked by an autosomal dominant inheritance pattern. Specifically, pedigrees are familial in 14.4%, 6%, and 5.6% of coronal, sagittal, and metopic synostoses, respectively.[44] Although some pedigrees show synostosis of a specific suture, others may show fusion of different sutures in affected relatives of the same family. Different chromosomal aberrations have been linked to craniosynostosis, with variable penetrance. In some conditions the penetrance is high, such as with dup(3q), del(7p), del(9p), and del(11q). In other cases, penetrance

can be very low, such as with dup(5p), del(6)(22q22.2-q23.1), del(8q), and dup(15q).[44]

Many syndromes are associated with craniosynostosis, and well over 100 are known.[44] It is important to accurately diagnose syndromal patients for three reasons.[22] First, most syndromes with craniosynostosis affect not only the cranial vault but also the cranial base and the midface. Deficiencies in these skeletal sites, which are variable in degree yet commonly serious, must be addressed as part of the staged reconstructive approach. Second, syndromes are often genetic and familial, thus necessitating proper counseling. Third, depending on the experience of the particular surgeon, the molecular diagnosis should be made. Often, the patient may be referred for molecular diagnosis and counseling to avoid the possibility of litigation.[37,43,46,47]

Primary forms of craniosynostosis are most common and include single- and multi-sutural synostosis. Some conditions, however, result in secondary synostosis. These include hyperthyroidism; rickets; mucopolysaccharidoses, such as Hurler syndrome and Morquio syndrome; hematologic disorders, such as thalassemia and sickle cell anemia; teratogens, such as diphenylhydantoin, retinoids, valproate, and aminopterin; and certain malformations, such as holoprosencephaly and microcephaly.[44]

Mutations in Craniosynostosis and Craniosynostosis Syndromes

Mutations responsible for craniosynostosis have been identified in *FGFR1*, *FGFR2*, *FGFR3*, *TWIST*, *MSX2*, *EFNB1*, *RAB23, EFNA4*, *POR*, and *ALPL*.[44] These include Apert syndrome (two common *FGFR2* mutations; Ser252Trp, Pro253Arg), Crouzon syndrome (more than 30 *FGFR2* mutations), Pfeiffer syndrome (more than 30 *FGFR2* mutations), are known and at least 6 of them are the same as those found with Crouzon syndrome; one Pfeiffer syndrome (mutation on *FGFR1*).* In addition, there are some cases of *FGFR2* mutations with isolated coronal synostosis as well as with some other forms of non-syndromic synostosis. A single known *FGFR2* mutation is known for Jackson-Weiss syndrome.[1,41,182] Although this disorder is distinctly uncommon, affected families are often large, with variable phenotypic expression. Beare-Stevenson cutis gyrate syndrome involves one of two possible mutations in the transmembrane domain of *FGFR2*.[37,42]

Muenke syndrome is the most common craniosynostosis syndrome that is currently known. It involves a single mutation on *FGFR3,* and it is characterized most commonly by unilateral coronal synostosis and less commonly by bicoronal synostosis. About 6% of affected patients have macrocephaly without synostosis, and cloverleaf skull has been reported in some instances.[37,42] Therefore, all patients with coronal synostosis should be tested for this very common *FGFR3* mutation; if they are negative, they should be tested for *FGFR2.*

Crouzonodermoskeletal syndrome is characterized by one specific *FGFR3* mutation. The name indicates its characteristic features: either a Crouzonoid or cloverleaf skull appearance; acanthosis nigricans; and a decreased interpediculate distance that is radiographically present but not clinically significant. Serious decreased interpediculate distance is a feature of the most common mutation for achondroplasia, which is only 11 amino acids away from the *FGFR3* mutation for Crouzonodermoskeletal syndrome. To call this disorder "Crouzon syndrome with acanthosis" is unwarranted, because only one *FGFR3* mutation is responsible for all cases; this is in contrast with Crouzon syndrome, which involves more than 30 different *FGFR2* mutations.[38]

Rarely, patients with achondroplasia and hypochondroplasia (both of which involve mutations on *FGFR3*) may also have craniosynostosis (there have been three reported cases of hypochondroplasia and two reported cases of achondroplasia). Saethre–Chotzen syndrome has *TWIST* mutations inside or outside of the coding region that result in haploinsufficiency.[48] Craniofrontonasal syndrome is caused by heterozygous loss-of-function mutations in *EFNB1*. *EFNA4* mutations rarely cause non-syndromal coronal synostosis. Carpenter syndrome, which is an autosomal recessive disorder, is caused by *RAB23* mutations. Boston-type craniosynostosis, which is an autosomal dominant disorder caused by *MSX2* mutations, presents with variable expressivity (coronal synostosis, frontal depression, or cloverleaf skull). Antley–Bixler syndrome is caused by *POR* mutations, and infantile hypophosphatasia is caused by *ALPL* mutation.[44,184]

Complex Craniosynostosis

Background

Complex craniosynostosis, which involves the fusion of multiple cranial sutures, occurs in about 5% of non-syndromic cases.[47] Two thirds of cases involve two affected sutures, whereas one third of cases involve more than two affected sutures.[203] As the number of affected sutures increases, so does the risk of intracranial hypertension and associated mental deficiency. As demonstrated by Renier and colleagues, the incidence of intracranial hypertension rises from 14% to 47% when going from single to several affected sutures.[228,226,227,229,230] In addition, increasing sutural involvement can be accompanied by worsened phenotypic severity. In a series reported by Chumas and colleagues,[34] 59% of cases of sagittal and bilateral lambdoid synostosis had an acute angulation of the posterior skull, which is known in French as *coup de serpe* and means "cut with a scythe." In a similar fashion, marked anterior dysmorphism with frontal bone hypoplasia was found in 23% of cases of metopic and bilateral coronal synostosis.

*References 5, 42, 45, 81, 82, 95, 115, 116, 131, 143, 159, 160, 165-168, 177, 183, 223, 233, 236, 248, 250, 251

Cloverleaf skulls, which represent the extremes of phenotypic severity, are pathogenetically variable (Fig. 30-1).[30,204] Synostosis may involve the coronal, lambdoid, and metopic sutures, which are marked by bulging of the cerebrum through an open sagittal suture or, in some cases, through patent squamosal sutures. Synostosis of the sagittal and squamosal sutures with cerebral eventration through a widely patent anterior fontanel may also be observed. A trilobular skull shape may also occur, with complete synostosis of all cranial sutures in some cases or with widely patent sutures and no craniosynostosis at birth in other instances.[36,51] In addition to being pathogenetically variable, cloverleaf skulls are also etiologically heterogeneous. This condition most commonly occurs with type 2 thanatophoric dysplasia (i.e., in about 40% of cases) with a specific *FGFR3* mutation, but stillborn status or an early demise during very early infancy precludes treatment. Isolated cloverleaf skull occurs in about 20% of cases. It also occurs in about 15% of cases of type 3 Pfeiffer syndrome, and, although the prognosis is guarded, these patients can be treated aggressively.[45,48]

Plagiocephaly is defined as the asymmetric distortion of the skull. Well-known types include synostotic anterior plagiocephaly (unilateral coronal synostosis), synostotic posterior plagiocephaly (unilateral lambdoid synostosis), deformational anterior plagiocephaly, and deformational posterior plagiocephaly.[146,148,268] Among these types, deformational posterior plagiocephaly is common, whereas unilateral lambdoid synostosis is rare. Deformational posterior plagiocephaly can be caused by intrauterine factors such as hypotonia, fetal positioning, and prematurity. This can produce asymmetric flattening of the occiput that becomes favored by infants sleeping on their backs, thereby exaggerating the plagiocephaly. Deformational posterior plagiocephaly has also increased dramatically since the 1992 "Back to Sleep" campaign by the American Academy of Pediatrics, which calls for supine infant sleeping to reduce the risk of sudden infant death syndrome.[44] Finally, rare forms of plagiocephaly can be produced by unilateral frontosphenoidal synostosis and unilateral frontozygomatic synostosis.[44]

Apert Syndrome

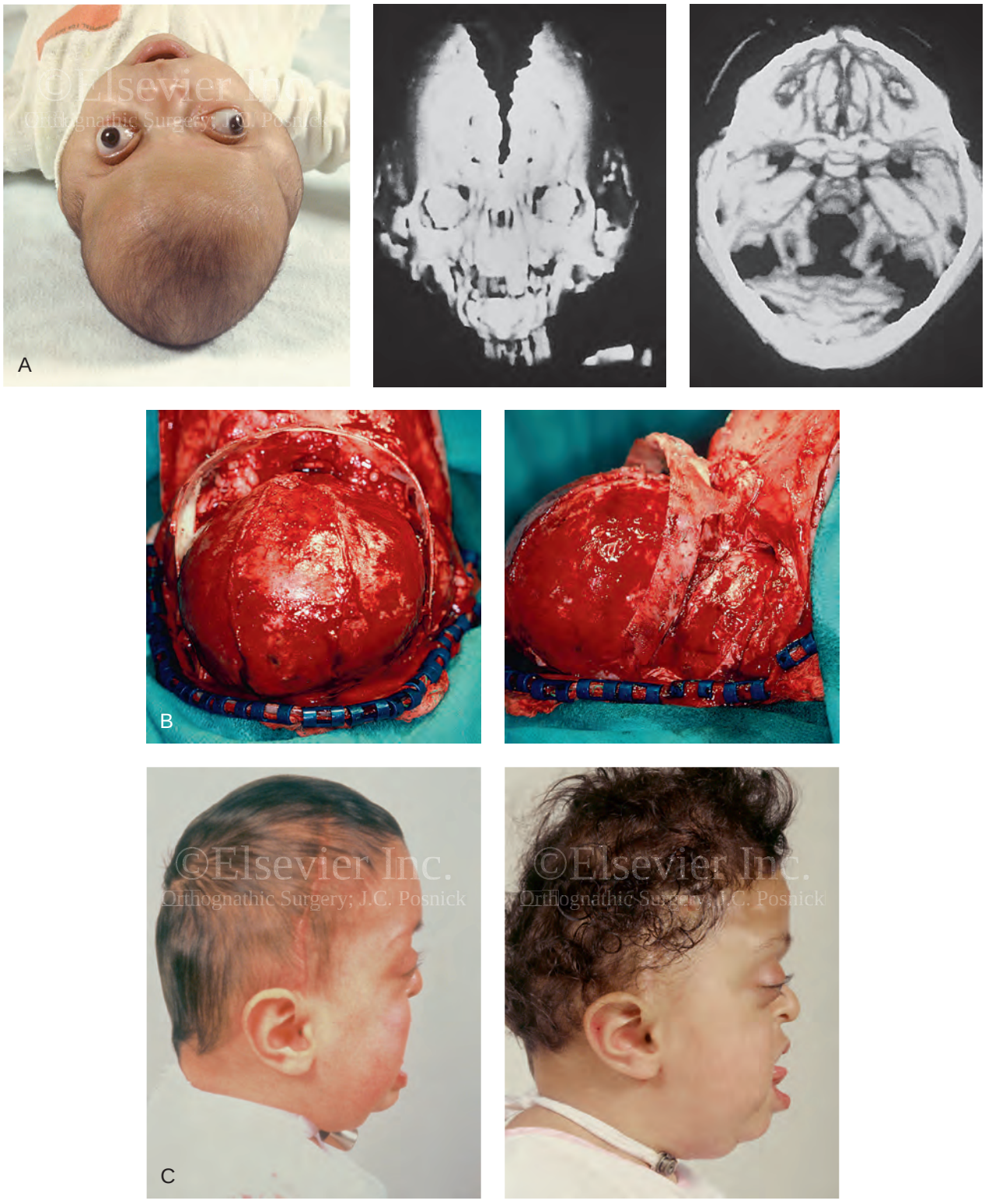
Apert syndrome is characterized by craniosynostosis, midface deficiency, symmetric syndactyly of the hands and feet, and other abnormalities* (Figs. 30-2 through 30-5). Most cases are associated with brachycephaly secondary to bicoronal synostosis. Megalencephaly and increased cranial height are characteristic of Apert syndrome; affected children typically display head circumference, length, and weight that are above the normal 50th percentile. Benign distortion ventriculomegaly is also a feature of Apert syndrome, and patent sutures and synchondroses (except for coronal synostosis) are present as well. The frequency of progressive hydrocephalus is much lower than in it is with Crouzon and Pfeiffer syndromes. Central nervous system anomalies may include hypoplasia of the corpus callosum, agenesis of the corpus callosum, agenesis of the septum pellucidum, cavum septum pellucidum, dorsally displaced hippocampi and hippocampal gyri, hypoplastic white matter, and heterotopic gray matter.[46,48]

Skeletal abnormalities with Apert syndrome include symmetric syndactyly of the hands and feet; abnormal shoulders with significant limitation of motion; and abnormalities of the elbows, hips, knees, rib cage, and spine.[46] It should be carefully noted that the radiologic interpretation of the hands, feet, and cervical spine as being affected by so-called "progressive fusion" is very misleading. In actuality, there is a failure of cartilage segmentation during embryonic and fetal life, so these abnormalities are preset (ab initio). These failures in cartilage segmentation cannot be readily observed radiographically at birth. When they do become evident radiographically after birth, they are said to be a "progressive abnormality," which they are not. The same can be said for the cervical vertebrae,[46] as 68% of these patients have cervical abnormalities, particularly involving C5 and C6. Before considering a surgical procedure, magnetic resonance imaging of the brain, radiography of the cervical spine, and the assessment of cardiovascular (10% of patients) and genitourinary (9.6% of patients) anomalies should be carried out.[44,46,48]

Patients with Apert syndrome also suffer from reduced nasopharyngeal dimensions and reduced patency of the posterior choanae; these features pose the risks of respiratory embarrassment, obstructive sleep apnea (OSA), cor pulmonale, and even sudden death. During infancy, patients may be trialed on a nasopharyngeal airway, but tracheostomy may be the only definitive treatment. Clinicians must always maintain a high index of suspicion for OSA and monitor patients for snoring or an unusual amount of daytime somnolence. When either of these is apparent, patients should be referred to a sleep center for proper diagnosis and treatment. It should be carefully noted that, although patients with Apert syndrome very commonly have OSA, the excessive sweating that occurs with this syndrome is not a sign of OSA but rather a consequence of the increased number of sweat and sebaceous glands that accompany the syndrome. Even those patients who have surgical advancement of the midface in combination with continuous positive airway pressure (CPAP) will still have excessive sweating of the head at night and of the hands all the time. Another consequence of an increased number of sweat and sebaceous glands is a high frequency of conglobate acne during adolescence, often with extension to the forearm, the thighs, and the buttocks. The acne vulgaris of Apert syndrome suggests exquisite end-organ responsiveness to steroid hormones.[46]

*References 7, 13, 17, 54, 88, 98, 99, 104, 121, 123, 124, 128-130, 132, 142, 147, 152, 186

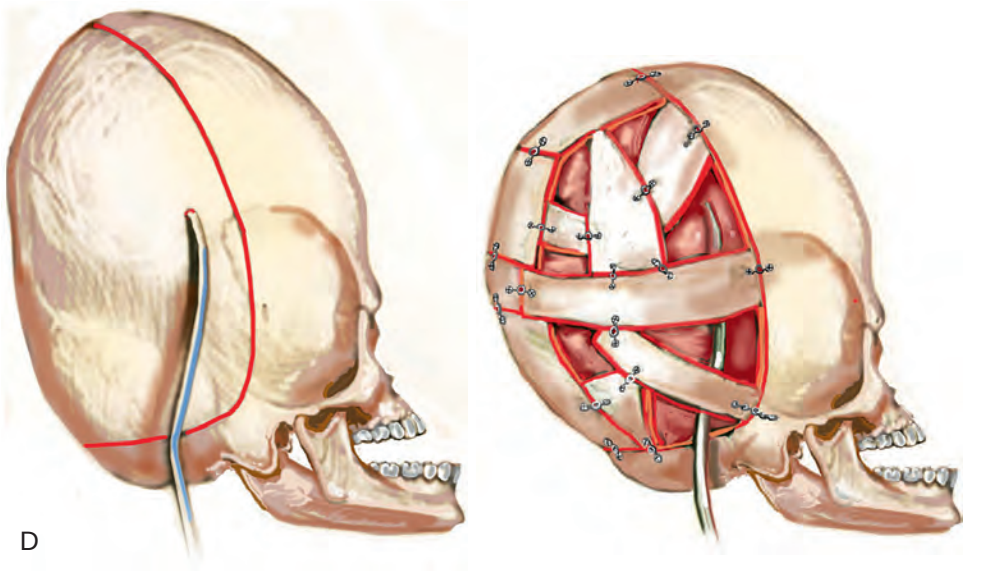
Text continued on p. 1200

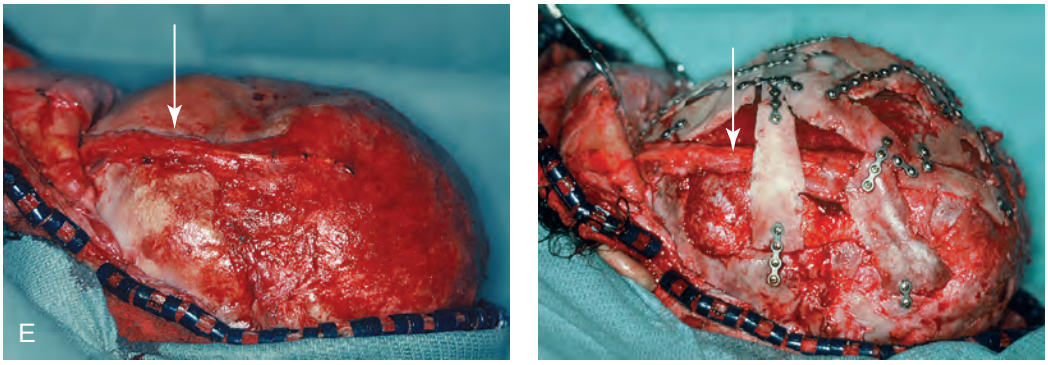


©Elsevier Inc.
Orthognathic Surgery; J.C. Posnick

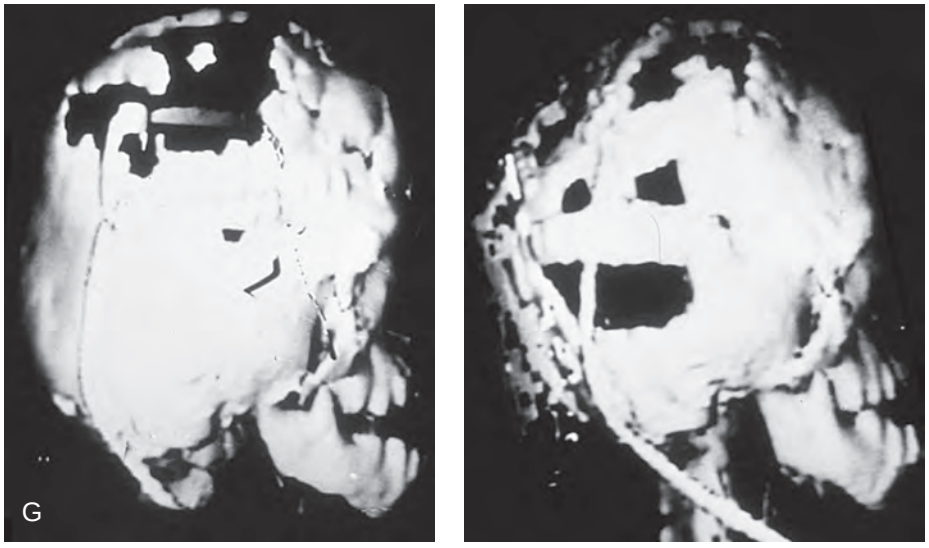
• **Figure 30-1** A child with a severe form of cloverleaf skull anomaly. At 10 months of age, she was referred to this surgeon and underwent first-stage cranial vault and upper orbital decompression with reshaping. She then required a ventriculoperitoneal shunt for the management of hydrocephalus. When the patient was 3½ years old, posterior cranial vault decompression with reshaping to increase the intracranial volume was performed. When she was 4½ years old, facial bipartition osteotomies in combination with anterior cranial vault reshaping and advancement were carried out. After the cranial vault and facial bipartition procedures, the patient's airway improved, and it was possible to remove the tracheostomy tube. The cranial vault reshaping expanded the intracranial volume, thereby providing more space for the brain. The midface advancement improved the eye proptosis as well as the patient's ability to chew and articulate speech. As part of the staged reconstruction, she will require orthognathic surgery in combination with orthodontic treatment at the time of early skeletal maturity. **A,** Frontal facial and computed tomography (CT) scan views at 10 months of age. **B,** Intraoperative views at 10 months of age after craniotomy and the removal of the cranial vault and the upper orbits. The "bandeau" is in place (3 cm advancement) before the reconstruction of the overlying cranial vault. **C,** Profile views at 2½ and then at 3½ years of age with flattened posterior cranial vault and severe midface deficiency.

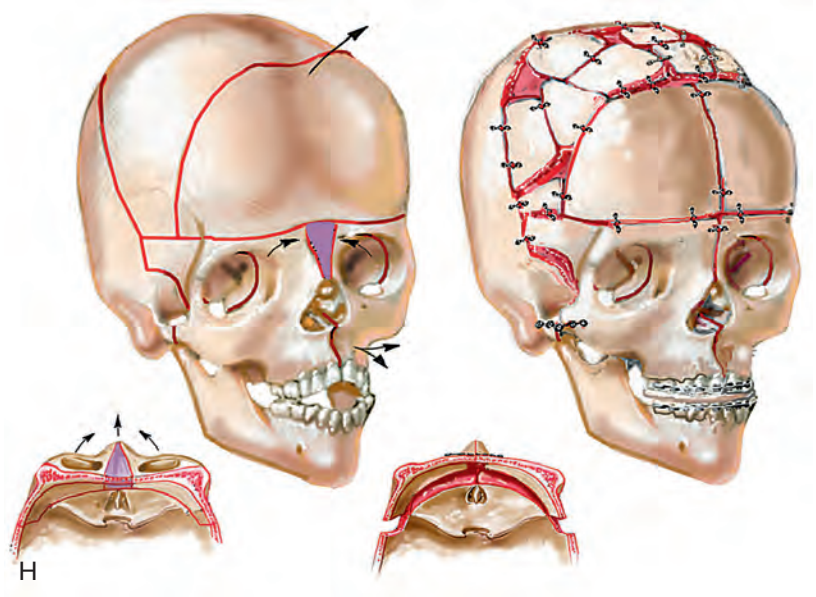
Continued





• **Figure 30-1, cont'd** **D,** Illustration of flattened posterior cranial vault with the ventriculoperitoneal shunt in place. Illustration after posterior cranial vault reshaping is also shown. **E,** Intraoperative side view with the patient in the prone position before and after the craniotomy and the reshaping of the posterior cranial vault. Arrows point to the ventriculoperitoneal shunt, which remains intact and deep to the skull reconstruction.

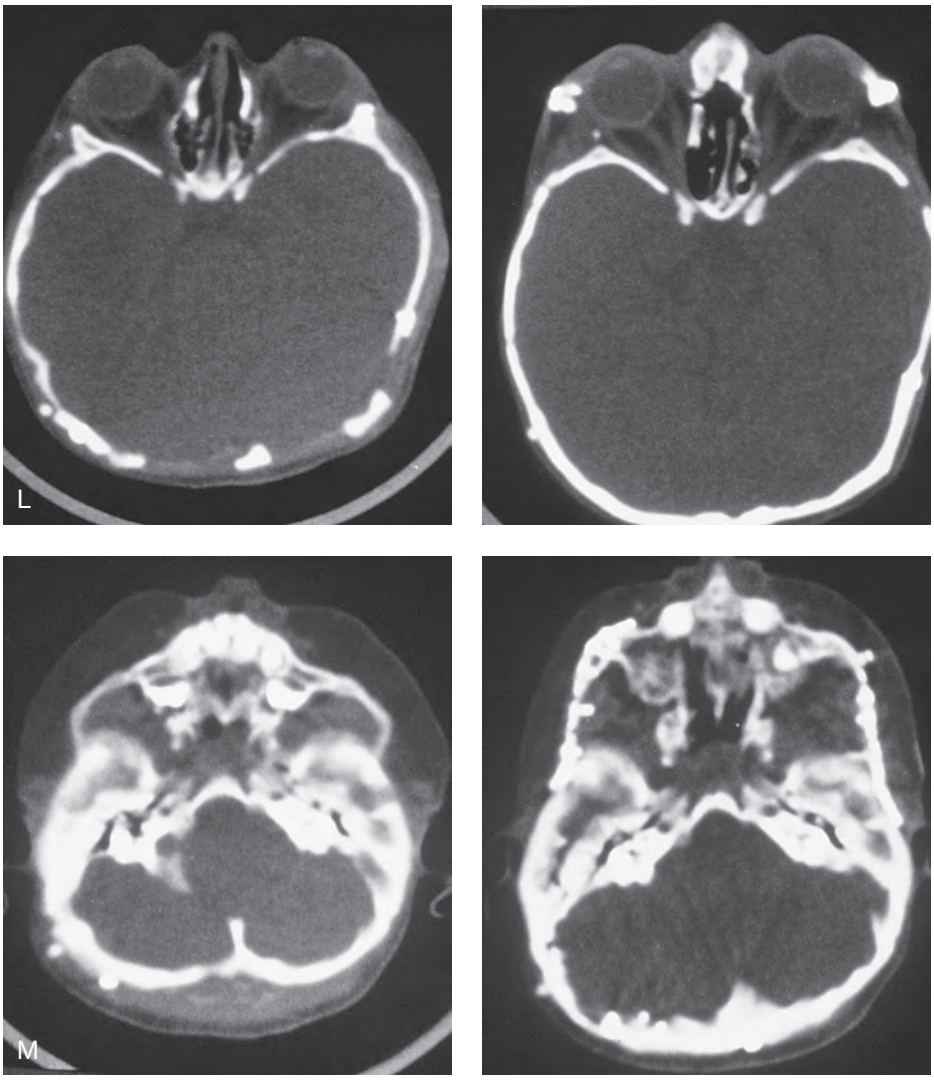




• **Figure 30-1, cont'd F,** Profile view at 3½ years of age, before and after posterior cranial vault reconstruction. **G,** CT scan views before and just after posterior cranial vault reshaping. **H,** Illustration before facial bipartition. The second illustration indicates the planned reconstruction.

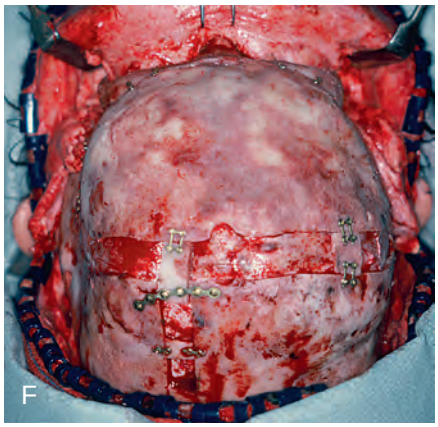
Continued

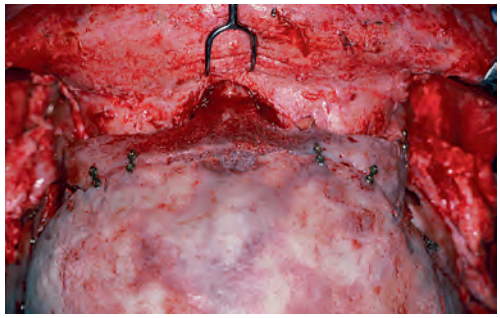
• **Figure 30-1, cont'd I,** Oblique facial views before and after facial bipartition reconstruction. The tracheostomy has been removed. **J,** Facial views before and after facial bipartition reconstruction. **K,** Profile views before and after facial bipartition reconstruction.



• **Figure 30-1, cont'd L,** Axial CT views through the midorbits before and after reconstruction. **M,** Axial CT views through the zygomatic arches before and after reconstruction. *A, B, C (right), D, E, F, H (left), I, K, L, M, From Posnick JC: The craniofacial dysostosis syndromes: secondary management of craniofacial disorders,* Clin Plast Surg *24:429-446, 1997.*

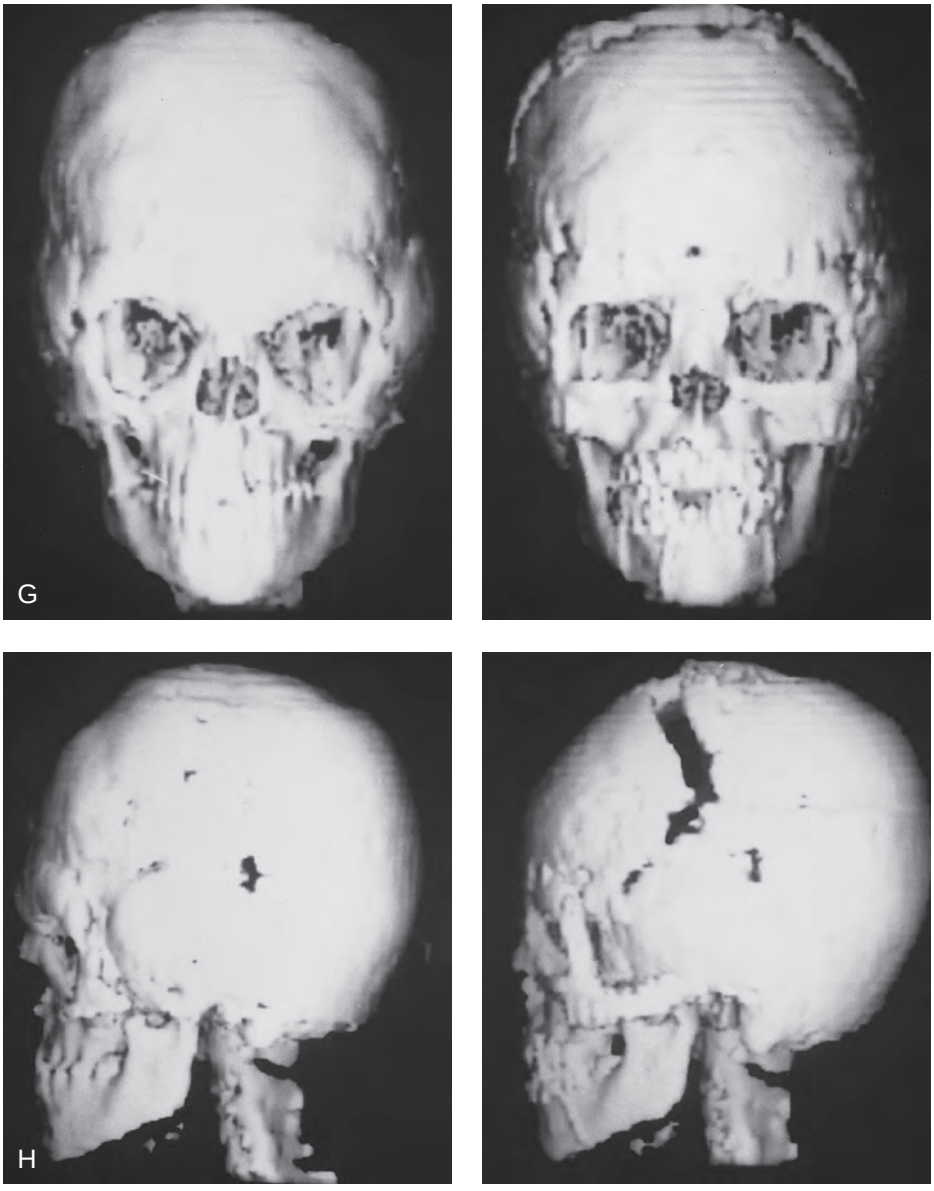
• **Figure 30-2** A child who was born with Apert syndrome underwent a bilateral lateral canthal advancement procedure when she was 6 weeks old; this was carried out by a neurosurgeon working independently. When she was 18 months old, she returned with turricephaly and a constricted anterior cranial vault that required further cranio–orbital decompression and reshaping. When she was 5 years old, she underwent anterior cranial vault and facial bipartition osteotomies with reshaping. As part of the staged reconstruction, she will require orthognathic surgery and orthodontic treatment during her teenage years. **A,** Illustrations of craniofacial morphology before and after the facial bipartition osteotomies and reconstruction. **B,** Frontal views before and after facial bipartition reconstruction. **C,** Oblique facial views before and after facial bipartition reconstruction.



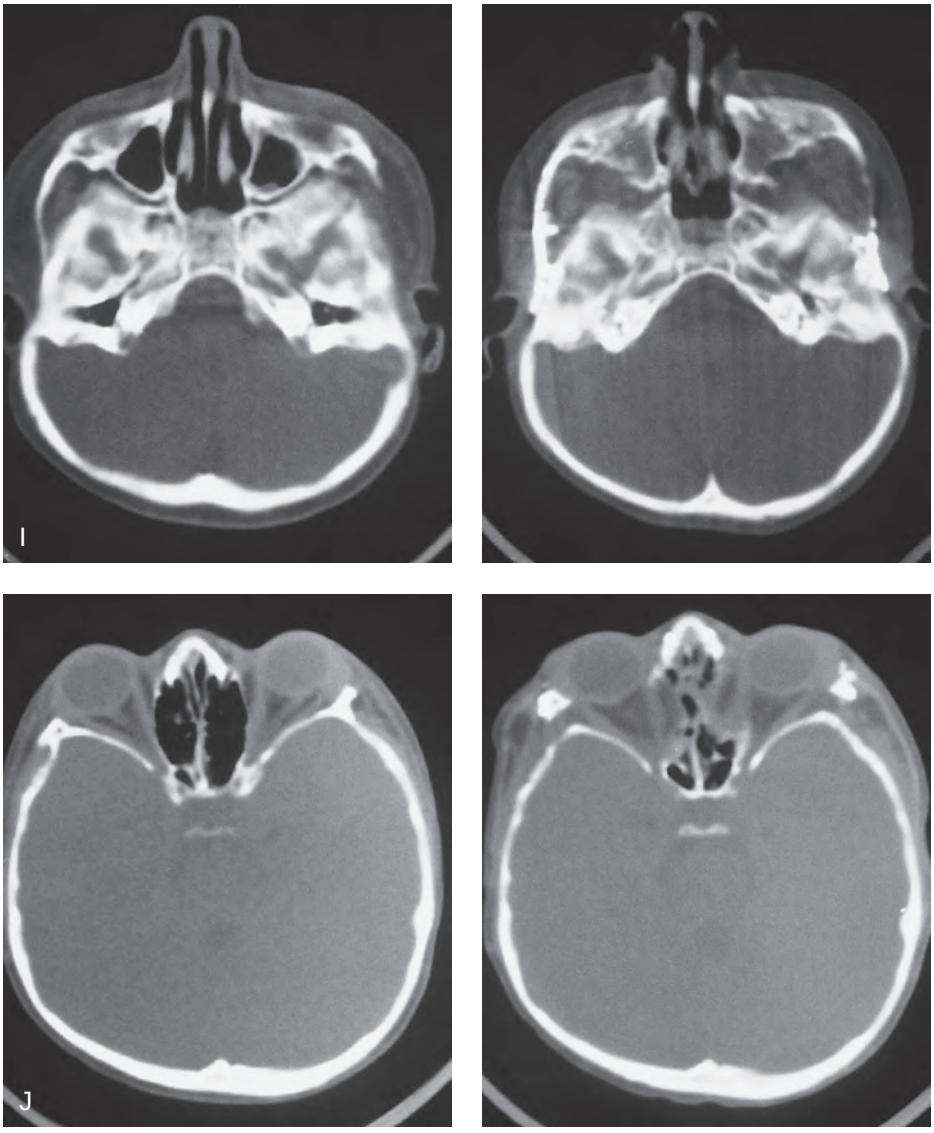


• **Figure 30-2, cont'd D,** Profile views before and after facial bipartition reconstruction. **E,** Worm's-eye views before and after facial bipartition reconstruction. **F,** Intraoperative views after facial bipartition and anterior cranial vault reconstruction.

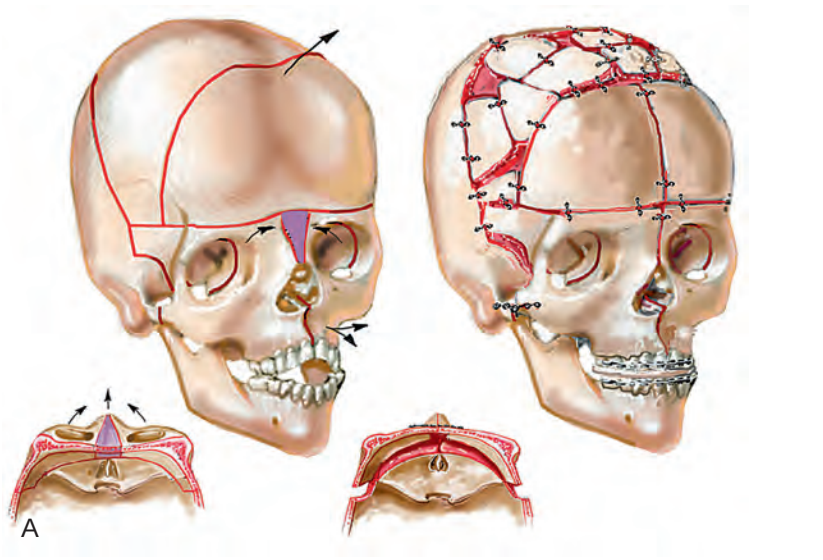
Continued



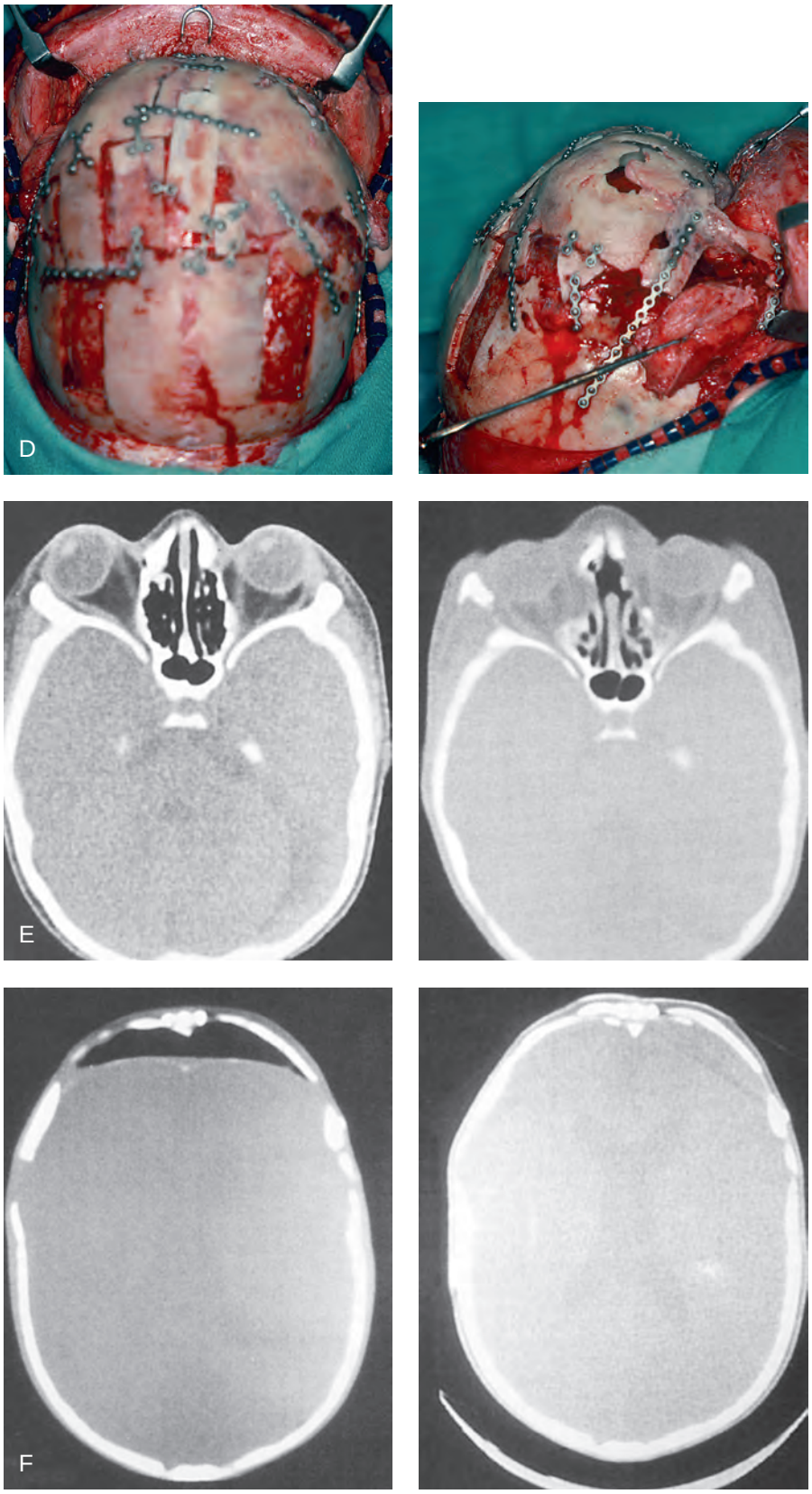
• **Figure 30-2, cont'd G** and **H,** Computed tomography scan views before and early after reconstruction.



• **Figure 30-2, cont'd I,** Axial computed tomography scan views through the zygomas before and after reconstruction. **J,** Axial computed tomography scan views through the midorbits before and after reconstruction indicating relief of proptosis.



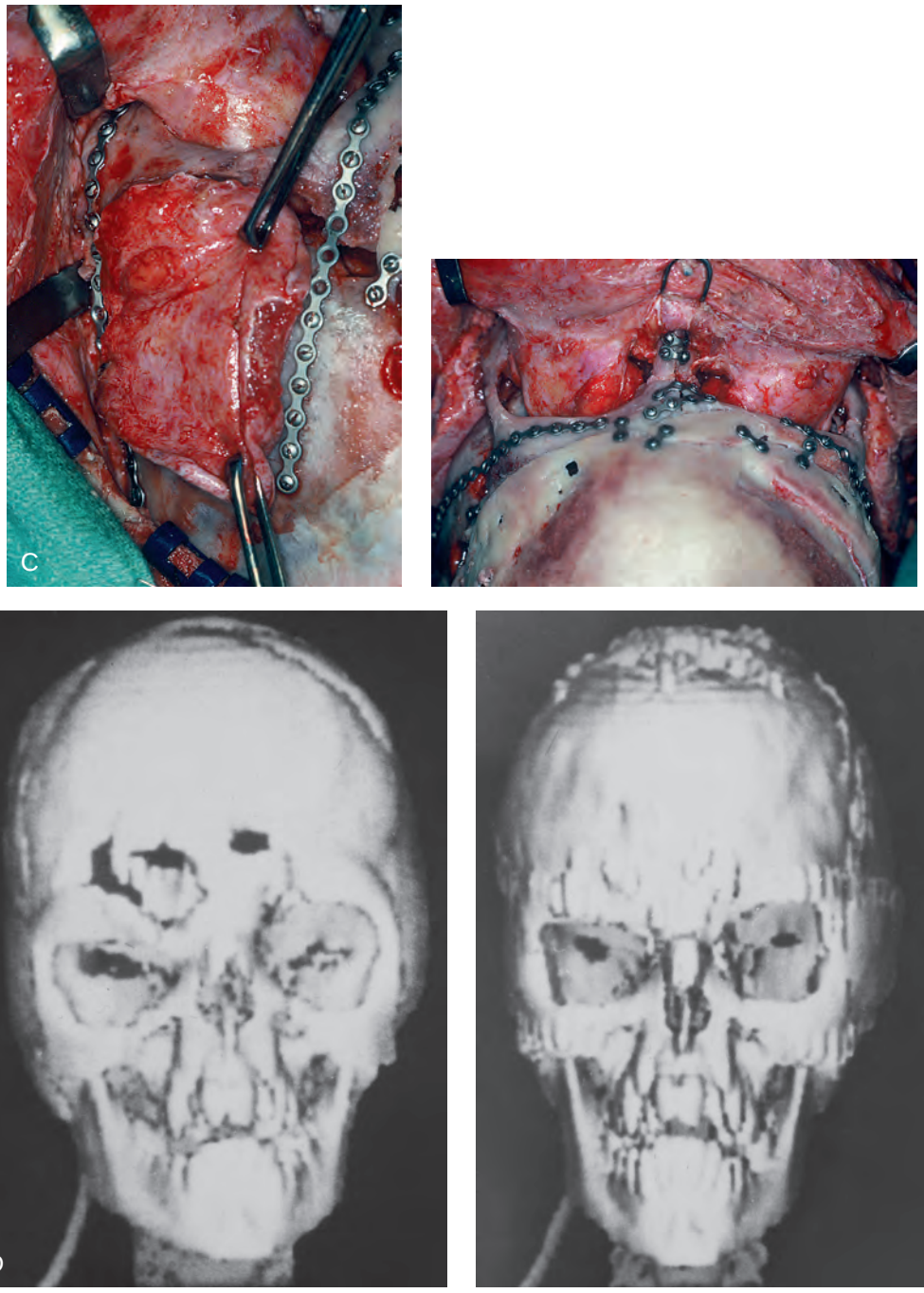
• **Figure 30-3** A 5-year-old girl with Apert syndrome who underwent lateral canthal advancements when she was 6 months old and in the care of another surgeon. She then presented to this surgeon with residual craniofacial deformities that required anterior cranial vault and facial bipartition osteotomies with reshaping. She will require orthognathic surgery and orthodontic treatment during her teenage years to complete the reconstruction. **A,** Illustrations of preoperative craniofacial morphology. The planned cranial vault and facial bipartition osteotomies and the planned reshaping are also shown. **B,** Frontal facial views before and after anterior cranial vault and facial bipartition reconstruction. **C,** Profile views before and after facial bipartition reconstruction.



• **Figure 30-3, cont'd D,** Intraoperative views after anterior cranial vault and facial bipartition reconstruction. **E,** Axial computed tomography scan views through the midorbits before and after reconstruction that demonstrate improvement in orbital hypertelorism and orbital depth with diminished eye proptosis. **F,** Axial computed tomography scan views through the cranial vault 1 week after facial bipartition (note the dead space in the retrofrontal region) and at 1 year (note that the initial retrofrontal dead space has been resolved by brain expansion). *A (right), C (left), E, From Posnick JC: Craniofacial dysostosis. Staging of reconstruction management of the midface deformity,* Neurosurg Clin North Am *2:683-702, 1991.*

©Elsevier Inc.
Orthognathic Surgery; J.C. Posnick

• **Figure 30-4** An 8-year-old girl who was born with Apert syndrome. She had undergone cranio–orbital surgery on two occasions and an attempted Le Fort III osteotomy at another institution. The previous procedures had resulted in complications, including infection and traumatic encephalocele. She was referred to this surgeon with cranial vault dysplasia, orbital dystopia, hypertelorism, midface deficiency, and skull defects. She then underwent anterior cranial vault, facial bipartition, and additional segmental orbital osteotomies with reshaping and reconstruction. She will require orthodontic treatment and orthognathic surgery during her teenage years to complete the reconstruction. **A,** Frontal views before and after anterior cranial vault and facial bipartition reconstruction. **B,** Oblique facial views before and after facial bipartition reconstruction.



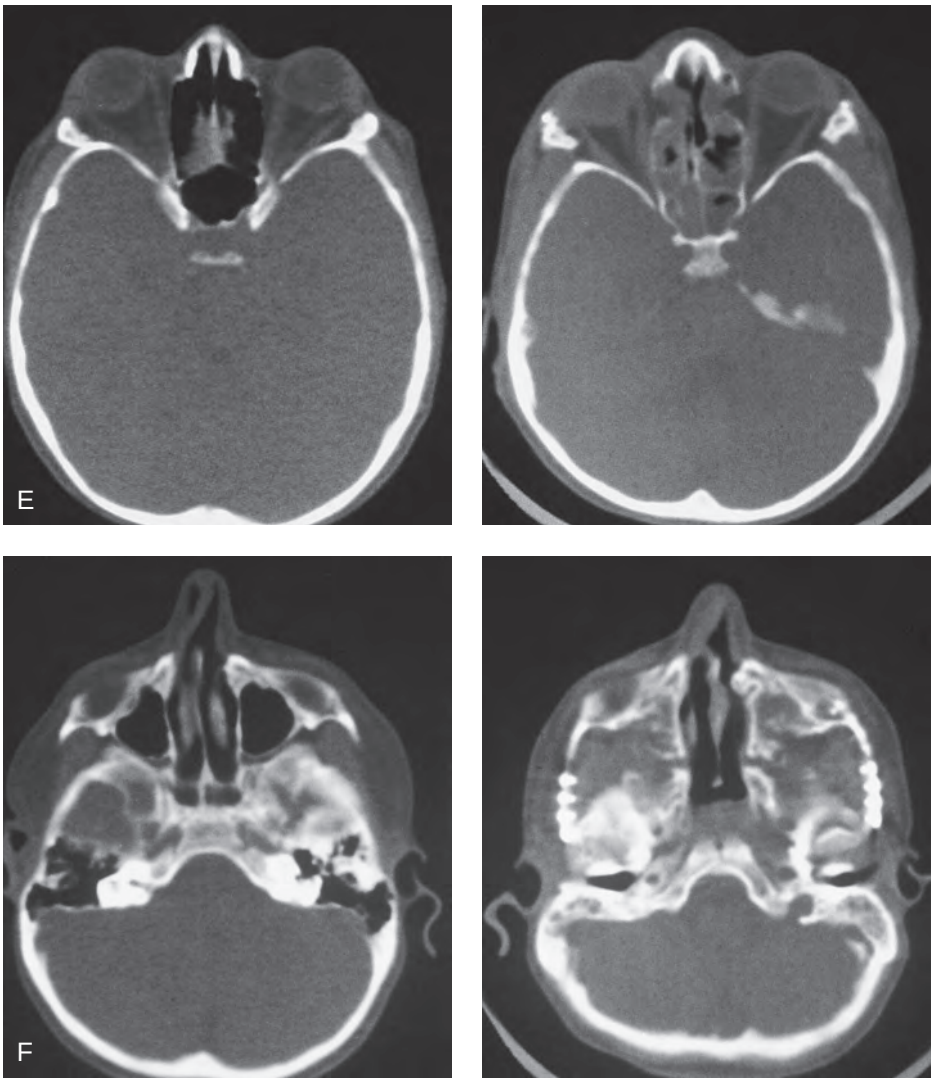
• **Figure 30-4, cont'd C,** Intraoperative views after anterior cranial vault and facial bipartition reconstruction. Encephalocele and skull defects have also been repaired. **D,** Computed tomography scan views before and early after reconstruction. *From Posnick JC: Craniosynostosis: surgical management in infancy. In Bell WH, ed:* Orthognathic and reconstructive surgery, *vol 3, Philadelphia, 1992, W.B. Saunders, p 1839.*

• **Figure 30-5** A child born with a mild form of Apert syndrome. When he was 10 years old, he was referred to this surgeon and underwent anterior cranial vault and monobloc osteotomies with advancement. He will require orthognathic surgery combined with orthodontic treatment to complete the reconstruction. **A,** Facial views during infancy. **B,** Views of the hands and feet that indicate complex compound syndactyly.

©Elsevier Inc.
Orthognathic Surgery; J.C. Posnick

• **Figure 30-5, cont'd** **C,** Frontal facial views before and after anterior cranial vault and monobloc osteotomies with advancement. **D,** Profile views before and after monobloc reconstruction.

Continued



• **Figure 30-5, cont'd** **E,** Axial computed tomography scan views through the midorbits before and after reconstruction indicating relief of proptosis. **F,** Axial computed tomography scan views through the zygomatic arches before and after reconstruction.

Ocular findings include hypertelorism, proptosis (often asymmetric), and down-slanting palpebral fissures. The presence of V-pattern strabismus, which is marked by divergent upgaze and esotropic downgaze, is common and secondary to structural abnormalities of the extraocular muscles (i.e., the absence of the superior rectus muscle). Hyperopia, myopia, and astigmatism can also be present. Strabismus and significant refractive errors can sometimes cause amblyopia. Proptosis in Apert syndrome may require tarsorrhaphies to prevent exposure keratitis.[131]

Hearing loss occurs in 90% of these patients, with 80% of cases resulting from conductive pathology. Inner-ear anomalies are found in all patients; these most commonly present as dilated vestibules, malformed semicircular canals, and cochlear dysplasia.[285] Otitis media is common, and it is possibly related to the presence of cleft palate and accompanying eustachian tube dysfunction.

Oral and maxillofacial anomalies can also be present. The palate can be highly arched and constricted with a median furrow in up to 94% of patients. The hard palate is often shorter than normal, whereas the soft palate is longer and thicker than normal. Clefting of the soft palate occurs in 41% of patients, and a bifid uvula is present in 35%. Dental anomalies include severely delayed eruption (68%), ectopic eruption (50%), and shovel-shaped incisors (30%). Dental crowding is more severe in the maxilla than in the mandible. Common dental deformities include anterior open bite (73%), posterior crossbite (63%), and mandibular overjet (81%).[46,48]

Crouzon Syndrome

Crouzon syndrome is characterized by craniosynostosis, maxillary hypoplasia, shallow orbits, and ocular proptosis.* (Figs. 30-6 through 30-11). Most cases are associated with brachycephaly secondary to bicoronal synostosis; however, trigonocephaly, scaphocephaly, and cloverleaf skull have also been noted. Cranial malformation depends on the order and rate of progression of the sutural synostosis. The cranial volume of patients with Crouzon syndrome is much smaller than that found in patients with Apert syndrome. Central nervous system findings include shunted hydrocephalus (25%), chronic tonsillar herniation (72%), jugular foramen stenosis with venous obstruction (30%), seizures (10.5%), frequent headaches (29%), and mental deficiency (3%).[46,48] As a result of severe maxillary deficiency, patients should be monitored for snoring and excess daytime somnolence. If this is severe during infancy, tracheostomy may be the only option. Older children should be initially worked up with a polysomnographic study. Treatment options range from CPAP therapy to midface advancement on the basis of the severity level.[46]

Cervical vertebral anomalies are found in 25% of patients with Crouzon syndrome, and all involve C2 and C3. Anomalies should be assessed before the patient undergoes anesthesia for any craniofacial procedure. Patients with Crouzon syndrome already have problematic airways as a result of their relatively inflexible necks; thus, cervical anomalies will compound the issue.[46]

Ophthalmologic abnormalities include hypertelorism, pronounced ocular proptosis, exotropia, exposure keratitis, poor vision, and optic atrophy. Luxation of the eye globes may be observed in some instances. Although emergency reduction followed by tarsorrhaphies may be necessary, some patients can luxate and reduce all by themselves.[131]

Mild to moderate conductive hearing deficit occurs in 55% of patients, and atresia of the external auditory meatus is found in 13%. Otitis media is common, and middle-ear disease may be related to the presence of cleft palate and associated eustachian tube dysfunction

Oral findings include lateral palatal swellings (50%), obligatory mouth breathing (32%), bifid uvula (9%), and cleft palate (9%). The maxillary dental arch is shortened and associated with a posterior crossbite, dental crowding, and ectopic eruption of the maxillary first molars. Anterior open bite, mandibular overjet, and crowding of the mandibular anterior teeth are also common.[46]

Pfeiffer Syndrome

Pfeiffer syndrome is characterized by craniosynostosis; midface deficiency; broad thumbs, great toes, or both; brachydactyly; variable soft-tissue syndactyly; and other anomalies (Figs. 30-12 through 30-15).[188,231] Three clinical subtypes have been proposed. Type 1 is representative of the aforementioned description. Type 2 is characterized by cloverleaf skull, elbow ankylosis or synostosis, and a cluster of unusual anomalies in addition to the Type 1 characteristics. Type 3 involves a very short cranial base, severe ocular proptosis, elbow ankylosis or synostosis, and an assortment of unusual anomalies. Types 2 and 3 also include an increased risk for neurodevelopmental difficulties. A favorable outcome can be achieved in some cases with aggressive medical and surgical management; however, normal outcome is not the rule, and neurodevelopmental outcome and life expectancy prognoses remain guarded in most cases.[44] Although these subtypes are useful, they have limited nosologic status, and some clinical overlap does occur.

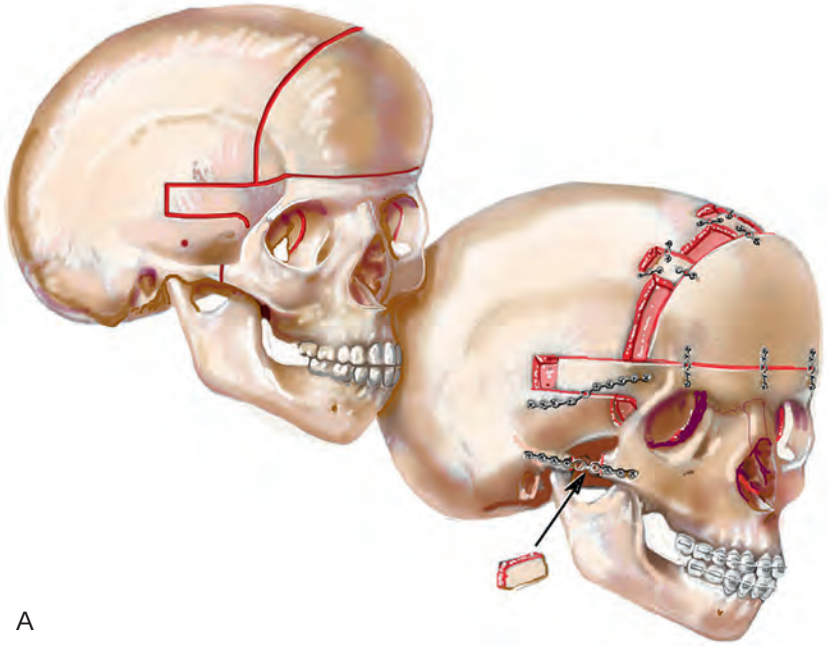
Craniofacial features include brachycephaly as a result of bicoronal synostosis, hypertelorism, ocular proptosis, and midface deficiency.[44] Distortion ventriculomegaly, progressive hydrocephalus, and cerebellar herniation are common. Progressive hydrocephalus is much more common among patients with Pfeiffer and Crouzon syndromes than it is among those with Apert syndrome. Gyral abnormalities have been observed in some cases.[44] Vertebral anomalies occur in 70% of patients, particularly at C2 and C3, but such anomalies may involve other cervical vertebrae as well as the lumbar vertebrae in some cases. [46] As in Apert and Crouzon syndrome, OSA can affect patients with Pfeiffer syndrome. For a further understanding of diagnosis and therapy, please refer to the relevant discussions in the sections of this chapter about the Apert and Crouzon syndromes. Other anomalies have been noted in some cases, including cardiovascular defects, gastrointestinal issues, and genitourinary anomalies.[44]

Saethre–Chotzen Syndrome

Saethre–Chotzen syndrome is characterized by a heterogeneous phenotypic presentation that involves craniosynostosis, a low-set frontal hairline, facial asymmetry, ptosis of the eyelids, a deviated nasal septum, brachydactyly, partial soft-tissue syndactyly of the second and third fingers, and various skeletal anomalies.[4,33,40,234] Craniosynostosis is facultative and not obligatory (i.e., some patients do not have it). When it is present, the time of onset and the degree of craniosynostosis can be quite variable. Brachycephaly is found most commonly, and this is followed by synostotic anterior plagiocephaly. Frontal bossing, parietal bossing, and occipital flattening can also accompany the cranial deformity. Large late-closing fontanelles and large parietal foramina have also been reported. Ptosis of the eyelids, hypertelorism, and strabismus are common. The ears may be low set, small, and posteriorly angulated, and mild conductive hearing loss is common. Oral anomalies include a narrow or highly arched palate, a cleft palate on occasion, malocclusion, and supernumerary teeth.[40]

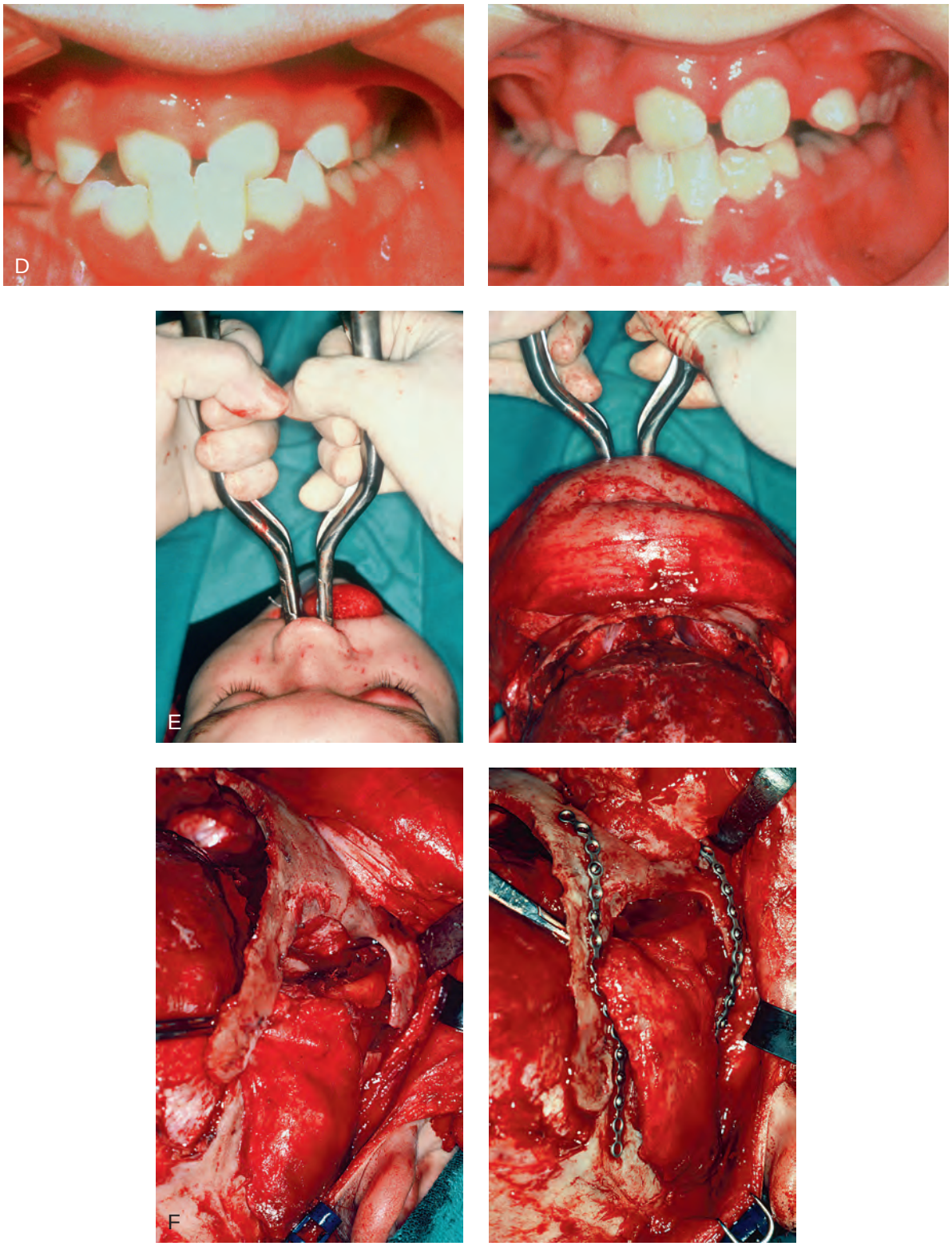
Text continued on p. 1226

*References 9, 12, 27, 51-53, 59, 61, 78, 83, 84, 90, 95, 98, 100, 118, 126, 127, 130, 163, 224, 225, 237, 238, 270, 271



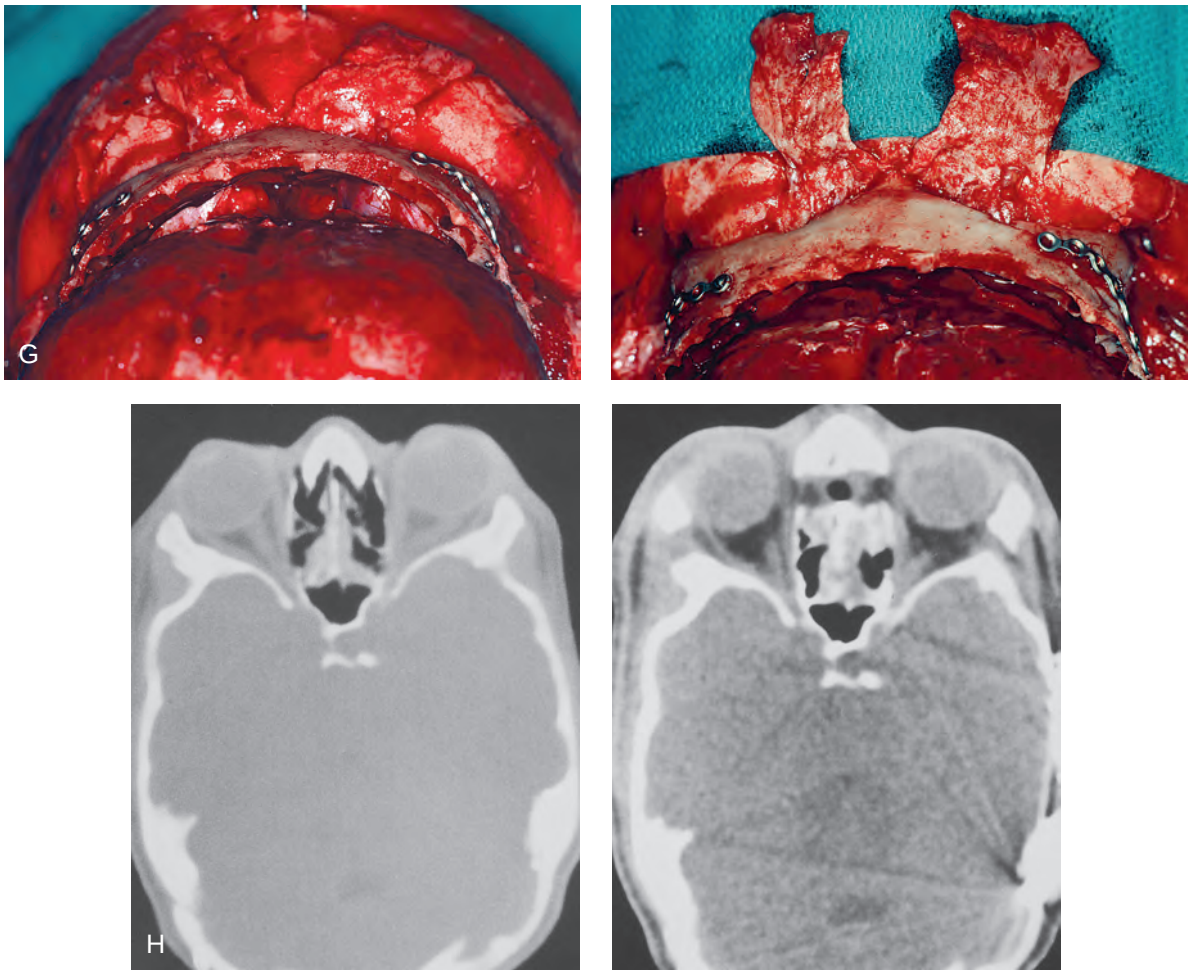
A

• **Figure 30-6** An 8-year-old boy who was born with Crouzon syndrome and who underwent a first-stage cranio–orbital procedure when he was 6 weeks old by a neurosurgeon who was working independently. He was referred to this surgeon and then underwent anterior cranial vault and monobloc osteotomies with advancement. **A,** Illustration of craniofacial morphology before and after anterior cranial vault and monobloc osteotomies with advancement. **B,** Profile views before and after reconstruction. **C,** Frontal views before and after anterior cranial vault and monobloc reconstruction.



• **Figure 30-6, cont'd** **D,** Occlusal views before and after reconstruction. Note the improvement but without a full correction of the malocclusion; this will await orthognathic correction during the patient's teenage years. **E,** Intraoperative view of disimpaction after monobloc osteotomy with the use of nasomaxillary forceps. **F,** Intraoperative view of monobloc showing tenon extension and zygomatic arch advancement before and after plate and screw fixation.

Continued



• **Figure 30-6, cont'd G,** After monobloc advancement and fixation, dead space in the retrofrontal region with communication into the nasal cavity can be seen. Pericranial flaps are elevated from under the surface of the scalp for the closure of cranio–nasal communication. **H,** Axial computed tomography scan views through the midorbits before and after reconstruction. Reconstruction resulted in increased intraorbital depth and decreased proptosis. *A, B, C ,D, E, H, From Posnick JC: Craniosynostosis: surgical management of the midface deformity. In Bell WH, ed:* Orthognathic and reconstructive surgery, *vol 3, Philadelphia, 1992, W.B. Saunders, p 1889.*

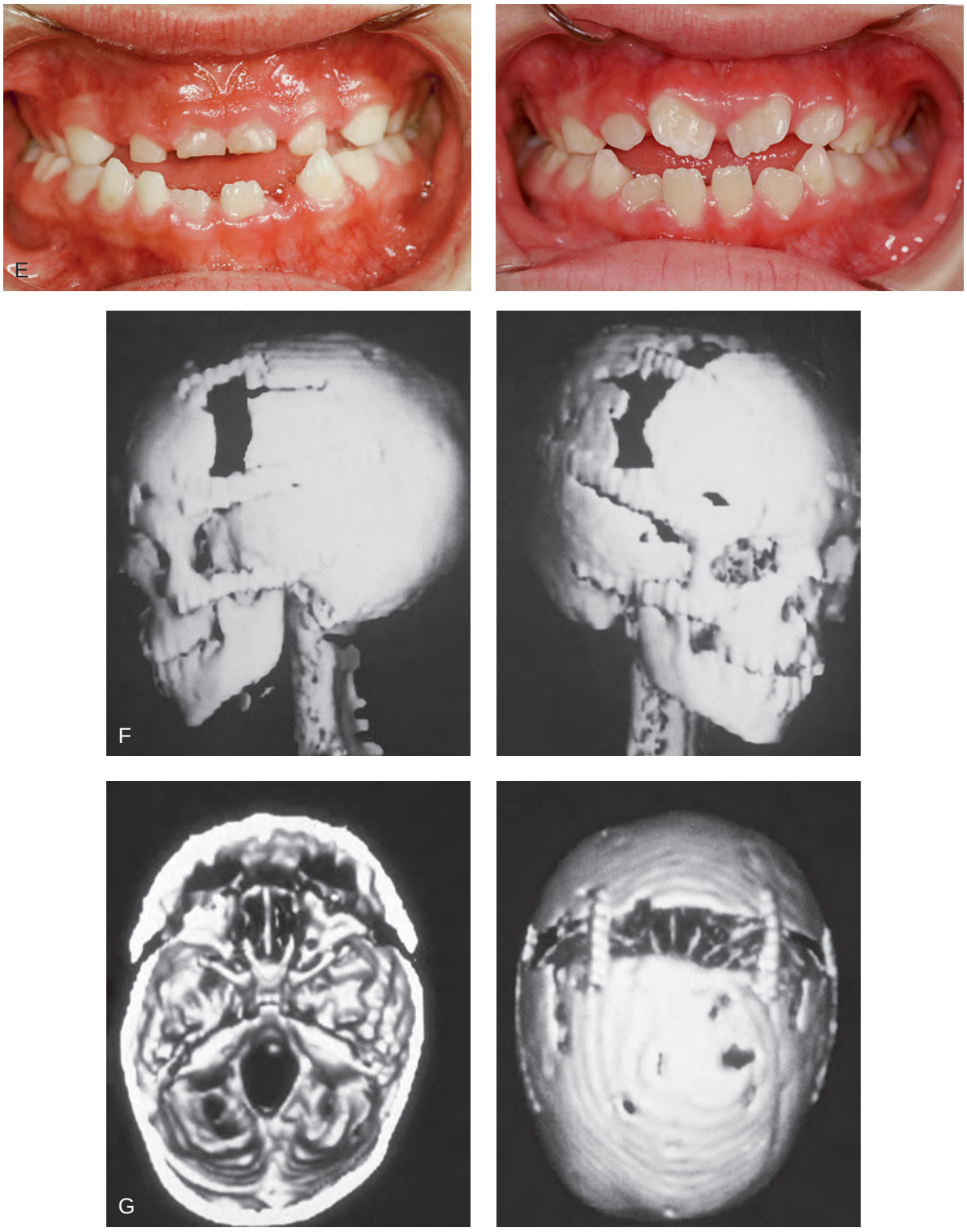
©Elsevier Inc.
Orthognathic Surgery; J.C. Posnick

• **Figure 30-7** A 6-year-old girl who was born with Crouzon syndrome and who underwent anterior cranial vault and monobloc osteotomies with advancement. **A,** Frontal facial views in repose before and after anterior cranial vault and monobloc reconstruction. **B,** Frontal views with smile before and after monobloc reconstruction.

Continued

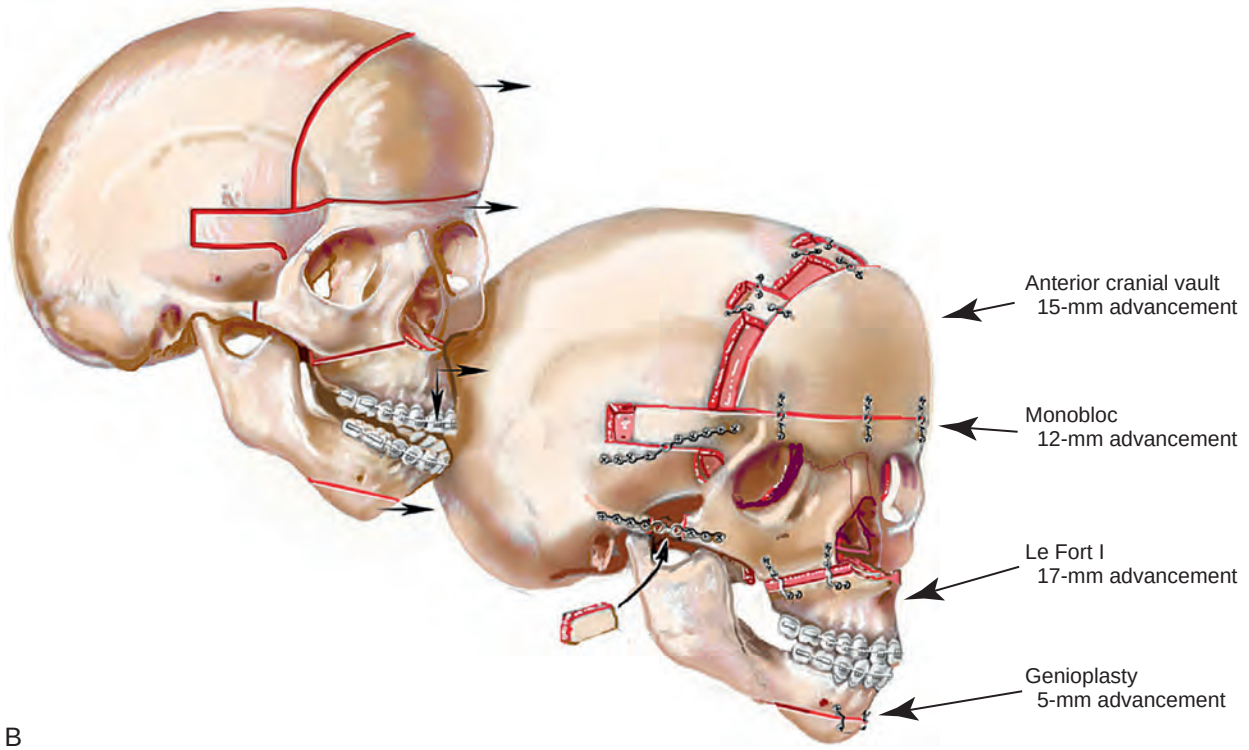
©Elsevier Inc.
Orthognathic Surgery; J.C. Posnick

• **Figure 30-7, cont'd C,** Profile views before and after monobloc reconstruction. **D,** Worm's-eye views before and after monobloc reconstruction.



• **Figure 30-7, cont'd** **E,** Occlusal views before and after reconstruction. Note the improvement but without the correction of the malocclusion; this will await orthognathic surgery during the patient's teenage years. **F** and **G**, Computed tomography scan views immediately after reconstruction that indicate advancement with increased volume in the anterior cranial vault and orbits. *A, C, D, From Posnick JC: Craniosynostosis: surgical management of the midface deformity. In Bell WH, ed:* Orthognathic and reconstructive surgery, *vol 3, Philadelphia, 1992, W.B. Saunders, p 1888.*

• **Figure 30-8** A child who was born with Crouzon syndrome and who underwent bilateral coronal suture release when she was 3 months old. Additional craniotomy and cranial vault reshaping were completed when she was 9 months old. When the patient was 2 years old, she underwent a Le Fort III midface osteotomy and forehead advancement procedure through an intracranial approach. She presented to this surgeon when she was 14 years old with residual deformity for which she underwent simultaneous anterior cranial vault, monobloc, Le Fort I, and chin osteotomies with differential advancements. **A,** Profile facial views at 1 year of age and at 2 years of age after Le Fort III and anterior cranial vault advancement.



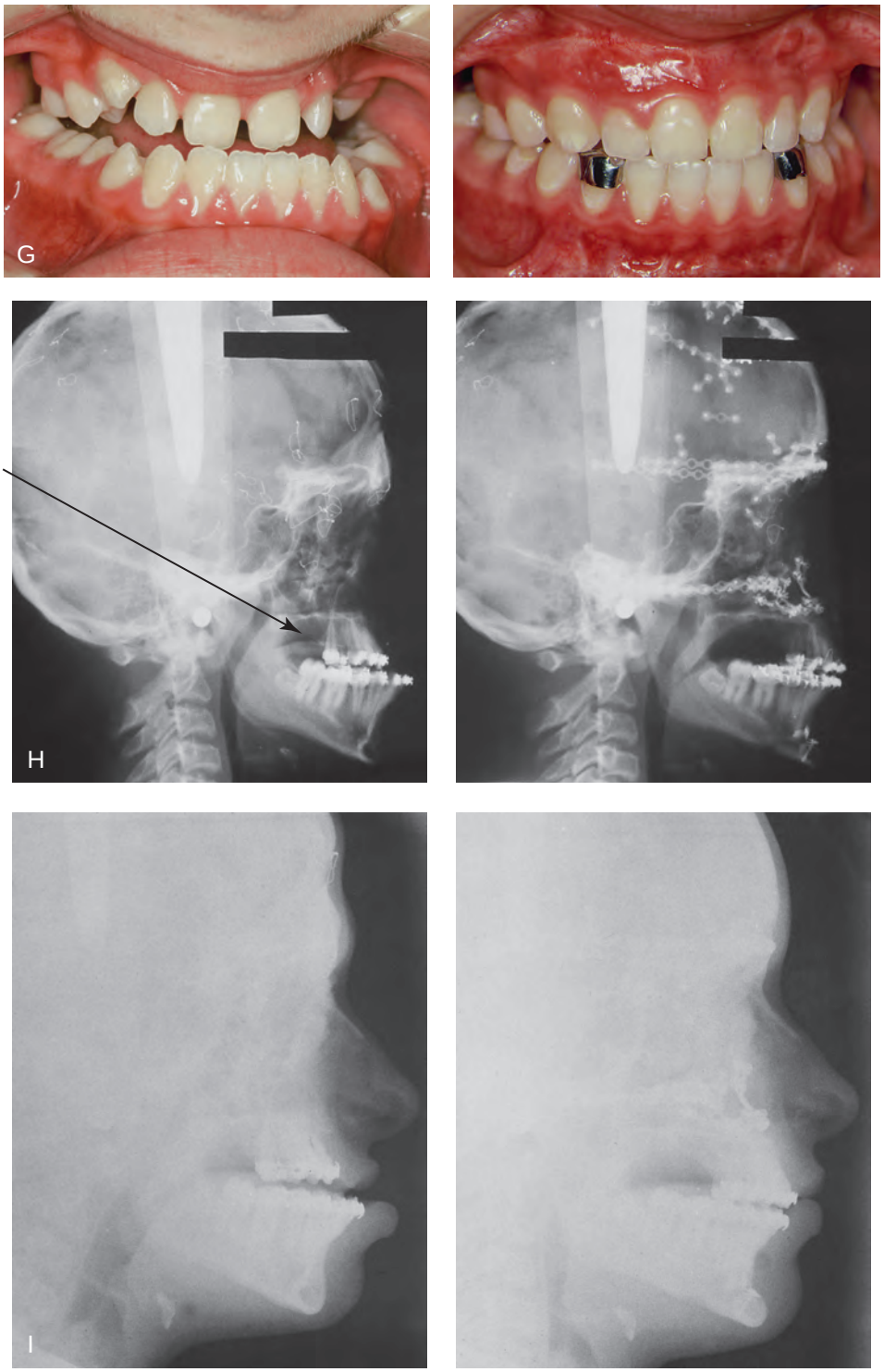
B

C

• **Figure 30-8, cont'd B,** Illustration of planned and completed anterior cranial vault, monobloc, Le Fort I, and chin osteotomies. **C,** Profile view and lateral cephalometric radiograph before surgery. Articulated dental casts after model planning that indicate that 17-mm advancement is required at the occlusal level. The amount of advancement required at the supraorbital ridge level was just 12 mm. Note the absence of permanent molars in maxilla; this is the result of the Le Fort III osteotomy carried out when the patient was 2 years old. The pterygomaxillary disjunction destroyed the developing teeth.

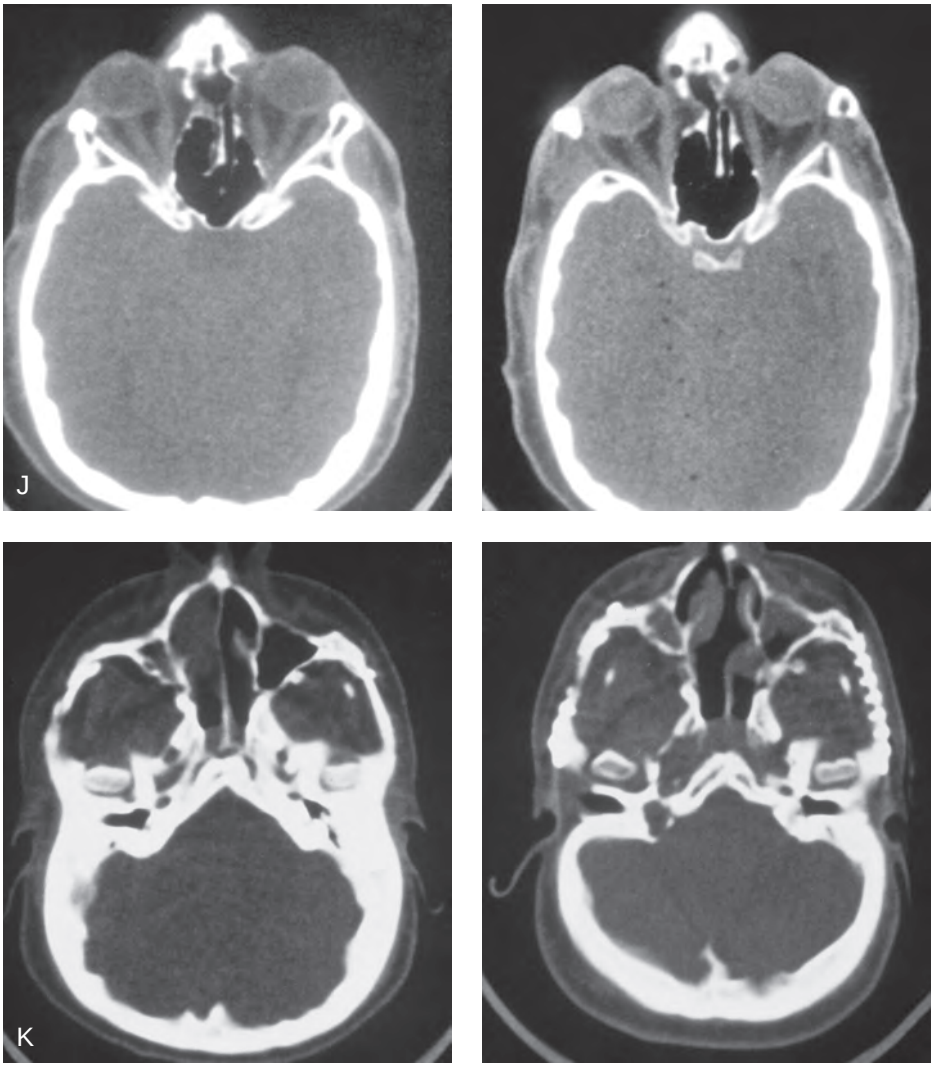
Continued

• **Figure 30-8, cont'd** **D,** Frontal facial views before and after reconstruction. **E,** Oblique facial views before and after reconstruction. **F,** Profile views before and after reconstruction.

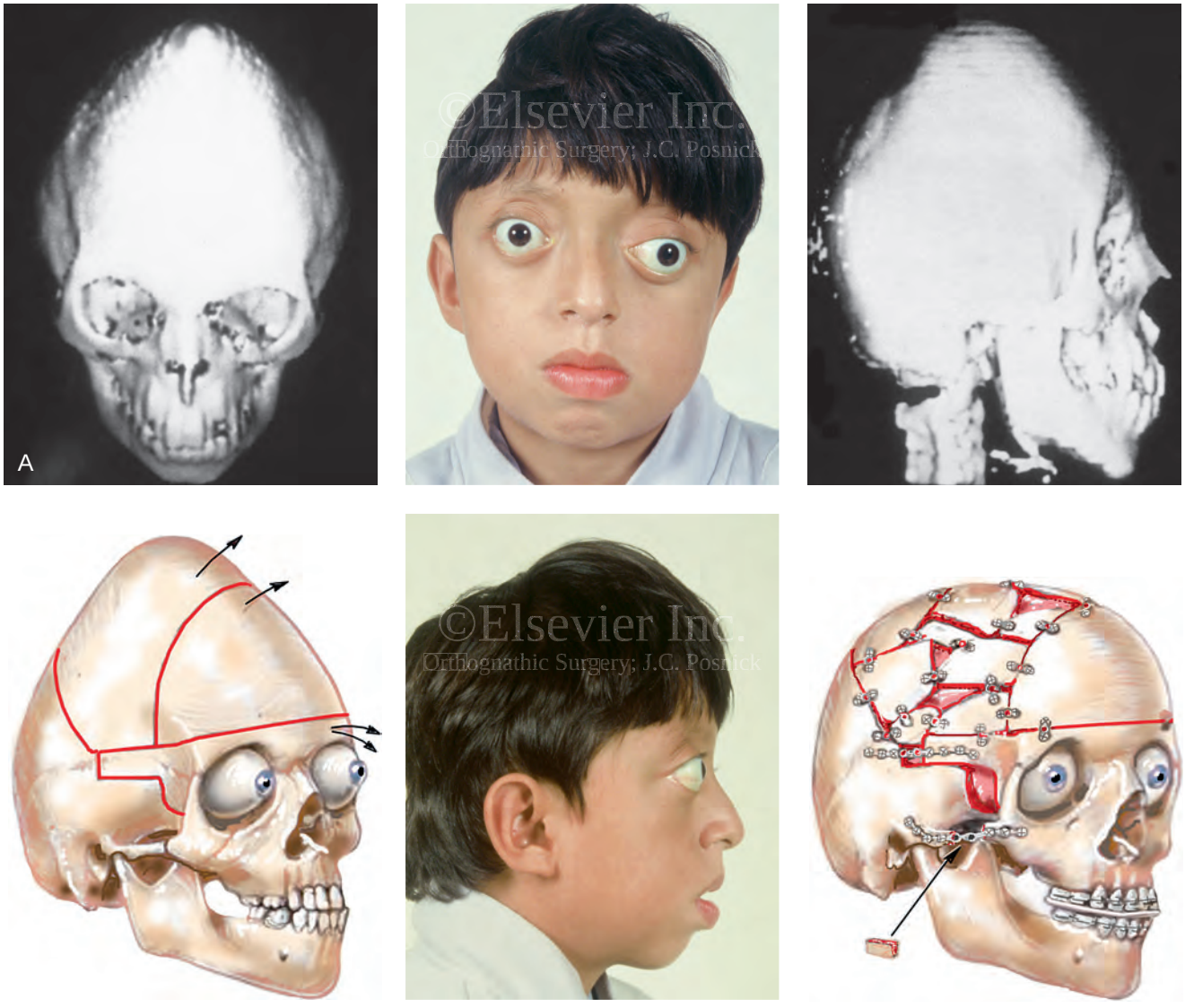


• **Figure 30-8, cont'd** **G,** Occlusal views before and after reconstruction and orthodontic treatment. **H,** Lateral cephalometric radiographs before and after reconstruction. **I,** Soft-tissue lateral cephalometric radiographs before and after reconstruction.

Continued

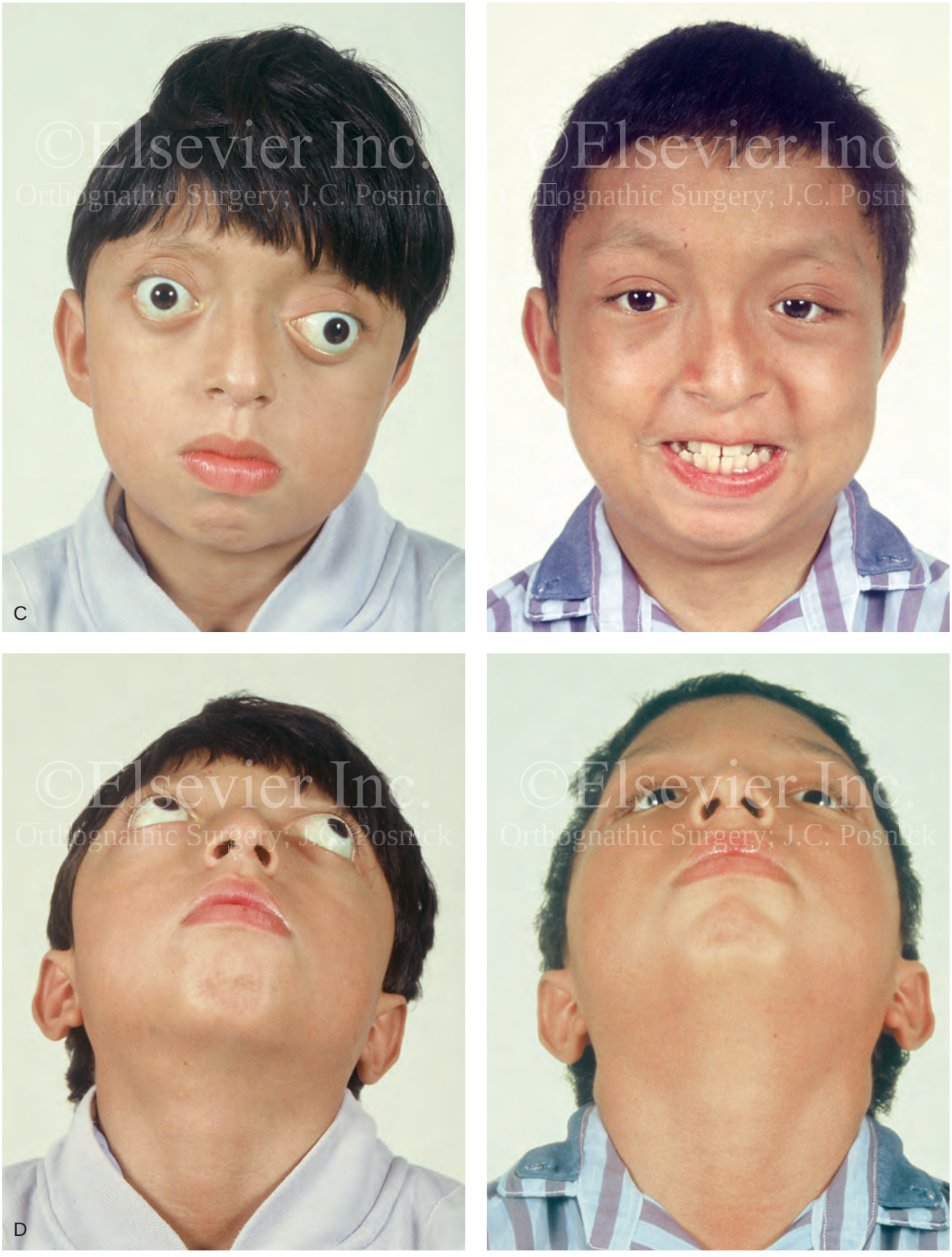


• **Figure 30-8, cont'd J,** Axial computed tomography scan views through the midorbits before and after reconstruction confirm relief of proptosis. **K,** Axial computed tomography scan views through the zygomatic arches before and after reconstruction. *A, B (right), D, G, H (left), I, J, From Posnick JC: Craniosynostosis: surgical management of the midface deformity. In Bell WH, ed:* Orthognathic and reconstructive surgery, *vol 3, Philadelphia, 1992, W.B. Saunders, p 1888.*

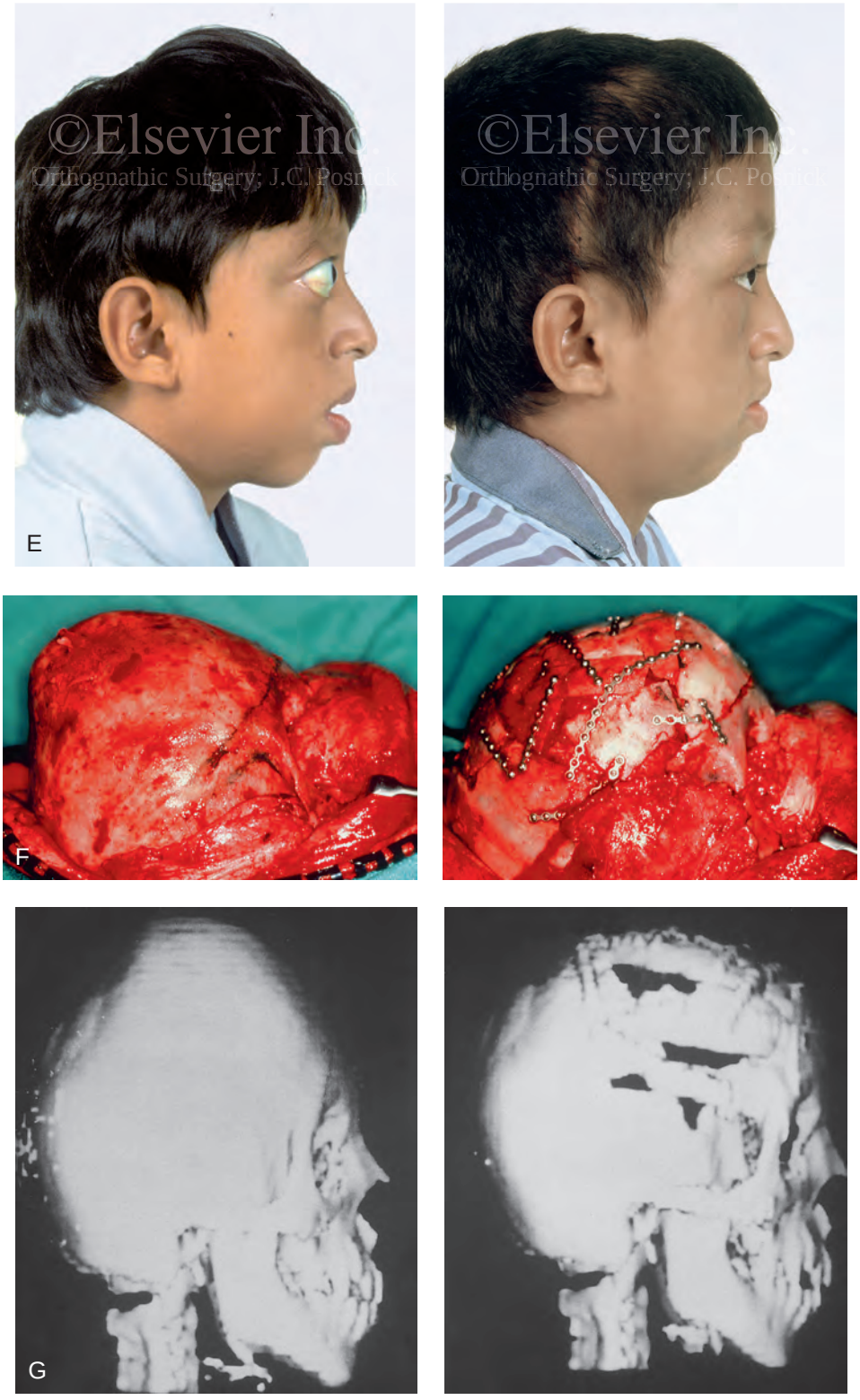


• **Figure 30-9** A 12-year-old boy with unrepaired Crouzon syndrome was referred to this surgeon for evaluation. He underwent total cranial vault and monobloc osteotomies with reshaping and advancement. **A,** Facial and computed tomography scan views before surgery. **B,** Profile facial view before surgery. Illustrations of the patient's craniofacial morphology with planned osteotomy locations indicated. A second illustration is shown after the proposed osteotomies were completed, with reshaping and advancement.

Continued



• **Figure 30-9, cont'd C,** Frontal facial views before and after reconstruction. **D,** Worm's-eye views before and after reconstruction.



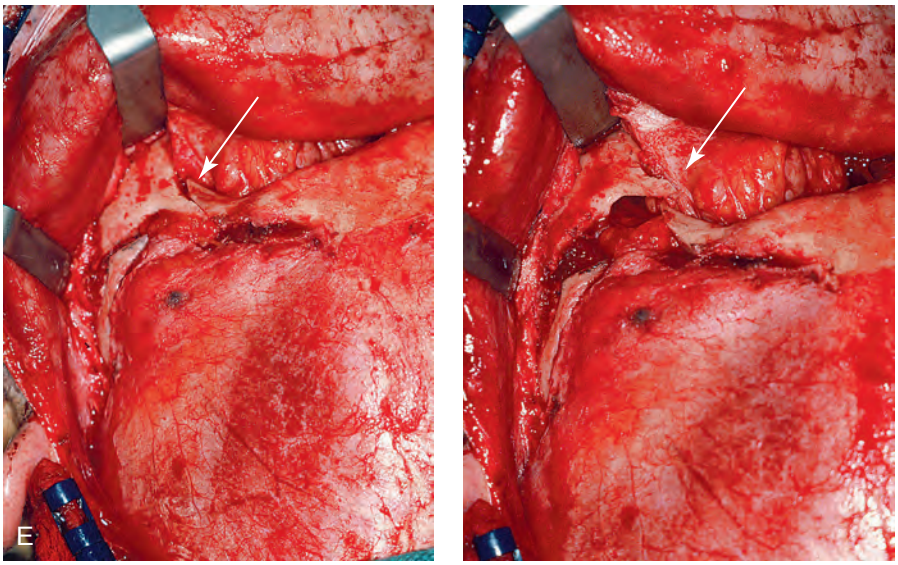
• **Figure 30-9, cont'd** **E,** Profile views before and after reconstruction. **F,** Intraoperative views before and after total cranial vault and monobloc osteotomy with reshaping and advancement. **G,** Computed tomography scan views before and immediately after reconstruction. *A (right), B (left, right), C (right), D (right), E (right), F, G, From Posnick JC: Craniosynostosis: surgical management of the midface deformity. In Bell WH, ed:* Orthognathic and reconstructive surgery, *vol 3, Philadelphia, 1992, W.B. Saunders, p 1888.*

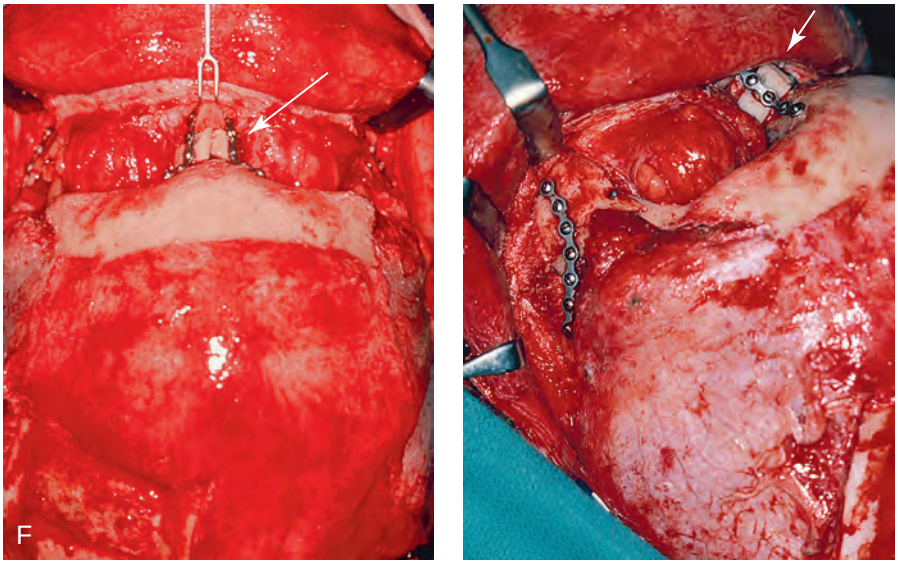
• **Figure 30-10** A 15-year-old girl with Crouzon syndrome that is characterized by mild to moderate midface deficiency with retrusion of the infraorbital rims, the zygomatic buttresses, and the maxilla. The midface hypoplasia results in increased scleral show, nasal obstruction, and malocclusion. The cranial vault and the upper orbits have normal morphology. Orthodontic camouflage treatment was in progress, including mandibular bicuspid extractions with retraction. The patient was referred to this surgeon for evaluation and treatment. She then underwent a Le Fort III extracranial osteotomy with advancement. **A,** Frontal facial views before and after reconstruction. **B,** Profile views before and after reconstruction.

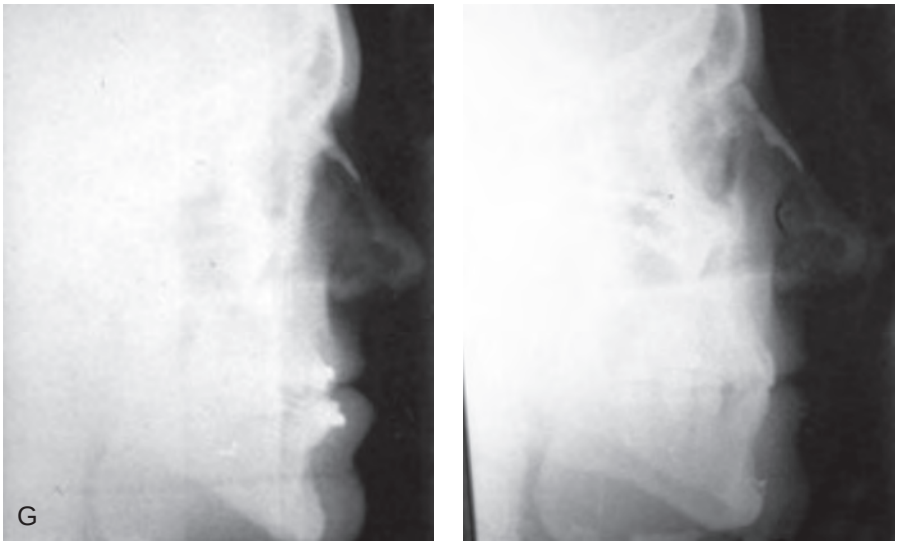
©Elsevier Inc.
Orthognathic Surgery, J.C. Posnick

• **Figure 30-10, cont'd C,** Worm's-eye views before and after reconstruction. **D,** Occlusal views before and after reconstruction.

Continued



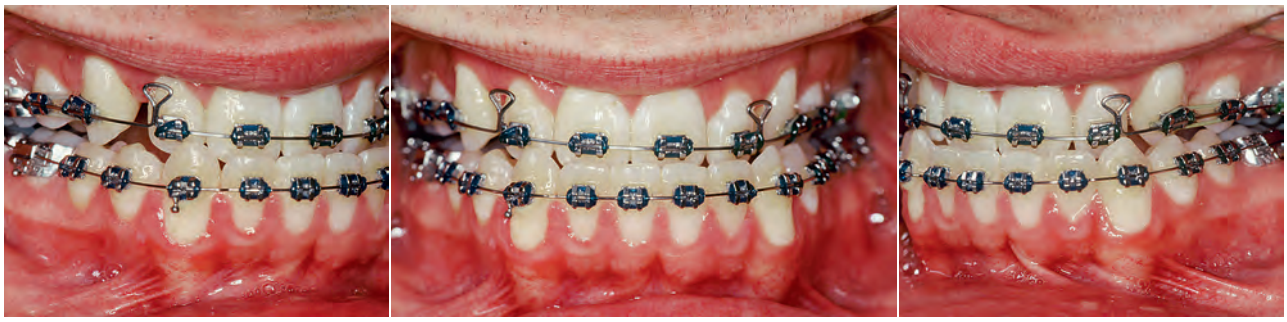




• **Figure 30-10, cont'd E,** Intraoperative view. Note the potential for unsightly step-off at the lateral orbital rim after advancement. **F,** Cranial bone graft is placed in the frontonasal region. Note the unavoidable lengthening of the nose and the potential for the flattening of the nasofrontal angle that occurs as part of the Le Fort III advancement. **G,** Lateral cephalometric radiographs before and after reconstruction. *A, B (right), C, D, E, F (left), G, from Posnick JC: Craniosynostosis: surgical management of the midface deformity. In Bell WH, ed:* Orthognathic and reconstructive surgery, *vol 3, Philadelphia, 1992, W.B. Saunders, p 1888.*

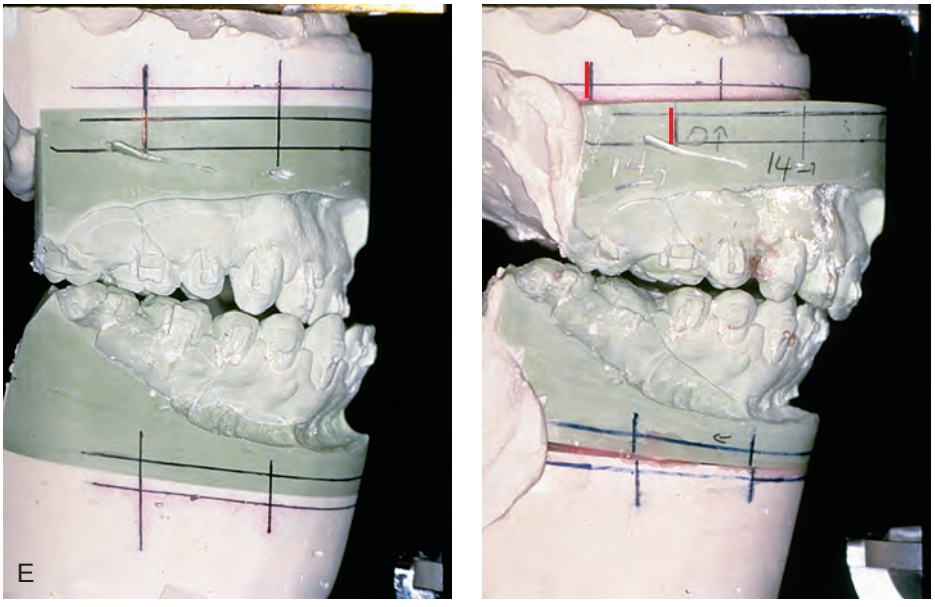
• **Figure 30-11** A 19-year-old boy who was born with Crouzon syndrome. When he was 11 years old, he underwent a Le Fort III osteotomy with advancement via an extracranial approach by another surgeon. He presented to this surgeon during his late teenage years with asymmetric and dystopic orbits, zygomatic asymmetry, a retrusive upper jaw, an asymmetric lower jaw, and a vertically long chin. He underwent a combined orthodontic (upper bicuspid extractions) and surgical approach. The procedures included Le Fort I osteotomy (horizontal advancement); bilateral sagittal split ramus osteotomies (correction of asymmetry); and osseous genioplasty (vertical reduction and horizontal advancement). He also underwent the reopening of a coronal scalp incision with the harvesting of split cranial grafts and the recontouring and augmentation of the orbits and the zygomas. **A,** Frontal views before and after reconstruction. **B,** Oblique facial views before and after reconstruction. **C,** Profile views before and after reconstruction.

Continued

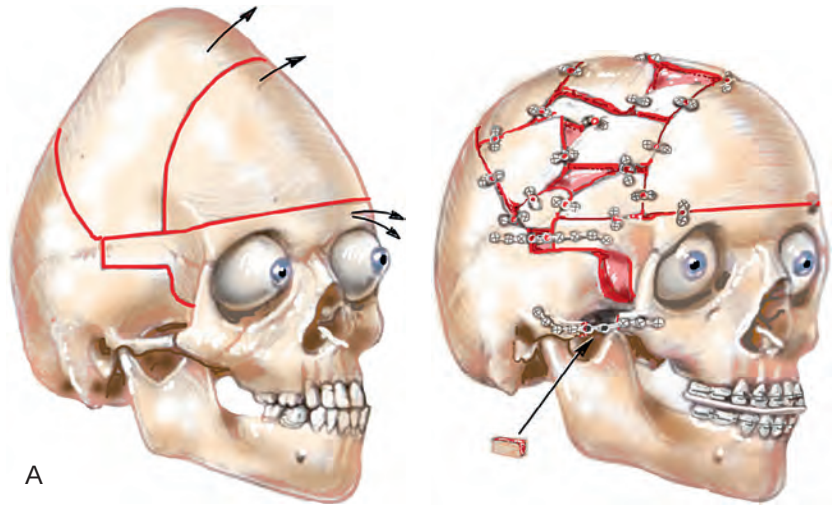


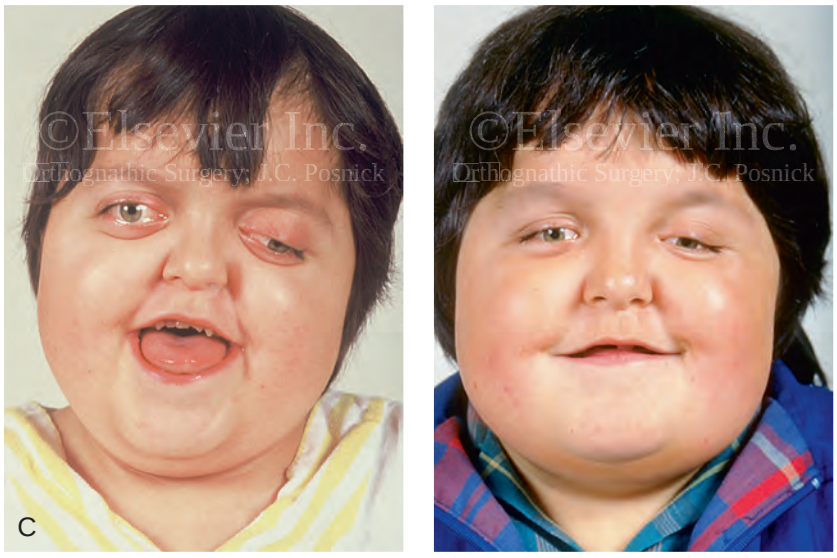
Pre surgery with maxillary bicuspid extractions

After treatment

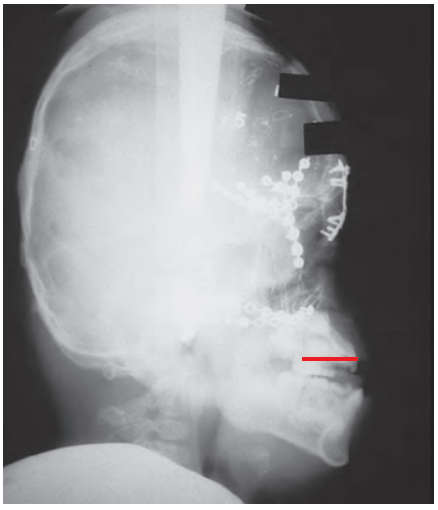


• **Figure 30-11, cont'd D,** Occlusal views before and after reconstruction. **E,** Articulated dental casts that indicate analytic model planning.





• **Figure 30-12** A 7-year-old boy who was born with Pfeiffer syndrome was referred for evaluation. He previously had undergone bilateral lateral canthal advancements when he was 3 months old; these were performed by a neurosurgeon who was working independently. The patient then required the placement of a ventriculoperitoneal shunt. He presented to this surgeon with total cranial vault dysplasia, orbital dystopia, and midface deficiency. He underwent cranial vault and monobloc osteotomies with reshaping and advancement. **A,** An illustration of the proposed cranial vault and monobloc osteotomies and reconstruction is also shown. **B,** Frontal facial views in repose before and after cranial vault and monobloc reconstruction. **C,** Frontal facial views with smile before and after reconstruction. *Continued*



• **Figure 30-12, cont'd D,** Profile views before and after reconstruction. **E,** Worm's-eye views before and after reconstruction. **F,** Lateral cephalometric radiographs before and after reconstruction. *A (left), B, D, from Posnick JC: Craniofacial dysostosis: staging of reconstruction and management of the midface deformity,* Neurosurg Clin North Am *2:683-702, 1991.*

©Elsevier Inc.
Orthognathic Surgery; J.C. Posnick

• **Figure 30-13** A 6-year-old girl who was born with Pfeiffer syndrome. She had undergone cranio–orbital reshaping earlier during her childhood. She presented to this surgeon with a constricted anterior cranial vault, orbital dystopia, and midface deficiency. She underwent anterior cranial vault and monobloc osteotomies with reshaping and advancement. **A,** Frontal facial views before and after reconstruction. **B,** Profile views before and after reconstruction. She still requires orthodontic treatment and orthognathic surgery, which are planned for her teenage years. **C,** Axial computed tomography scan views through the midorbits before and after reconstruction.

• **Figure 30-14** A 12-year-old girl who was born with Pfeiffer syndrome was referred to this surgeon for evaluation. She underwent anterior cranial vault and facial bipartition osteotomies with reshaping. She is shown before and after reconstruction. Quantitative analysis of the presenting deformity on the basis of computed tomography scan views confirmed the abnormal cranial vault length (85% of normal), medial orbital wall length (68% of normal), and zygomatic arch length (83% of normal) as well as the extent of globe protrusion (140% of normal). All measurements demonstrated horizontal deficiency in the upper face and the midface. In addition, the anterior interorbital distance (136% of normal), the mid-interorbital distance (129% of normal), the lateral orbital distance (121% of normal), and the intertemporal distance (132% of normal) all indicated a degree of upper-face hypertelorism. The patient then underwent anterior cranial vault and facial bipartition osteotomies for reconstruction. She achieved improved horizontal facial depth, and the upper face hypertelorism was also improved. **A,** Frontal facial views in repose before and after reconstruction. **B,** Frontal facial views with smile before and after reconstruction.

• **Figure 30-14, cont'd C,** Oblique facial views before and after reconstruction. **D,** Profile views before and after reconstruction.

Continued

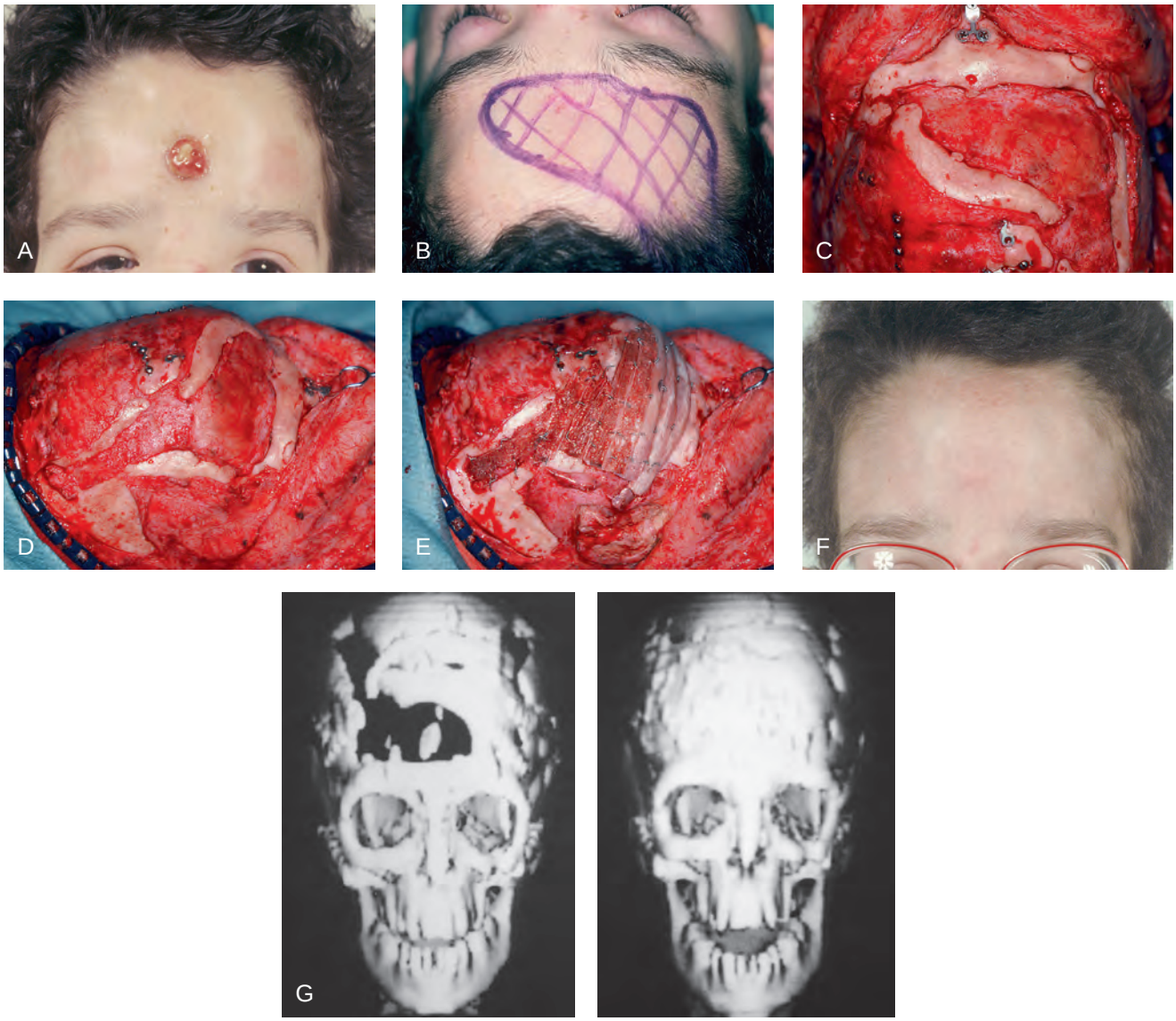
• **Figure 30-14, cont'd E,** Worm's-eye views before and after reconstruction. **F,** Occlusal views before and after reconstruction. Note that the occlusion is improved but that the patient will require orthognathic surgery and orthodontic treatment during her teenage years. *From Posnick JC, Waitzman A, Armstrong D, Pron G: Monobloc and facial bipartition osteotomies: quantitative assessment of presenting deformity and surgical results based on computed tomography scans,* J Oral Maxillofac Surg *53:358-367, 1995.*

Carpenter Syndrome

Carpenter syndrome is characterized by craniosynostosis (commonly, but not always); preaxial polydactyly of the feet; brachydactyly, clinodactyly, or both; congenital heart defects (these are seen in 33% of patients), such as ventricular septal defect, atrial septal defect, patent ductus arteriosus, pulmonary stenosis, and tetralogy of Fallot; short stature, with a height that is usually below the 25th centile; obesity; and mental deficiency in some cases.[28,39] Craniosynostosis involves the sagittal and lambdoid sutures first, with the coronal suture being the last to close. The head is usually broad, but it is variable in shape, and asymmetry may be a feature. Other features include dystopia canthorum, down-slanting palpebral fissures, epicanthal folds, low-set ears, and a short neck. The mandible may be hypoplastic, and the palate may be narrow or highly arched.[39]

Neurologic Aspects of Craniosynostosis

Generally, craniosynostosis tends to restrict intracranial volume. The earlier synostosis occurs, the more dramatic the effect on subsequent cranial growth and development. The later synostosis occurs, the less the effect on cranial growth and development. Growth restriction also worsens with increasing sutural involvement; complex craniosynostosis with two or more affected sutures demonstrates higher growth restriction as compared with single sutural involvement. There are two exceptions to this observation: scaphocephaly and Apert syndrome. With scaphocephaly,



• **Figure 30-15** A 12-year-old boy with Noonan syndrome. When he was 5 years old, he underwent bilateral total orbital osteotomies with medial translocation for the correction of orbital hypertelorism; this was carried out by another surgeon. When he was 12 years old, he presented to this surgeon with a retruded irregular forehead, shallow orbits, residual proptosis, and midface deficiency. He underwent anterior cranial vault and monobloc osteotomies with horizontal advancement. An occult cerebral spinal fluid leak with drainage through the nose occurred. Eventually, there was erosion through the mid-forehead skin and resorption of a segment of the anterior and lateral cranial vault. One year after the monobloc procedure, the patient was taken back to the operating room for the repair and closure of the cerebral spinal fluid fistula and a cranioplasty that consisted of autogenous split-rib grafts. The cranial vault reconstruction was successful, without further infection or long-term sequelae. **A,** Frontal view of the upper face 6 months after the initial surgery, with a cerebral spinal fluid fistula through central forehead. **B** and **C,** Intraoperative views of the anterior cranial vault documenting the region of bone loss. **D,** Intraoperative lateral view that shows the area of bone loss. **E,** Intraoperative lateral view after autogenous split-rib graft reconstruction. **F,** Frontal view of the upper face 1 year after dura repair and the split-rib reconstruction of the skull defects. **G,** Computed tomography scan views before and after successful split-rib graft cranioplasty.

mechanical forces produce a large head circumference; with Apert syndrome, brain weights have been shown to be above the 97th centile, regardless of age.[46,48]

Mental deficiency is correlated with the number of fused sutures, and it occurs more frequently with two or more sutural involvements than with single-suture synostosis. In addition, mental deficiency occurs more commonly with coronal synostosis than with other single-suture craniosynostoses. With single-suture craniosynostoses (other than coronal), mental deficiency may be more attributed to a brain malformation rather than to growth restriction. This may occur in patients with trigonocephaly and even in some cases of sagittal synostosis.[25]

In the section about Apert syndrome, a distinction was made between benign megalencephaly or distortion megalencephaly and progressive hydrocephalus. The mechanism of hydrocephalus may vary. Although communicating hydrocephalus with obstruction at the level of the basal cisterns is most common, aqueductal stenosis is fairly frequent. Chronic tonsillar herniation occurs in 73% of

patients with Crouzon syndrome as compared with only 1.9% of patients with Apert syndrome.[62] Premature synostosis of the lambdoid suture may be responsible for this finding. It has also been suggested that the obstruction of the venous drainage of the brain may play a role by narrowing the jugular foramina. Some patients are able to compensate well for their hydrocephalus.[25]

Clinicians must maintain a high index of suspicion for hydrocephalus in all patients with craniosynostosis, particularly those with complex or syndromic types.[77,91,108,170,174] Among the syndromic craniosynostoses, shunted hydrocephalus is found in 26.6% of patients with Crouzon syndrome and in 27.8% of patients with Pfeiffer syndrome but in only 6.5% to 8% of patients with Apert syndrome.[46] Increased intracranial pressure in craniosynostosis may be the result of a mismatch in cranial volume to brain growth or of hydrocephalus. Increased intracranial pressure is found most commonly in patients with multiple suture synostoses.[9,26,49,58,122,191,222,226,227-230,245,266] However, recent studies have indicated that increased pressure may also occur in patients with single-suture synostosis. In one report, 3 out of 18 children with scaphocephaly had increased intracranial pressure during 12- to 24-hour recordings.[265]

When pressure is low grade, clinical symptoms can be subtle or absent. Headache is the classic symptom that is associated with increased intracranial pressure of any cause. However, children seem to experience this inconsistently, and infants are not developed enough to communicate their symptoms. The most common physical finding is papilledema, although this may not be as apparent during infancy or early childhood, when bulging may occur at the anterior fontanel instead. Longstanding papilledema may result in optic atrophy and eventual blindness.[25]

Regardless of the cause, increased intracranial pressure tends to rise during rapid eye movement sleep, presumably as a result of increased cerebral blood flow. Indeed, plateaus of increased intracranial pressure that last approximately 10 to 120 minutes have been observed during sleep. Conversely, intracranial pressure may be normal while the patient is awake or between periods of rapid eye movement sleep. Thus, the diagnosis of increased intracranial pressure may be missed by a single lumbar puncture measurement. Reasonably safe ways of long-term, direct measurement of increased intracranial pressure are possible with the use of an epidural pressure transducer to record pressure for at least 12 hours. In this way, the significance of increased intracranial pressure as a contributing factor to mental deficiency can be examined.[25]

With these considerations in mind, the neurologic assessment of patients with craniosynostosis can be performed as follows. The medical history should concentrate on genetic factors as well as the on child's development. The physical examination should include a careful search for associated anomalies, with scrupulous attention paid to each of the cranial nerves (and particularly to cranial nerves II and VIII). Head circumference should be plotted on the standard growth curve. Investigations should include a skull radiograph, head computed tomography (CT) scan, brain magnetic resonance imaging, initial assessment for hydrocephalus, and formal assessment for hearing. An ophthalmologic examination is mandatory. Depending on the findings, a 12- to 24-hour pressure recording should be considered. Whenever possible, a formal developmental or newborn psychologic battery should be administered before surgery. The family should be assessed for psychosocial problems, especially when a complicated course of surgical interventions is contemplated. Follow up by a pediatrician or pediatric neurologist at regular intervals is essential. Assessments at monthly intervals for the first 12 months, every 3 months during the second year, and every 6 months until 6 years of age seem appropriate.[25]

Effects of Midface Deficiency on the Upper Airway

All newborn infants are obligate nasal breathers. Most infants who are born with syndromic craniosynostosis have moderate to severe hypoplasia of the midface. They have diminished nasal, nasopharyngeal, and retropalatal airspaces with resulting increased upper airway obstruction. The affected child is thus forced to breath solely through the mouth. For a newborn infant to ingest food through the mouth, he or she must suck from a nipple to achieve negative pressure and have an intact swallowing mechanism. The neonate with severe midface hypoplasia is unable to accomplish this task while breathing adequately through the nose at the same time. Complicating this clinical picture may be an abnormal palate, enlarged tonsils, and enlarged adenoids. Thus, the compromised infant expends significant energy on respiration, possibly pushing the child into a catabolic state (i.e., a negative nitrogen balance). Failure to thrive can occur in the absence of nutritional support (e.g., nasogastric tube, feeding gastrostomy). It is also essential to establish a secure airway, typically via a tracheostomy, which may require air tubing at night. It is also essential to obtain evaluations from a pediatrician, a pulmonologist, a pediatric otolaryngologist, and a feeding specialist with craniofacial experience to help distinguish minor feeding difficulties from those that require more aggressive treatment.

Sleep apnea may be central, obstructive, or of mixed origin; proper workup and assessment are crucial to the establishment of the correct diagnosis and treatment.* Central apnea may result from intracranial hypertension. If so, the condition should improve upon brain decompression by appropriate cranio–orbital or posterior cranial vault expansion or when effective shunting of the hydrocephalus is accomplished.

OSA has already been addressed in the sections about the Apert, Crouzon, and Pfeiffer syndromes.[105,138,149,190]

*References 2, 35, 79, 92, 101, 105, 106, 114, 135, 141, 162, 172, 173, 175, 181, 185, 187, 190, 275, 284

OSA in a child who is affected by one of these conditions is frequently treated with tracheostomy. It may also be treated by adenoidectomy, tonsillectomy, midface advancement, or CPAP. If left untreated or ineffectively treated, OSA will result in disabilities that include failure to thrive, recurrent upper respiratory infection, cognitive dysfunction, developmental delay, cor pulmonale, and sudden death.[173] Midface hypoplasia in the setting of a craniosynostosis syndrome is often a primary cause of OSA. If feasible, surgical midface advancement is the preferred biologic treatment approach; even with such treatment, some patients will still require a tracheostomy or CPAP therapy.

Clinical signs of OSA may include loud snoring; observed apnea during sleep; excessive perspiration during sleep (except in patients with Apert syndrome, as described previously in this chapter); difficulty during daytime breathing; and daytime somnolence. Polysomnography is essential for accurately diagnosing the type of obstruction (i.e., central, obstructive, or mixed). In the pediatric age group, apnea is defined as the absence of airflow for more than two breaths, whereas hypopnea is defined as a reduction of at least 50% of nasal flow signal amplitude. The apnea–hypopnea index (AHI) is the number of obstructive apneas in combination with hypopneas (followed by desaturations) per hour. The oxygen–desaturation index is the number of desaturations (i.e., ≥4% decrease with respect to the baseline) per hour. A score of less than 1.0 in a child is considered normal. The severity of the condition in children is defined as follows: between 1 and 5 events per hour is considered mild OSA; 6 to 25 events per hour constitute moderate OSA; and more than 25 events per hour results in severe OSA. In the child with a craniosynostosis syndrome and a midface deficiency with documented OSA, neither tonsillectomy nor adenoidectomy generally provides sufficient opening of the upper airway.

Al-Saleh and colleagues completed a retrospective review of children with syndromal craniosynostosis who were referred to the Hospital for Sick Children in Toronto, Canada, between 1996 and 2008 for initial polysomnography to rule out sleep-related disordered breathing.[2] The research confirmed a high prevalence of this type of breathing in children with Crouzon, Apert, Pfeiffer, and Saethre–Chotzen syndromes (N = 35). Despite a spectrum of surgical and non-surgical interventions having been carried out, the complete resolution of the sleep-related disordered breathing could not be routinely achieved.

Ishii and colleagues used lateral cephalometric radiographs to evaluate the nasopharyngeal airway after Le Fort III advancement in patients with Apert or Crouzon syndrome and OSA.[114] The researchers documented improvement in the nasopharyngeal airway space after successful midface advancement surgery. Arnaud and colleagues reviewed respiratory improvement in patients with syndromic craniosynostosis and OSA after monobloc (MB) advancement.[8] Eighty-eight percent of patients (n = 16) showed improvement as measured by oxygen level during sleep; 4 of 6 patients underwent successful tracheostomy decannulation. In one patient, a tracheostomy was required 6 months after midface advancement for severe and recurrent OSA.

Twenty-four months after MB advancement, Witherow and colleagues documented the significant improvement of airway obstruction on polysomnography in all patients who had been diagnosed with Apert, Crouzon, or Pfeiffer syndrome.[280] Before MB advancement, 14 patients were managed with tracheostomy or CPAP therapy for severe OSA. Of these 14 patients who were undergoing MB advancement for severe OSA, only 43% (n = 6) had resolution of their OSA; 8 of 14 patients (57%) remained dependent on tracheostomy or CPAP despite MB advancement.

Nelson and colleagues studied 18 patients with craniosynostosis syndromes who also had coronal synostosis and midface deficiency.[172] Eighty-three percent (16 of 18 patients) were treated with tracheostomy or CPAP for known OSA before midface advancement. After the midface advancement procedures, five of the tracheostomy patients were decannulated; in six others, CPAP was no longer required. This represents a 73% (11 of 15 patients) initial success rate for the relief of OSA. The mean postsurgical follow up was 3 years.

In patients with syndromic craniosynostosis who were undergoing midface advancement, the lack of universal success for the relief of OSA could result from a combination of factors, including inadequate midface advancement; midface relapse after initial advancement; other anatomic sites or conditions of airway obstruction not addressed by midface advancement (e.g., septum, turbinates, soft palate, tonsils, adenoids, or mandible; tracheomalacia); a central sleep apnea component; or inadequate contraction of the pharyngeal dilator muscles during sleep. The anatomy of the upper airway is different for each syndrome and also widely variable within each syndrome. Patients with a craniosynostosis syndrome and OSA who are non-responders to an effective midface advancement may have pharyngeal wall collapse caused by compromised pharyngeal dilator muscle function during sleep. Although a tendency for airway collapse can be overcome by midface advancement, the patient must also have the sufficient ability to control, contract, and maintain the tone of the pharyngeal muscles while asleep.

Morphologic Considerations

Examination of the patient's entire craniofacial region should be meticulous and systematic. The skeleton and the soft tissues are assessed in a standard way to identify all normal and abnormal anatomy.[65,66,68,202,205,206,211,212,216,217,272,273] Specific findings tend to occur in particular malformations, but each patient is unique. The achievement of symmetry, normal proportions, and the reconstruction of specific aesthetic units is essential to forming an unobtrusive face in a child who is born with one of the craniosynostosis syndromes.

Fronto–Forehead Aesthetic Unit

The fronto–forehead region is dysmorphic in an infant with a craniosynostosis syndrome. Establishing the normal position of the forehead is critical for overall facial symmetry and balance. The forehead may be considered as two separate aesthetic components: the supraorbital ridge/lateral orbital rim region and the superior forehead. The supraorbital ridge/lateral orbital rim unit includes the nasofrontal process and the supraorbital rim extending inferiorly down each frontozygomatic suture toward the infraorbital rim and posteriorly along each temporoparietal region. The shape and position of the supraorbital ridge/lateral orbital rim region is a key element of upper facial aesthetics. In a normal forehead, at the level of the nasofrontal suture, an angle that ranges from 90 to 110 degrees is formed by the supraorbital ridge and the nasal bones when the face is viewed in profile. In addition, the eyebrows, which overlie the supraorbital ridge, should be anterior to the cornea. When the supraorbital ridge is viewed from above, the rim should arc posteriorly to achieve a gentle 90-degree angle at the temporal fossa with a center point of the arc at the level of each frontozygomatic suture. The superior forehead component, which is about 1.0 to 1.5 cm up the supraorbital rim, should have a gentle posterior curve of about 60 degrees, thus leveling out at the coronal suture region when the face is viewed in profile.

Posterior Cranial Vault Aesthetic Unit

The symmetry, form, and adequate intracranial volume of the posterior cranial vault are closely linked. Posterior cranial vault flattening may result from unilateral or bilateral lambdoid synostosis, which is rare; previous craniectomy with reossification in a dysmorphic flat shape, which is frequent; or postural molding as a result of intrauterine positioning or repetitive supine sleep positioning. A short anteroposterior cephalic length may be misinterpreted as an anterior cranial vault (forehead) problem when the occipitoparietal (posterior) skull represents the primary region of the abnormality. Careful examination of the entire cranial vault is essential to defining the dysmorphic region so that, when indicated, appropriate cranial vault expansion and reshaping may be carried out.

Orbito–Naso–Zygomatic Aesthetic Unit

In the craniosynostosis syndromes, the orbito–naso–zygomatic regional abnormality is a reflection of the cranial base malformation. In patients with Crouzon syndrome, when bilateral coronal suture synostosis is combined with skull base and midface deficiency, the orbito–naso–zygomatic region will be dysmorphic and consistent with a short anteroposterior and wide transverse anterior cranial base. In patients with Apert syndrome, the nasal bones, orbits, and zygomas—like the anterior cranial base—are transversely wide as a result of the anterolateral bulging of the temporal lobes of the brain and horizontally short (i.e., retruded); this results in a shallow, hyperteloric, "reverse curved" upper midface. Surgically advancing the midface without simultaneously addressing the increased transverse width and the "reverse curve" will not adequately correct the dysmorphology.

Maxillary/Nasal Base Aesthetic Unit

In the patient with a craniosynostosis syndrome and a midface deficiency, the upper anterior face is vertically short (i.e., from the nasion to the maxillary incisor), and there is a lack of horizontal anteroposterior projection. These findings may be confirmed through cephalometric analysis, which indicates the presence of a sella–nasion–A point angle that is less than the mean value as well as a short upper anterior facial height (i.e., from the nasion to the anterior nasal spine). The width of the maxilla in the dentoalveolar region is generally constricted, with a highly arched palate. To normalize the maxillary/nasal base region, multidirectional surgical expansion and reshaping are generally required. The abnormal maxillary lip-to-tooth relationship and the occlusion are improved through Le Fort I segmental osteotomies and orthodontic treatment as part of the staged reconstruction. The mandible and the chin are frequently secondarily involved and will benefit from surgical repositioning as part of the orthognathic correction.

Surgical Approach to Craniosynostosis Syndromes

Historical Perspectives

As early as 1890, Lannelogue[134] and then Lane[133] described a surgical approach to the treatment of craniosynostosis. Lannelogue's aim was to remove the fused suture (i.e., strip craniectomy) in the hope of controlling the problem of brain compression within a congenitally small cranial vault. By the turn of the century, Harvey Cushing, who was the most prominent neurosurgeon of his day, suggested that surgical intervention for the problem of craniosynostosis was misdirected and that more attention should instead be given to the schooling of these children.[55] Shillito disagreed with Cushing and enthusiastically supported the concept of surgical intervention to improve the outlook for these children.[243] He believed that the linear "strip" craniectomy of the fused sutures would "release" the skull and allow the cranium to reshape itself and continue to grow in a normal and symmetric fashion. The strip craniectomy procedures were supposed to allow for a new suture line at the site of the previous synostosis. With the realization that this goal was not biologically feasible, attempts were made to remove portions of the cranial vault surgically and leave large open areas or to use the removed segments as free grafts to refashion the cranial vault shape. Problems with these methods included uncontrolled postoperative skull molding,

reossification into dysmorphic configurations, and the occurrence of large residual skull defects.

The concept of the simultaneous release of the fused suture in combination with more meticulous cranial vault reshaping in infants was initially suggested by Rougerie and colleagues[232] and then refined by Hoffman and Mohr in 1976 for children who were born with unilateral coronal synostosis.[107] In 1977, Whitaker and colleagues proposed a more formal anterior cranial vault and orbital reshaping procedure for unilateral coronal synostosis.[279] Marchac and Renier published their experience with a "floating forehead" technique to manage craniosynostosis during infancy; this consisted of simultaneous unilateral coronal and bilateral coronal suture release and forehead and upper orbital osteotomies with advancement.[144] Unfortunately, the "floating forehead" technique resulted in unpredictable reossification of the open cranial vault areas.[145] In addition, bitemporal constrictions and bulging of the skull concavities were frequent occurrences as reossification occurred. In any case, the hope for midface growth did not materialize.

During the 1950s, Gillies and Harrison reported their experience with an extracranial Le Fort III osteotomy to improve the anterior projection of the midface in an adult with Crouzon syndrome. The initial Gillies procedure was actually carried out in 1942.[89] Gillies mobilized the midface via a variety of osteotomies performed through skin incisions directly over each osteotomy site. The midface was mobilized and advanced, and intermaxillary fixation was applied. After the removal of the intermaxillary fixation (i.e., 2 weeks after the operation) a metal cast-cap dental splint was attached to a plaster headcap and maintained for 3 weeks. There was no evidence that Gillies used bone grafts to bridge the surgical gaps that were created. The early enthusiasm for this technique later turned to discouragement when the patient's facial skeleton relapsed to its preoperative status. Relapse occurred at the maxillary incisor level and also resulted in ocular proptosis.

Dr. Tessier was aware of the previous work of Gillies and its accompanying difficulties. In 1967, Tessier described a new intracranial–cranial base approach to the management of Crouzon syndrome.[242] This work was first presented in France at a meeting in Montpellier in 1966 and then again the following year at the International Plastic Surgery Meeting in Rome.[252-254] Tessier's landmark presentations and publications were the beginning of modern craniofacial surgery (see chapter 2). [252-264] To overcome the earlier problems encountered by Gillies, Tessier developed an innovative basic surgical approach that included new locations for the Le Fort III osteotomy, a combined intracranial–extracranial (cranial base) approach, the use of a coronal skin incision to expose the upper facial bones, and the use of fresh autogenous bone graft. He also applied an external fixation device to help maintain bony stability until healing had occurred.

In 1971, Tessier described a single-stage frontofacial advancement in which the fronto–orbital bandeau was advanced as a separate element in conjunction with the Le Fort III complex below and the frontal bones above.[256] Seven years later, Ortiz-Monasterio and colleagues refined the MB osteotomy to advance the orbits and midface as one unit; this was combined with frontal bone (anterior cranial vault) repositioning to correct the upper and midface deficiency associated with Crouzon syndrome.[178,179] In 1979, Van der Meulen described the medial fasciotomy for the correction of midline facial clefting.[267] Van der Meulen split the MB osteotomy vertically in the midline, removed central nasal and ethmoid bone, and then moved the two halves of the facial skeleton together for the correction of the orbital hypertelorism. To correct the midface dysplasia and the associated orbital hypertelorism in patients with Apert syndrome, Tessier refined the vertical splitting and reshaping of the midline split MB segments, thereby correcting the midface deformity in three dimensions via a procedure that is now known as *facial bipartition.* Wolfe and colleagues,[281,282] Kawamoto,[19] and Posnick and colleagues[198,207,208,220] have all independently documented the advantages of Tessier's facial bipartition technique for the correction of the upper and midface abnormalities associated with Apert syndrome. The widespread use of autogenous cranial bone grafting has virtually eliminated the need for rib and hip grafts when bone replacement is required during cranio–orbito–zygomatic procedures.[213] This represents another of Tessier's contributions to craniofacial surgery.[255]

In 1968, Luhr introduced the use of small metal (vitalium) plates and screws to stabilize maxillofacial fractures and then osteotomies (see Chapter 2). In current practice, the use of internal miniplate and microplate and screw (titanium) fixation of various sizes is the preferred form of rigid fixation when the stability and three-dimensional craniomaxillofacial reconstruction of multiple osteotomized bone segments and grafts are required. In infants and young children, resorbable materials are now generally used for the stabilization of non–load-bearing osteotomy segments (e.g., the cranial vault).[139,196,199,221] This avoids the issues or uncertainty regarding growth restriction and brain trauma from retained non-resorbable hardware.

Philosophy Regarding the Timing of Surgery

In considering both the timing and type of intervention, the experienced surgeon will take several biologic realities into account, including the natural course of the malformation (i.e., progressively worsening dysmorphology versus a nonprogressive craniofacial abnormality); the tendency for growth restriction in operated skeletally immature bones (i.e., akin to the maxillary hypoplasia that occurs following cleft palate repair); the relationship between the underlying developing viscera (i.e., brain) and the congenitally affected and/or surgically altered skeleton (i.e., brain compression if the cranial vault is not expanded); and the child's airway needs (i.e., midface deficiency resulting in OSA).

To limit impairment while simultaneously achieving long-term preferred facial aesthetics and head and neck

function, the surgeon must ask an essential question, "*during the course of craniofacial development, does the operated-on facial skeleton of a child with a craniosynostosis syndrome tend to grow abnormally, resulting in further distortions and dysmorphology, or are the initial positive skeletal changes achieved (at operation) maintained during ongoing growth?*" Unfortunately, the proposed theory that craniofacial procedures carried out in early infancy will "unlock growth" has not been documented through the scientific method.[194,200-206,218,219]

Final reconstruction of the upper midface deformities in those born with a craniosynostosis syndrome can be managed as early as 7 to 10 years of age. By this age, the cranial vault and orbits normally attain approximately 85% to 90% of their adult size. Whenever feasible, waiting until the maxillary first molars have erupted is also preferred. When the "upper midface" reconstruction is carried out after this age, the objective is to attain adult morphology in the cranio-orbito-zygomatic region with the expectation of a stable result (no longer influenced by growth) once healing has occurred. Psychosocial considerations also support the age 7 to 10 years time frame for the upper midface procedure. When a successful reconstruction is achieved at this age, the child may progress through school with an opportunity for a healthy body image and self-esteem.*

Management of the Upper Midface Deformity in Children and Young Adults

Incision Placement and Soft-Tissue Management

For the exposure of the craniofacial skeleton above the Le Fort I level, the approach used is the coronal skin incision. This allows for relatively camouflaged access to the anterior and posterior cranial vault, the orbits, the nasal dorsum, the zygomas, the upper maxilla, the pterygoid fossa, and the temporomandibular joints. For added cosmetic advantage, the placement of the coronal incision more posteriorly on the scalp and with postauricular rather than preauricular extensions is useful.[214] When the exposure of the maxilla at the Le Fort I level is required, a circumvestibular maxillary intraoral incision is used. Unless complications occur that warrant unusual exposure, no other incisions are required to manage any aspect of reconstruction in patients with a craniosynostosis syndrome. These incisions (i.e., coronal [scalp] and maxillary [circumvestibular]) may be reopened as needed to further complete the individual's staged reconstruction.

A layered closure of the coronal incision (i.e., the galea and the skin) optimizes healing and limits scar widening. Resuspension of the midface periosteum to the temporalis fascia may facilitate the redraping of the soft tissues. Each lateral canthus should be reattached in a superoposterior direction to the newly repositioned lateral orbital rim. In children, the use of chromic gut on the skin may obviate the need for postoperative suture or staple removal.

Management of the Ventricular System

In the patient with a craniosynostosis syndrome who is to undergo intracranial volume expansion with the concurrent management of hydrocephalus as part of the reconstruction, the potential for morbidity increases.[25,77,91,108,170,174] Complications may arise from either excessive cerebrospinal fluid drainage (overshunting) or inadequate shunting that leads to increased intracranial pressure. With overshunting there is decreased central nervous system mass to fill any surgically created retrofrontal dead space. Uncontrolled hydrocephalus may result in raised intracranial pressure, which leads to its own set of problems. In either situation, fronto–facial advancement via an intracranial approach to the midface or isolated cranial vault expansion procedures should be thoughtfully staged with ventriculoperitoneal shunt management. We believe that the presence or absence of a ventriculoperitoneal shunt is not in itself a major factor in the success of a fronto–facial advancement procedure. An important aspect is the satisfactory physiologic function of the ventricular system, with or without the placement of a shunt. Ultimately, the decision regarding the need for and sequencing of shunting is based on the patient's neurologic findings and the neurosurgeon's judgment. In a patient with a ventriculoperitoneal shunt in place before the surgery, experienced neurosurgical evaluation, including imaging studies of the ventricular system, is carried out to confirm satisfactory physiologic function.

Upper Midface Reconstruction Options

The osteotomy selected (i.e., extra cranial Le Fort III or intracranial, monobloc or facial bipartition) to manage the upper midface dysmorphology in the individual with a craniosynostosis syndrome should reflect the presenting skeletal deformities and provide a realistic opportunity for long term upper midface (naso-orbito-malar) region aesthetic enhancement.

A main objective of this phase of reconstruction is to "normalize" the orbits, zygomas, and cranial vault. Correction of the maxillo-mandibular deformity will require orthognathic surgery including at a minimum a separate Le Fort I osteotomy. The patient specific dysmorphology will depend on the original malformation, the previous procedures carried out, and the effects of ongoing growth.

When evaluating the upper and midface morphology in the mixed dentition child or young adult born with *Crouzon syndrome*, one should note: 1) if the supraorbital ridge is in good position when viewed from the sagittal plane (are the depth of the upper orbits adequate?); 2) if the midface and forehead have an acceptable arc of rotation in the transverse plane (is the midface arc concave?); and 3) if the root of the nose and orbits are of normal width (is there orbital hypertelorism?). If these structures are confirmed to be with acceptable morphology, then there is no need to further reconstruct the forehead and upper orbits. In those few,

*References 8, 10, 11, 23, 32, 36, 56, 57, 63, 69, 70-72, 89, 97, 109, 110, 113, 117, 120, 119, 136, 149-151, 155-158, 169, 171, 176, 180, 189, 192, 195, 197, 215, 235, 239, 241, 244, 277

craniosynostosis syndrome patients, in whom the residual deformity is only in the lower half of the orbits, the zygomatic buttress, and the maxilla an extracranial Le Fort III osteotomy is likely to be effective treatment.

If the supraorbital region, anterior cranial base, zygomas, root of the nose as well as the lower orbits, and maxilla all remain deficient in the sagittal plane (horizontal retrusion), then an MB is indicated. In these patients, the forehead is also generally flat and retruded and will require reshaping and advancement. If upper midface hypertelorism (increased transverse width) with midface flattening (horizontal retrusion) and a concave facial curvature (reverse facial arc) is also present, then the MB unit is split vertically in the midline (facial bipartition). A wedge of interorbital (nasal and ethmoidal) bone is removed, and the orbits and zygomas are repositioned medially while the maxillary arch is widened. A facial bipartition is rarely required in *Crouzon syndrome* but the MB is. When a monobloc or facial bipartition osteotomy is carried out as the "upper midface" procedure, additional segmentation of the upper and lateral orbits may also be required to normalize the morphology of the orbital aesthetic units.

For almost all *Apert syndrome* patients, facial bipartition osteotomies combined with further cranial vault reshaping permits a better correction of the dysmorphology than can be achieved through any other upper midface procedure (i.e., MB or Le Fort III). When using facial bipartition osteotomies, correction of the concave midface arc of rotation is also possible. This further reduces the stigmata of the Apert "flat, wide and retrusive" facial appearance. The facial bipartition allows the orbits and zygomas to shift to the midline (correction of hypertelorism) as units while the maxilla is simultaneously widened (i.e., relief of the V-shaped face). Horizontal advancement of the reassembled upper midface complex is then possible to improve orbital depth and zygomatic length. The forehead is generally flat, tall, and retruded, with a constricting band just above the supraorbital ridge. Reshaping of the anterior cranial vault is also simultaneously carried out. A Le Fort III osteotomy is virtually never adequate for an ideal correction of the residual upper midface anomalies documented in *Apert syndrome*.

The study by McCarthy confirms that the Le Fort III osteotomy is not effective as an aesthetic option to manage the upper midface deformity in the majority of craniosynostosis syndrome patients (see section later in this chapter concerning Le Fort III option).[276] By anatomic design, the Le Fort III prevents management of the whole orbital aesthetic unit during one operative setting. Therefore a major aesthetic shortcoming of the Le Fort III osteotomy, when its indications do not fit the presenting dysmorphology, is the creation of irregular step-offs in the lateral orbital rims. This will occur even when only a moderate Le Fort III advancement is carried out. These lateral orbital step-offs will be visible to the casual observer as unattractive at conversational distance and surgical attempts at modification performed later will be suboptimal. Another problem with the Le Fort III osteotomy is the difficulty in judging an ideal orbital depth. This frequently results in either residual proptosis or enophthalmos. Simultaneous correction of upper face (orbital) hypertelorism and the concave midface arc-of-rotation typical in Apert syndrome is also not possible with the Le Fort III procedure. Excessive lengthening of the nose, accompanied by flattening of the nasofrontal angle, will also occur if the Le Fort III osteotomy is selected when the skeletal dysmorphology favors an MB or facial bipartition. Unfortunately, it is not possible to later correct the elongated nose or the flattened naso-frontal angle. To avoid these short comings, it is not a matter of becoming more proficient at the Le Fort III osteotomy or simply "managing the overlying soft tissues" in a different way (i.e., canthopeties or midface lift). The Le Fort III osteotomy is not consistent with the presenting dysmorphology in most craniosynostosis syndrome patients and, therefore, will not provide the opportunity to achieve the desired aesthetic result. Nevertheless, the Le Fort III is often considererd the "go to" approach by surgeons as it is: 1) an extracranial procedure; 2) requires less surgeon skill/experience; 3) is less likely to result in significant blood loss; and 4) is less likely to result in perioperative complications (i.e., cranionasal fistula, intracranial abscess, bone resorption).

In most syndromal craniosynostosis patients, a suboptimal aesthetic result will occur if the surgeon attempts to simultaneously adjust the orbits and idealize the occlusion using the Le Fort III, MB or facial bipartition osteotomy without completing a separate Le Fort I osteotomy. The degree of horizontal deficiency observed at the orbits and at the maxillary dentition are rarely uniform. If a Le Fort I osteotomy to separate the "lower midface" from the "upper midface" complex is not carried out, excess advancement at the orbits with enophthalmos is likely to occur as the surgeon attempts to achieve a positive overjet at the incisors. The Le Fort I osteotomy is generally *not* performed at the time of the "upper midface" procedure. This will await skeletal maturity and is combined with orthodontic treatment. Until then, a degree of an Angle class III anterior open bite negative overjet malocclusion will remain. When the mature teenage or adult patient presents for surgical correction and requires both "upper midface" (i.e., naso-orbito-zygomatic) and "lower midface" (i.e., maxilla) management the procedures may be carried out simultaneously.

Monobloc and Facial Bipartition Osteotomies (Intracranial Approach to the Orbits and the Midface)

Cranial vault reconstruction in the child with a craniosynostosis syndrome should provide space for the compressed brain to expand. Immediately after completing cranial vault and monobloc (MB) or facial bipartition (FB) osteotomies with advancement, extradural (retrofrontal) dead space remains in the anterior cranial fossa above the skull base gap created by the osteotomy.[208] The skull base gap is in direct communication with the nasal cavity. Therefore, the postoperative recovery may be complicated by cerebrospinal fluid leakage across the skull base gap followed by infection,

fistula formation, and subsequent bone resorption in the glabella region. After fronto–facial advancement by either MB or FB, the communication between the nasal cavity and the cranial fossa must be managed to limit these potential complications. The most effective method to do so remains unclear, but all agree that it is a critical aspect for successful reconstruction. Technical aspects of management include the following: 1) gentle tissue handling; 2) achieving good hemostasis; 3) effective repair of dural tears; 4) avoidance of over expansion of the cranial vault; 5) maximum separation of dura and nasal mucosal tissue planes (i.e., interposing tissue such as bone grafts, tissue sealants, and flaps); 6) rigid plate and screw fixation of the osteotomies and bone segments; 7) avoidance of pressure gradients across the opening to facilitate nasal mucosa healing; and 8) prevention of too much or too little ventriculoperitoneal shunting.

After cranio–orbital reconstruction in the infant with a craniosynostosis syndrome, rapid filling of the expanded intracranial volume by the previously compressed frontal lobes of the brain has been documented.[208] This has also been shown to occur after MB and FB advancement in children and young adults.[249] More gradual filling of the space is thought to occur in older adults. At the time of the MB or FB osteotomies, the nasal cavity can be sealed from the cranial fossa by doing the following: 1) inserting pericranial tissue; 2) placing bone grafts to bridge the osteotomy gaps; and 3) using tissue sealants. This provides time for the re-epithelialization (i.e., healing) of the nasal mucosa. Until the torn nasal mucosa heals, communication between the nasal cavity and the anterior cranial fossa may result in the transfer of air, fluid, and bacteria followed by infection and naso–cranial fistula formation. The postoperative continuation of nasotracheal intubation for several days and nasopharyngeal tube placement after extubation have also proven useful to limit pressure gradients across the communication. The avoidance of positive pressure ventilation, the enforcement of sinus precautions, and the restriction of nose blowing further limits the reflux of air, fluid, and bacteria early after surgery.

Four clinical studies clarify issues of morbidity related to MB or FB osteotomies for the reconstruction of an individual with a craniosynostosis syndrome.

Posnick and colleagues studied the issues of retrofrontal dead space; communication across the skull base osteotomy gap; and associated morbidity in a consecutive series of children in the mixed dentition and young adults in the permanent dentition (n = 23) who were undergoing either MB or FB osteotomies in combination with cranial vault expansion.[208] The procedures were carried out by a single craniofacial surgeon (Posnick) and one of three neurosurgeons at a single tertiary care hospital from 1987 to 1991. The extradural (retrofrontal) dead space was measured from consistent CT scan images at specific postoperative intervals (immediate, 6 to 8 weeks, and 1 year). The study confirmed the presence of an immediate retrofrontal dead space that generally filled in with the expanding brain and dura by 6 to 8 weeks after surgery. Specific intraoperative maneuvers were undertaken by the surgeons to close the nasofrontal communication, including flaps, fibrin glue, bone grafts, and Gelfoam. After surgery, care was taken to limit a pressure gradient across the communication (via the repair of dural tears, sinus precautions, and nasal stenting), with the objective of providing time for nasal mucosa healing. The infection rate in this study group was limited to 2 of 23 patients (9%). In both patients who developed an infection, a retrofrontal (extradural) fluid collection was noted, with drainage across the residual nasofrontal communication into the nose. Both patients healed without significant comorbidity (i.e., brain or eye injury) but did require further reconstruction of the resorbed portions of the anterior cranial vault and the supraorbital ridges (see Fig. 30-15).

Wolfe completed a critical analysis of 81 MB advancements carried out over a 27-year period.[281] This was a retrospective chart analysis of a series of patients who underwent either MB (frontofacial) advancement (MFFA) or FB (MFFA plus FB). The procedures were carried out at seven different craniofacial centers and included 49 MFFA and 32 MFFA plus FB. The MFFA and MFFA plus FB osteotomies were either placed in their preferred location in the operating room (standard approach) or gradually distracted (DO technique) with internal or external devices. Complications included two deaths (cardiac arrest in one patient and complications arising from hypovolemia in the other). One case was aborted as a result of a large-volume blood loss; there were three infections or sequestrations and one persistent cerebrospinal fluid leak (no meningitis). There were significant complications documented in the DO group and fewer in the non-DO group. Blood loss and operative time were equivalent for both standard and DO techniques. Interestingly, the incidence of infection and cerebrospinal fluid leaks was not diminished with the use of the alternative distraction DO approach. The author also concluded that, for the majority of patients, the standard approach offered improved morphologic results. The author then compared the morphologic results of the MMFA and MMFA plus FB with those of the extracranial Le Fort III option and concluded that the Le Fort III was less favorable. Regardless of the technique used, all patients required orthognathic surgery (at the Le Fort I level) to complete the reconstruction.

Bradley and colleagues completed a single-center retrospective study that compared differences in morbidity in a series of patients who were born with a craniosynostosis syndrome and who then underwent MB osteotomy for the correction of upper and midface anomalies or hypoplasia.[19] The authors describe three different sequential treatment groups that were followed during a period of 23 years. Group I patients (1979 to 1989; n = 12) underwent MB osteotomies without any special attention paid to the retrofrontal dead space or the communication through the skull base between the anterior cranial fossa and the nasal cavity. Group II patients (1989 to 1995; n = 11) underwent MB

osteotomies with various attempts at closure of the skull base gap with pericranial flaps and fibrin glue. Group III (1995 to 2002; n = 24) patients underwent MB osteotomies without immediate advancement. An internal distraction device was placed across the osteotomized zygomatic arch on each side. After a 7-day latency period, the advancement of the MB and forehead unit was initiated at 1 mm per day for approximately 2 to 4 weeks. The infection rate for Group III patients was significantly lower (2 of 24 [8%]) than it was for those in Groups I and II. Neither of the two infections in Group III resulted in bone loss. Group I patients had the greatest morbidity likely as a result of the limited fixation performed during the 1970s and 1980s.

As described by Bradley and colleagues, the DO technique does allow more time for the brain to expand into the retrofrontal dead space after the completion of an MB osteotomy (i.e. delayed for 7 days) before advancing the upper midface.[19] In theory, this should facilitate early nasal mucosa healing and thereby limit the communication of fluid, air, and bacteria across the surgically created skull base gap. This likely explains the drop in infection rate in the Group III patients as compared with the Group I and II patients. The rate of infection in Bradley's Group III patients, who were treated with a DO approach, essentially matches that of the patients described by Posnick and colleagues who were treated with a standard approach (8% and 9%, respectively).

Bradley and colleagues also described greater advancement in Group III (DO approach) patients as compared with Group I and II patients.[19] Confounding variables may explain these differences, including increased surgical experience during the later years of the study (i.e., group III patients) and the impact of complications during the earlier years of the study (i.e., extremely high rates of infection in Groups I and II), which likely resulted in relapse and limited the long-term midface advancement. More importantly, there was no correlation between the number of millimeters of MB advancement and either greater functional gains or enhanced facial aesthetics.

COMMENT: With an MB osteotomy, as much aesthetic damage is done by overadvancement (enophthalmos) as by underadvancement (residual eye proptosis). In addition, the achievement of a "normal" occlusion is not usually a treatment objective at the time of MB advancement. Accomplishing an ideal occlusion without creating enophthalmos generally requires a separate Le Fort I osteotomy to differentially reposition the maxilla.

A fourth clinical study sheds further light on this subject. **Ahmad and colleagues** described a series of 12 children who were born with a craniosynostosis syndrome who also had multiple functional problems, including the following: 1) raised intracranial pressure as a result of a diminished cranial vault volume; 2) exposure of the eyes (i.e., corneal irritation) as a result of shallow orbits; 3) airway obstruction from a reduced upper airway space (i.e. OSA); and 4) feeding difficulties.[3] Each of the study patients underwent fronto–facial (MB) advancement with the use of DO techniques. The mean age at operation was 18 months (range, 4 to 30 months). The mean advancement was 16.6 mm at the forehead level and 17 mm at the midface level. Ocular protection and the reduction of intracranial pressure (when raised) was achieved in all children. At least a degree of airway improvement was achieved in all but one child. The authors subjectively state that there was marked improvement in every patient's appearance. Complications included cerebrospinal fluid leaks (2 of 12 patients [16.6%]); pin-site infections (3 of 12 patients [25%]); external DO device frame slippage that required replacement (2 of 12 patients [16.6%]); and overadvancement with resulting enophthalmos (1 of 12 patients [8.4%]). In addition, one patient died 9 months after the procedure in conjunction with tracheal reconstruction. The published article was discussed by **Hopper**,[111] who recommends that clinicians consider a less invasive approach and accept limited aesthetics objectives in an attempt to lower perioperative morbidity. The approach that he suggests includes the following: 1) either anterior cranial or posterior cranial vault expansion without simultaneous midface advancement to relieve intracranial pressure; 2) extracranial Le Fort III advancement without simultaneous cranial vault expansion to open the upper airway; 3) tarsorrhaphies to protect the eyes from exposure rather than orbital expansion; and 4) continued tracheostomy for airway management rather than midface advancement.

COMMENT: The four studies reviewed and the discussion by Hopper that followed demonstrate the wide variation of opinions on how best to reconstruct individuals with a craniosynostosis syndrome. In this author's opinion, for the majority of these children, only the MB or facial bipartition osteotomies offer a realistic opportunity to achieve close to normal morphology and the opportunity to develop healthy self-esteem. Favorable craniofacial function is expected to follow the enhanced facial form. This includes cranial vault expansion (i.e., relief of ICP); orbital expansion (i.e., relieve of proptosis and eye protection); and upper airway expansion (i.e., relief of OSA and improved breathing during the day). Gains in mastication, swallow mechanism, speech articulation, breathing, vision, and cognitive function are also expected.

For the individual with a craniosynostosis syndrome, to achieve the most favorable facial harmony and head and neck function, the surgeon needs both an aesthetic sense and the technical expertise to execute effective upper/midface surgical procedures. Several key aspects include:

1. The ability to remove, segment, reshape and then stabilize the cranial vault
2. The ability to separate the orbits and midface as a unit MB from the skull base
3. The ability to segment and reshape the upper orbits of the MB including interposing bone grafts as needed to reconstruct each orbital aesthetic unit during a single operative setting.
4. The ability to separate the MB into halves (facial bipartition) and then three-dimensionally reposition and stabilize (plate and screw fixation) the two facial halves to achieve the most favorable morphology in all three planes (i.e., pitch, role, yaw orientation). This often requires increasing the maxillary width (i.e., arch expansion) and decreasing the upper face width (i.e., correction of hypertelorism of the orbits, zygomas, and bitemporal regions). Facial bipartition also provides the ability to correct the transverse facial arc of rotation. An example is changing the concave facial arc of rotation (i.e., yaw orientation) in Apert syndrome.

In current clinical practice when either a monobloc or facial bipartition osteotomy is selected, the technique of "distraction" to gradually advance the upper/midface, after closure of the wounds is frequently used. Any potential for reduced infection across the skull base using the DO technique should be considered in light of limitations to achieving the aforementioned aesthetic objectives (points 1 to 4 mentioned previously). At the same time, clinicians must take into account the procedure specific morbidity associated with the DO technique including: 1) pin tract infection; 2) soft tissue scarring; 3) hardware loosening requiring re-application; 4) brain trauma from the screw penetration; and 5) the need for device removal. 6) In addition, effective DO technique depends on the patient's, the patient's family, and the clinician's continued commitment to staying the course after surgery (approximately 4 months). *It is hoped that in the future, clinicians will both master the known surgical techniques and also find novel solutions to manage the potential for morbidity associated with MB and facial bipartition osteotomies (i.e., CSF leak, intracranial abscess, and bone resorption) while focusing to the aesthetic details so that individuals with a craniosynostosis syndrome can more commonly benefit from the procedures inherent advantages.*

Le Fort III Osteotomy (Extracranial Approach to the Midface)

Shetye and colleagues set out to examine long-term (10-year) midface skeletal stability and the potential for maxillary growth after extracranial Le Fort III advancements in children with craniosynostosis syndromes.[242] Their study group included 25 children (10 had Crouzon syndrome, 9 had Apert syndrome, and 6 had Pfeiffer syndrome) who underwent extracranial Le Fort III advancement before they were 11 years old (mean, 5.8 years; range, 3.8 to 10.9 years). Two different forms of fixation were used: Group 1's treatment included intraosseous wires, bone grafts, suspension wires, dental splint, and 8 weeks of intermaxillary fixation; Group 2's treatment involved titanium plates and screws, bone grafts, dental splints, and elastics. Good stability of the midface was documented in both groups at 1 year, 5 years, and 10 years. As has been found with previously published studies, no significant horizontal growth occurred in the midface after surgery. The form of fixation used was not a factor (i.e., plate and screw fixation did not increase incidence of growth restriction). The mandible was measured at the pogonion and found to advance 5.72 mm and 7.32 mm at the 5- and 10-year time points, respectively. This study confirms that, when the Le Fort III advancement is carried out at a young age, the occurrence of jaw disharmony with ongoing growth is inevitable and will require further staged reconstruction.

In a second publication, Shetye and colleagues evaluated a larger group of syndromal craniosynostosis patients who also underwent extracranial Le Fort III advancement between the years of 1973 and 2006.[238] Three different stabilization techniques were sequentially used over the study period: Group 1 (n = 20) was treated with intraosseous wires, suspension wires, bone grafts, dental splinting, and 8 weeks of intermaxillary fixation; Group 2 (n = 20) was treated with titanium plates and screws, bone grafts, dental splinting, and elastics; and Group 3 (n = 20) was treated with the extended use of an external DO device during 3 months of healing. The stability of midface advancement was assessed with the use of measurements from 1- and 5-year postoperative lateral cephalometric radiographs. Patients in Groups 1 and 3 showed equal levels of skeletal stability, whereas Group 2 showed slightly less stability at the A point. At the orbitale, all three groups showed similar levels of stability. Interestingly, all three methods of fixation for the extracranial Le Fort III advancements showed good levels of stability. The study also confirmed that all study subjects who were in the mixed dentition demonstrated progressive facial disharmony as a result of ongoing mandibular growth, thereby necessitating further skeletal reconstruction by the late teenage years (i.e., different forms of fixation had no bearing on the incidence of growth restriction).

In the discussion, McCarthy, who was the senior investigator, stated his personal preference for the management of the midface deformity in syndromal craniosynostosis with an extracranial Le Fort III advancement followed by orthognathic surgery. When early Le Fort III advancement is indicated in a growing child, he prefers distraction to standard techniques. According to McCarthy, it is easier to overcorrect the advancement with the use of DO, which is one of his stated objectives for young children (i.e., those who are <11 years old). Standard extracranial Le Fort III advancements with plate and screw fixation and bone grafts are preferred by McCarthy for older children and adults (i.e., those who are >11 years old), because this allows for

reduced treatment time and less patient psychosocial issues (i.e., there is no need to wear an external DO device for 3 months after surgery).

McCarthy and colleagues recently reviewed the long-term midface growth and periorbital/malar (upper midface) aesthetic results in individuals who were born with a craniosynostosis syndrome and who had undergone Le Fort III advancement.[276] The study represented a follow-up period of more than 20 years for four identified patients (one with Apert syndrome and three with Crouzon syndrome) who were 4, 6, 14, and 20 years old at the time of the Le Fort III advancement. Thus, this study represented two young children and two skeletally mature individuals who underwent Le Fort III midface advancement. The authors' review confirmed several points: 1) a Le Fort III advancement carried out during early childhood results in no further horizontal growth of the midface; 2) a Le Fort III advancement carried out during early childhood results in further vertical growth of the midface with clockwise rotation of the mandible; and 3) a Le Fort III advancement carried out in individuals with either Crouzon or Apert syndrome—whether during childhood or adulthood—does not effectively correct the presenting zygomatic, orbital, and nasal malformations. The upper midface aesthetic results as viewed through the overlying soft-tissue envelope (i.e., the eyelid–cheek–malar region) were suboptimal after Le Fort III advancement. Furthermore, with age, the upper midface aesthetic results became even more unfavorable.

McCarthy's findings of continued vertical growth of the midface after Le Fort III osteotomy carried out during early childhood is consistent with the inability to achieve adequate nasal airflow in the operated patient. In addition, even if adequate nasal airflow is accomplished, at least some individuals will have limited masticatory muscle tone. In either case, when a child continues with an open-mouth protrusive tongue posture, hypereruption (i.e., vertical growth) of the maxillary dentition through the alveolar bone with clockwise rotation of the mandible is expected (see Chapter 4). A lack of horizontal growth of the maxilla after either a Le Fort I, Le Fort III, or MB osteotomy is carried out during childhood has been documented by a number of independent investigators.[31,50,75,76,102,103,112,192] This results from both intrinsic growth limitations of the malformation and injury to the sutural growth centers from the osteotomies that are carried out.

Monobloc and Facial Bipartition Osteotomies: Step-by-Step Description of the Surgical Technique

The lack of consensus about the ideal timing and techniques for the management of complex upper midface malformations and deformities in patients with craniosynostosis syndromes reflects not only uncertainty about the potential results with any one approach to treatment but also confusion about how to perform these technically sensitive procedures.

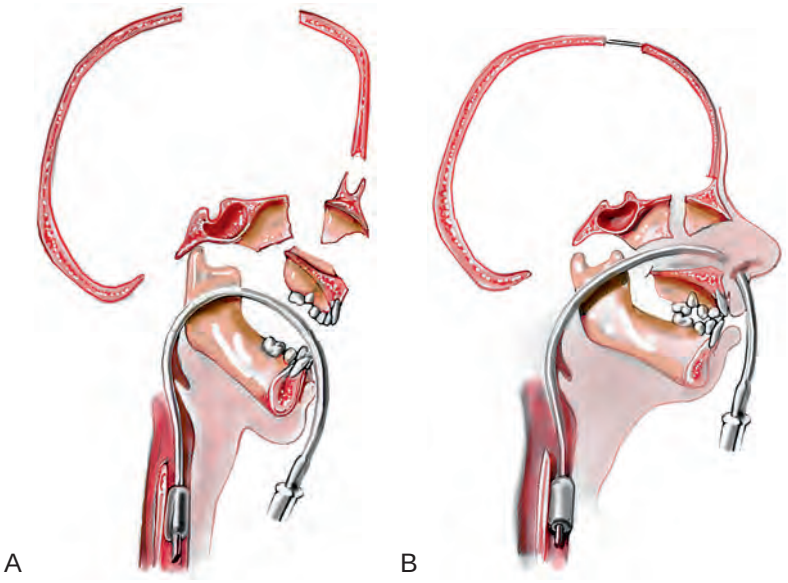
This section provides the surgeon with a step-by-step technical description for the MB and FB osteotomies as this author has routinely performed them. The detailed operative technique is illustrated through the case of a 12-year-old Persian girl who was born with Pfeiffer syndrome and who had never undergone surgery. The patient then underwent FB osteotomies in combination with anterior cranial vault reshaping (see Fig. 30-14). A review of the October 1992 Plastic Surgery Education Foundation Teleplast Conference: Monobloc and Facial Bipartition for Reconstruction of Craniofacial Malformations may serve as an additional educational tool to complete the surgeon's understanding of the technical aspects of the procedures that will be described (Video 15).

Monobloc and Facial Bipartition Osteotomies: Step-by-Step Approach

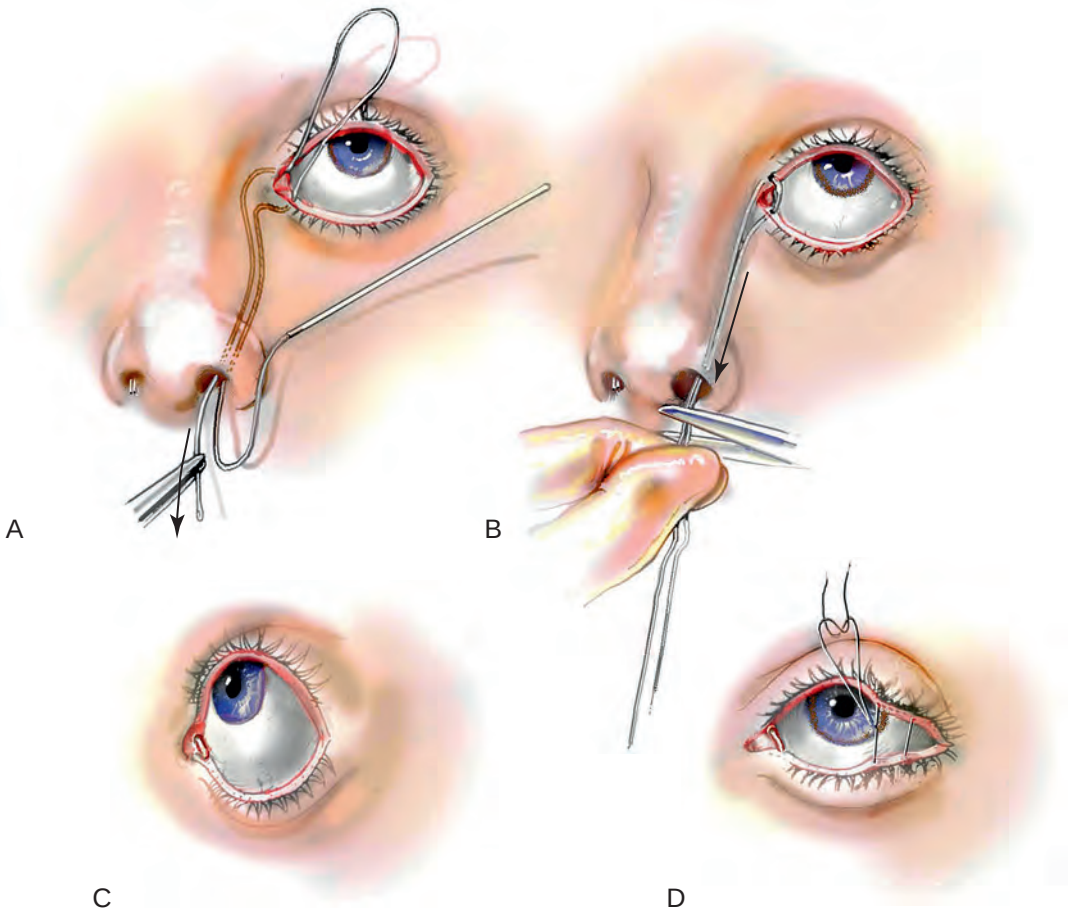
Step 1. Satisfactory airway management in a patient who is undergoing an MB/FB osteotomy is essential. A method that I have used effectively involves an orotracheal tube that is secured adjacent to the cusp edge of the mandibular incisors with a circummandibular wire (Fig. 30-16, *A*). After the MB/FB osteotomies and disimpaction are completed, a nasotracheal tube is placed, and the orotracheal tube is removed (Fig. 30-16, *B*). Through this controlled approach, endotracheal tube injury at the completion of the osteotomies and dislodgement during disimpaction are prevented. In addition, direct contact between the maxillary and mandibular teeth can be achieved for the improved control of the occlusion. The nasotracheal tube remains in place at the end of the procedure to allow for nasal mucosal stenting during the initial postoperative phase. Other approaches to managing the airway have been described and used effectively (e.g., tracheostomy, submental intubation, orotracheal intubation without exchange).

Step 2. When feasible, I recommend the placement of Crawford tubes to protect the nasolacrimal apparatus during surgery (Fig. 30-17). The puncta are dilated, and a probe is inserted through each punctum to confirm entrance into the nose. The Crawford tube is inserted through each punctum and pulled through the nose. Within the nose, the Silastic sheeting is stripped; the tubing is tied in the nose, and the excess is cut in the nose with scissors.

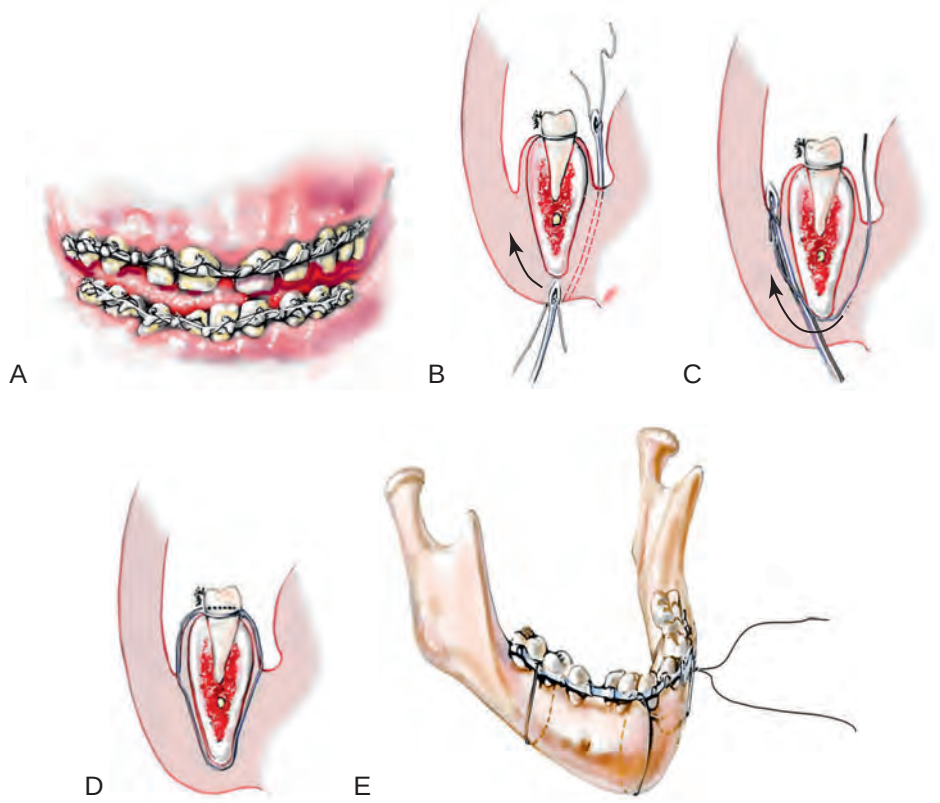
Temporary tarsorrhaphies are placed with 6-0 nylon suture from gray line to gray line just lateral to the pupil of each eye. As an alternative, corneal shields may be used to protect the cornea, but this



• **Figure 30-16 A** and **B,** Step 1. Illustrations of a 12-year-old girl with Pfeiffer syndrome (see Fig. 30-14) before and after facial bipartition. The step-by-step approach for the surgical technique that was carried out is described in the text. *From Posnick JC: Craniofacial dysostosis syndromes: a staged reconstructive approach. In Turvery JA, Vig TWL, Fonseca RJ, eds:* Facial clefts and craniosynostosis: principles and management, *vol 26, Philadelphia, 1995, W.B. Saunders, p 645.*



• **Figure 30-17 A** through **D,** Step 2.



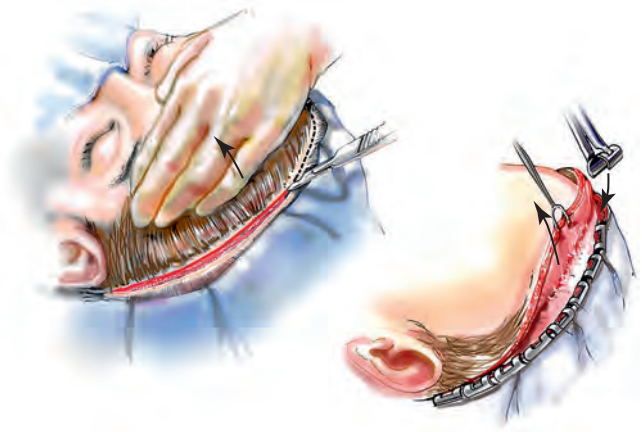
• **Figure 30-18** **A** through **E,** Step 3.

prevents the direct examination of the pupils during surgery.

Step 3. Surgical arch wires are generally applied (Fig. 30-18). A throat pack is placed, and the mouth is cleansed. Erich arch bars are applied to the maxillary and mandibular teeth. Circum-mandibular wires are placed to further stabilize the mandibular arch bar, and the orotracheal tube is secured with a wire to the symphyseal region of the surgical arch bar.

Step 4. The patient is prepared and draped. The patient's head is placed in a Mayfield (horseshoe-shaped) head holder in the neutral neck position. Cleansing of the entire scalp is performed with povidone–iodine (Betadine) soap; the soap is then washed out with sterile water. Next, the application of povidone–iodine solution to the scalp, face, and neck is carried out. The surgical field is draped to expose the neck down to the clavicles; the full face, including the external ears; and the anterior scalp back to the planned incision. The separation of the mouth/nose from the eyes/forehead is achieved with an additional sterile towel drape; this is done to limit the contamination of the intracranial cavity with oral or nasal flora.

Step 5. A coronal (scalp) skin incision is completed (Fig. 30-19). The incision site is postauricular and posterior in the scalp. Other incision modifications



• **Figure 30-19** Step 5.

have been described (e.g., Z-plasty in the temporal regions). Lidocaine with epinephrine is injected to facilitate hemostasis.

Step 6. The anterior scalp flap is then elevated by remaining deep to the superficial layer of the deep temporal fascia over the temporalis muscles; subperiosteal over the mid-forehead region; subperiosteal down the lateral orbital rims; subperiosteal with exposure of the anterior maxilla; subperiosteal for exposure of the zygomatic aches;

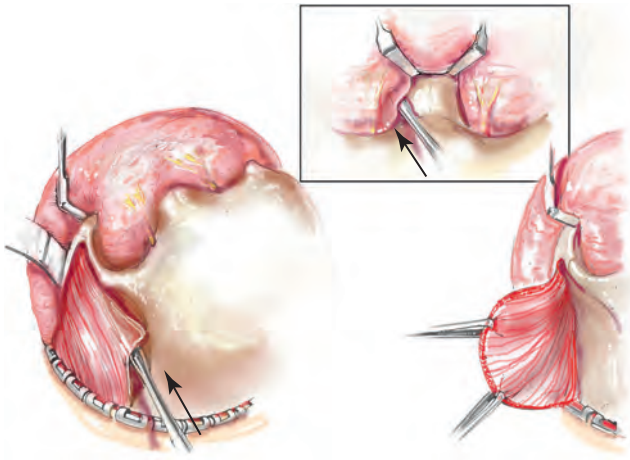
and subperiosteal over the dorsum of the nose. Elevation of the temporalis muscles off of the squamous temporal bones is then carried out (Fig. 30-20).

Step 7. Bifrontal craniotomy is completed (Fig. 30-21, *A*). The craniotomy lines are drawn with a sterile pencil, and bur holes are completed with a perforator as needed. The craniotomies are completed with a craniotome. The frontal bones are separated from the underlying dura and then removed. The frontal and temporal lobes of the brain are adequately retracted to safely accomplish skull base osteotomies. The brain is protected with cottonoid pledgets (Fig. 30-21, *B*).

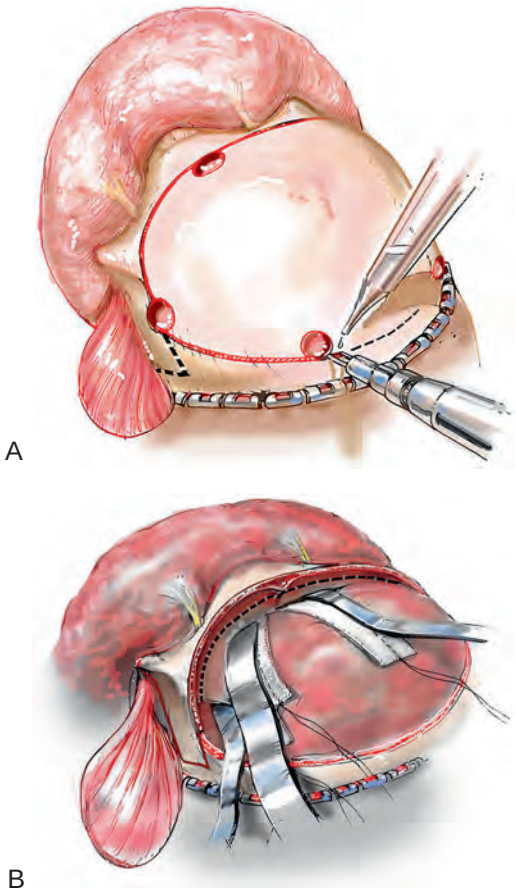
Step 8. Zygomatic arch osteotomies are performed (Fig. 30-22). Retractors are placed, and an osteotomy is completed through the midzygomatic arch on each side with a sagittal (reciprocating) saw.

Step 9. With the use of the sagittal saw, the lateral orbital wall osteotomy is initiated into the inferior orbital fissure. The osteotomy is extended superiorly through the lateral orbital wall (Fig. 30-23).

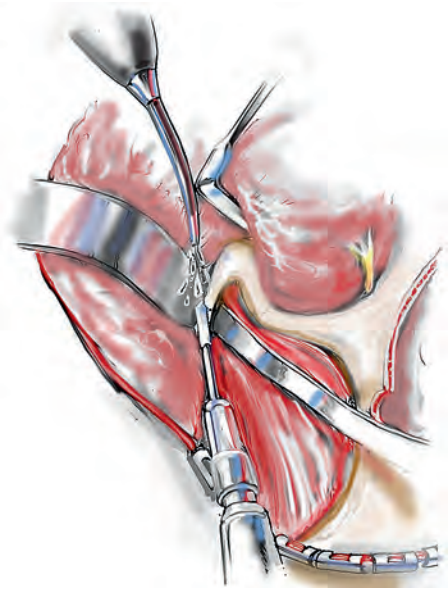
Step 10. With the continued use of the sagittal saw, the lateral tenon extension of the osteotomy through



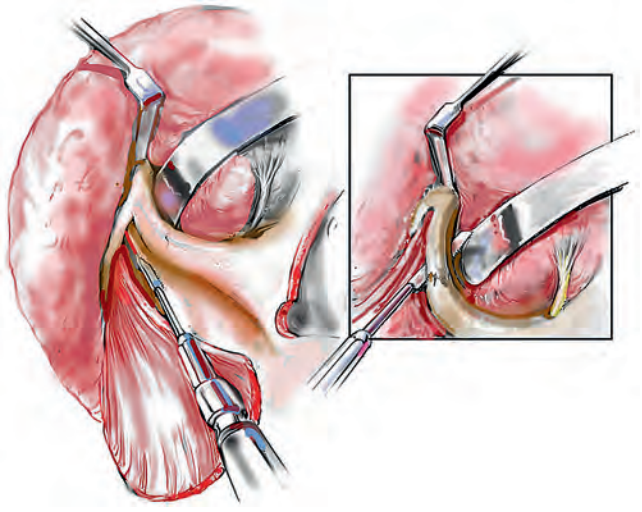
• **Figure 30-20** Step 6.



• **Figure 30-21** **A** and **B,** Step 7, *A* and *B*.



• **Figure 30-22** Step 8.



• **Figure 30-23** Step 9.

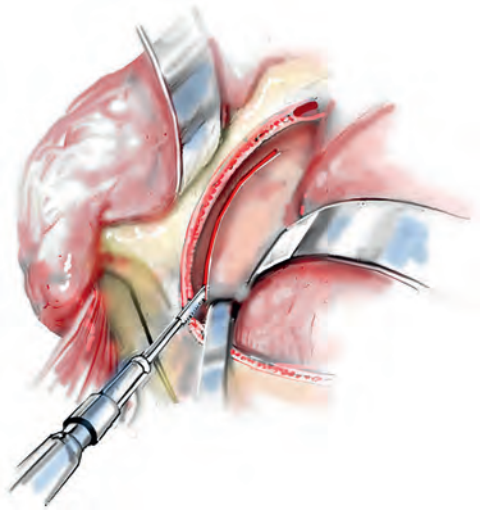
the squamous temporal bone and skull base is carried out (Fig. 30-24).

Step 11. The orbital roof osteotomy is completed with the sagittal saw through the anterior skull base (Fig. 30-25).

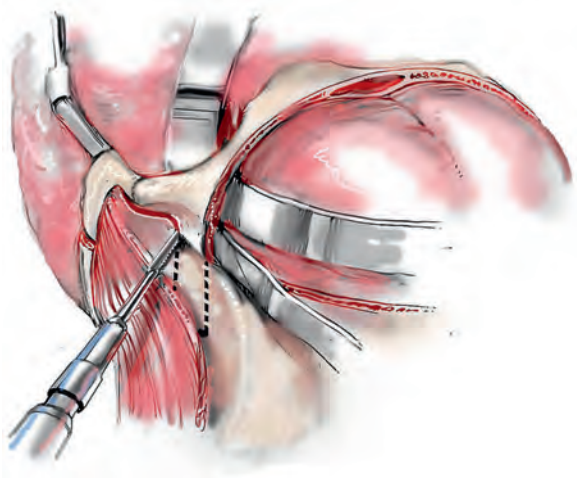
Step 12. The orbital roof osteotomy continues laterally through the sphenoid wing. This osteotomy will join up with the previous tenon extension on each side (Fig. 30-26).

Step 13. A thin chisel placed through the anterior skull base is used to confirm the completion of the sphenoid wing osteotomy and continuity with the tenon extension (Fig. 30-27).

Step 14. With a thin chisel and working through the skull base, the medial orbital wall osteotomy is completed posterior to the medial canthus and nasolacrimal apparatus and inferiorly into the inferior orbital fissure (Fig. 30-28).

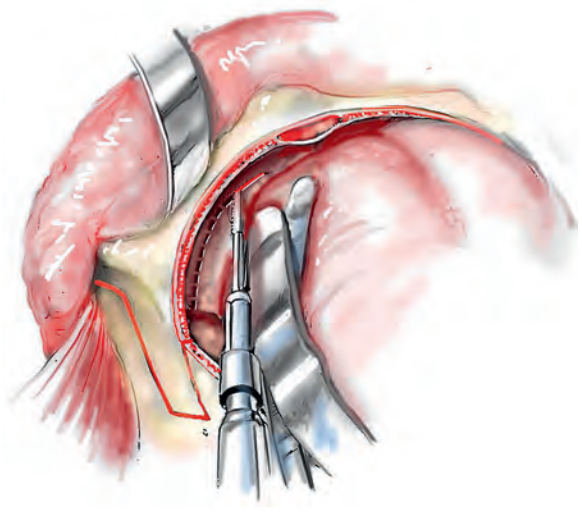


• **Figure 30-26** Step 12.

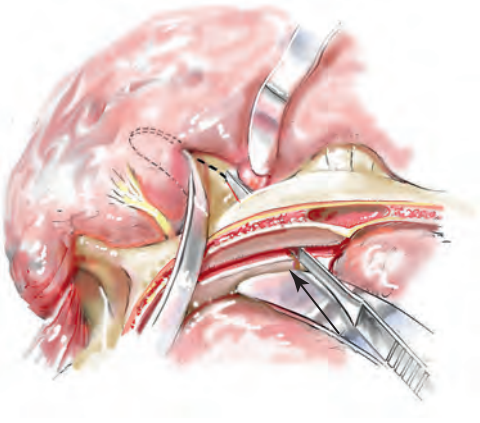


• **Figure 30-24** Step 10.

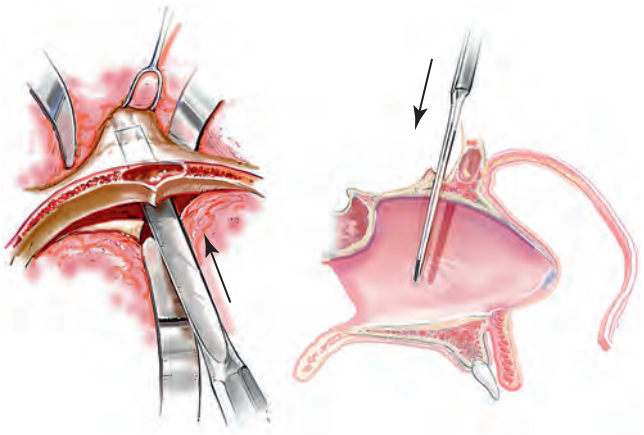
• **Figure 30-27** Step 13.



• **Figure 30-25** Step 11.



• **Figure 30-28** Step 14.



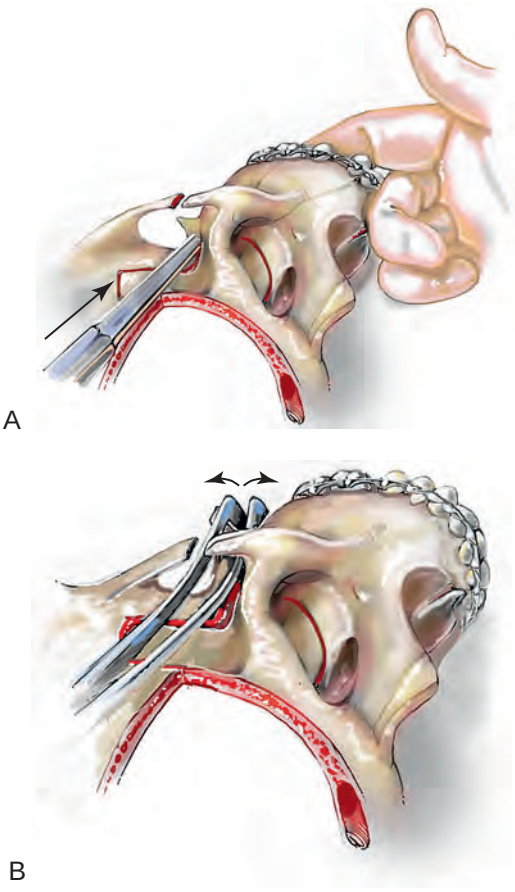
• **Figure 30-29** Step 15.

Step 15. The anterior aspect of the nasal septum is separated from the cranial base. A straight 15-mm wide chisel is placed through the cranial base just anterior to the crista galli and used to complete this osteotomy and to further separate the midface from the base of the skull (Fig. 30-29).

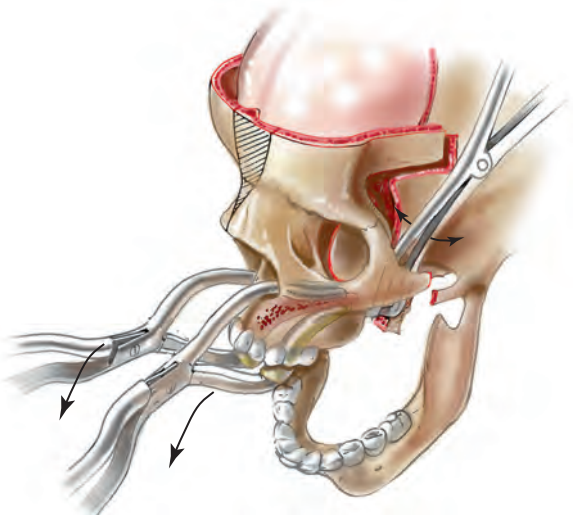
Step 16. The separation of the pterygomaxillary sutures is completed. A long 10-mm wide chisel is placed through the coronal incision and the infratemporal fossa to the pterygomaxillary suture. One double-gloved hand is placed in the patient's mouth, and the other hand is used to place the chisel through the coronal incision and into the infratemporal fossa (Fig. 30-30, *A*). A mallet is then used to separate the pterygomaxillary suture with the chisel. The success of the separation is confirmed with the pterygomaxillary spreader forceps (Fig. 30-30, *B*).

Step 17. The midface (MB) is disimpacted with the use of two nasomaxillary forceps that are placed in the nose and the mouth. Pterygomaxillary spreader forceps are simultaneously placed through the coronal incision on each side. The midface is then disimpacted and stretched forward to confirm adequate advancement at the occlusal level (Fig. 30-31). Next, the endotracheal airway exchange is completed (see Fig. 30-16, *B*). Additional sterile drapes are placed over the scalp and the face and neck regions, and the throat pack is removed. The surgeon places the nasotracheal tube through the nose and into the oropharynx. The anesthesiologist then removes the orotracheal tube and completes the insertion of the nasotracheal tube through the larynx using the direct or GlideScope laryngoscopic technique.

Step 18. For FB, a midnasal osteotomy (i.e., an ostectomy) is completed. Working through the coronal incision, the surgeon marks out the proposed midnasal osteotomy with calipers and a pencil and then completes it with a sagittal saw. When the



• **Figure 30-30** **A** and **B,** Step 16.

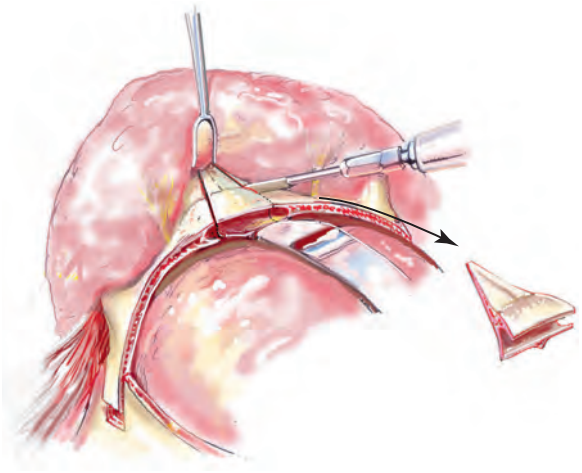


• **Figure 30-31** Step 17.

ostectomy is completed, the removal of portions of the underlying cartilaginous nasal septum is also accomplished (Fig. 30-32).

Step 19. For FB, separate sterile oral instruments are used to split the maxilla into two segments. An intraoral maxillary vestibular incision is made, and this

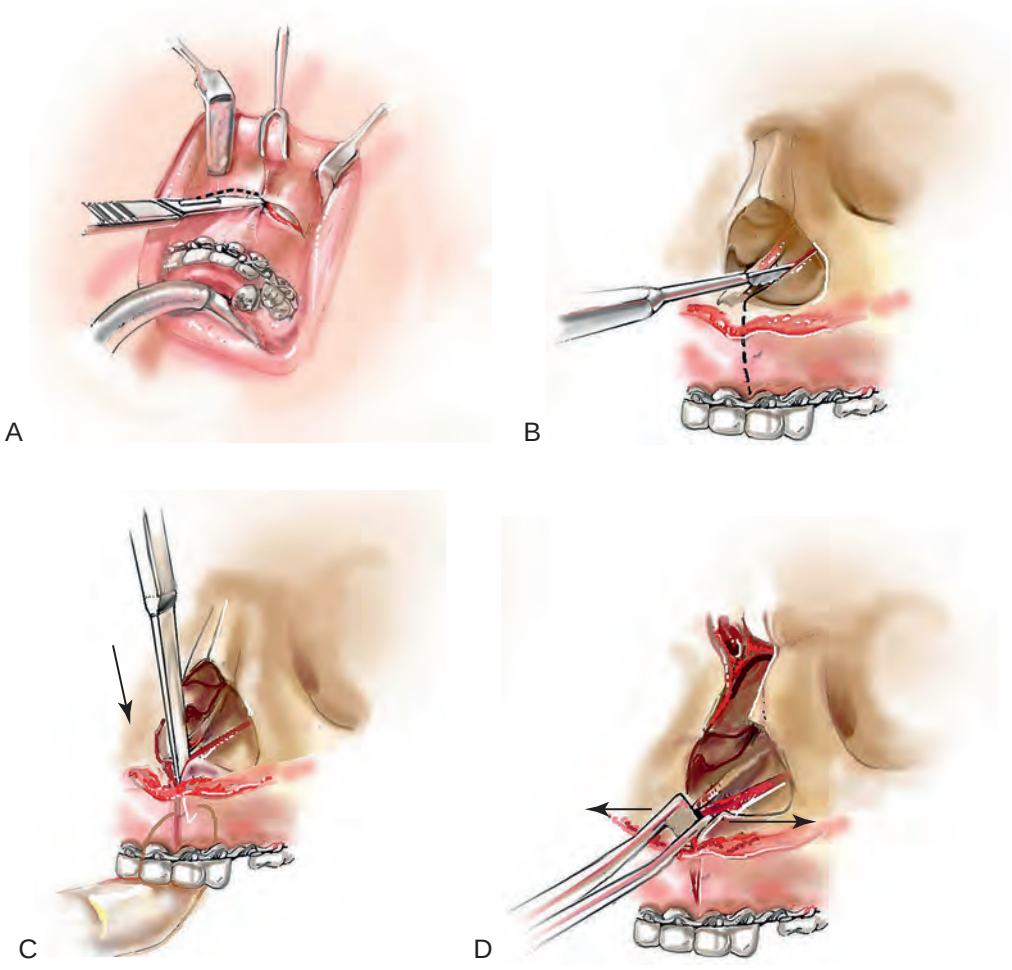
is followed by the subperiosteal exposure of the anterior maxilla, the anterior nasal spine, and the nasal floor (Fig. 30-33, *A*). With a sagittal saw, a midline osteotomy is completed between the central incisors and then parasagittally down the hard palate; the palatal mucosa is left undisturbed (Fig. 30-33, *B*). The segmental separation is completed with a thin 5-mm wide chisel placed between the central incisors (Fig. 30-33, *C*). The Erich arch bar is also cut between the incisors. Further separation of the posterior maxilla is completed with a spreader forceps as needed (Fig. 30-33, *D*). The oral wound is closed, the intraoral instruments are discarded, and fresh gloves are put on the hands.



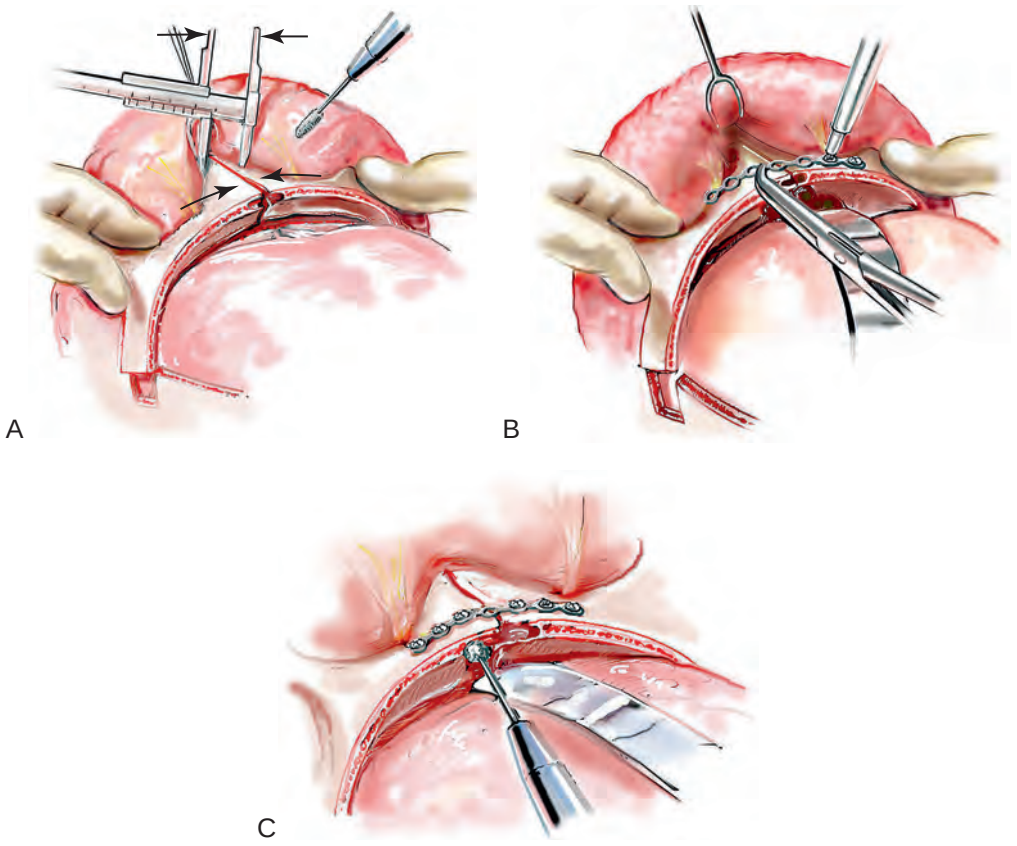
• **Figure 30-32** Step 18.

Step 20. For FB, stabilization of the upper orbits and the nasal bones is completed next (Fig. 30-34, *A, and B*). Repositioning of the facial halves medially with correction of the midface arc of rotation is completed; this will require refinement with a rotary drill (Fig. 30-34, *A*). The upper orbits and the nasal bones are fixed with a titanium plate and screws (Fig. 30-34, *B*). A rotary drill is also used to cranialize the frontal sinus, if needed (Fig. 30-34, *C*).

Step 21. Midface advancement at the level of the maxillary dentition is accomplished (Fig. 30-35). Working through the coronal scalp incision, the primary



• **Figure 30-33 A** through **D,** Step 19.



• **Figure 30-34 A, B,** and **C,** Step 20.

surgeon advances the midface. With the use of separate sterile oral instruments, the assistant to the surgeon wires the jaws together into the planned occlusion.

NOTE: The patient is rarely placed in an ideal occlusion. The mid face advancement at the occlusal level is of secondary importance. The advancement should be based on the most favorable depth of the orbits and position of the zygomas.

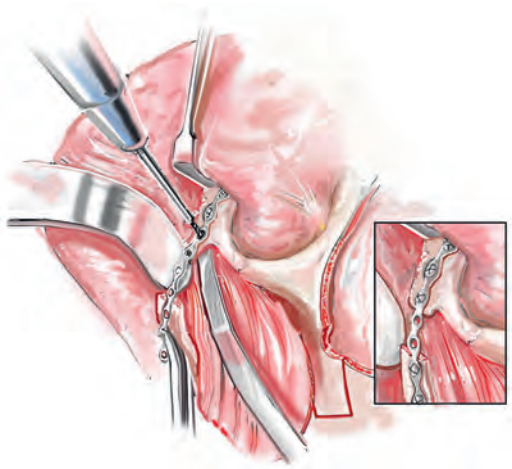
Step 22. Upper midface advancement is established at the zygomatic arches (Fig. 30-36). The amount of advancement at each zygomatic arch is measured with a caliper. A titanium plate is conformed to extend from the anterior maxilla across the arch and surgical gap to the posterior zygoma on each side. The plate is secured with titanium screws.

Step 23. Additional segmental osteotomies of the orbits are often required. Occasionally, the lateral superior orbits have further dysplasia and segmental osteotomies with reshaping for reconstruction is necessary (Fig. 30-37, *A*). If so, the lateral orbital segments are removed with a reciprocating saw.

• **Figure 30-35** Step 21.

Additional segmental orbital osteotomies are then completed with a reciprocating saw (Fig. 30-37, *B*). The lateral orbital rim and superior orbital rim segments are further reshaped with a rotary drill (Fig. 30-37, *C*). The segments are fixed with titanium plates and screws (Fig. 30-37, *D*).

Step 24. Stabilization of the midface advancement at the upper orbital tenon extension is accomplished

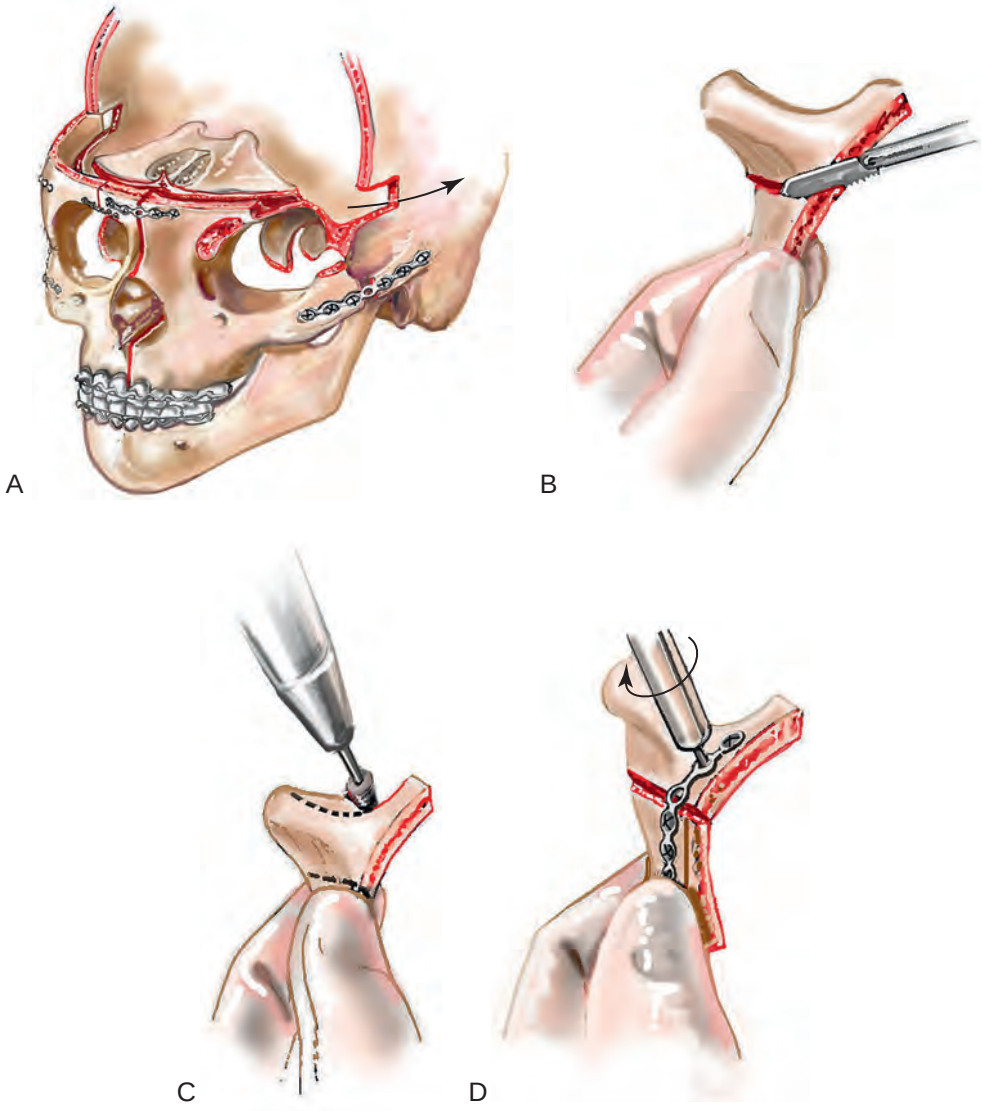


• **Figure 30-36** Step 22.

(Fig. 30-38). The desired advancement is measured with a caliper; a miniplate is adapted to bridge the gap between the tenon extension and the posterior cranial vault. Titanium screws are used for plate stabilization.

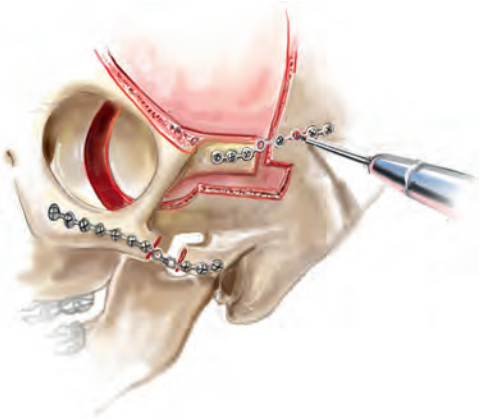
Step 25. Hyperplastic ethmoidal air cells are debrided as needed in patients with orbital hypertelorism (Fig. 30-39). With visualization through the anterior cranial base, rongeurs are used to debride hyperplastic ethmoidal air cells and to reduce midorbital hypertelorism.

Step 26. The opening between the anterior cranial fossa and the nasal cavity is managed (Fig. 30-40, *A* and *B*). The surgeon irrigates and suctions the intranasal cavity through the anterior cranial base exposure. My usual approach is to then apply a

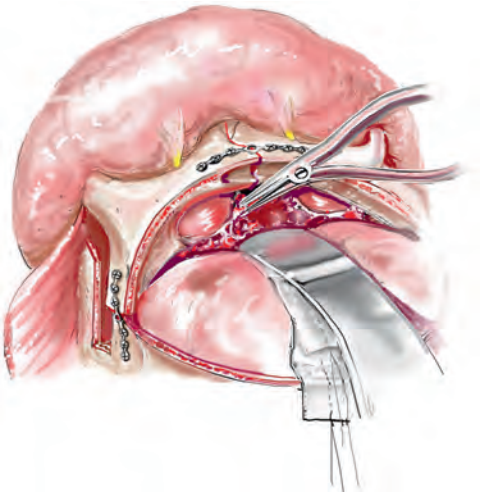


• **Figure 30-37 A** through **D,** Step 23.

sheet of Gelfoam to separate the opening between the two cavities (Fig. 30-40, *A*). I then inject fibrin glue over the Gelfoam to seal the separation (Fig. 30-40, *B*). Other methods of managing the separation between the nasal cavity and the anterior cranial fossa may be used, depending on the clinical circumstances (e.g., cranial bone grafts, soft-tissue flaps).



• **Figure 30-38** Step 24.



• **Figure 30-39** Step 25.

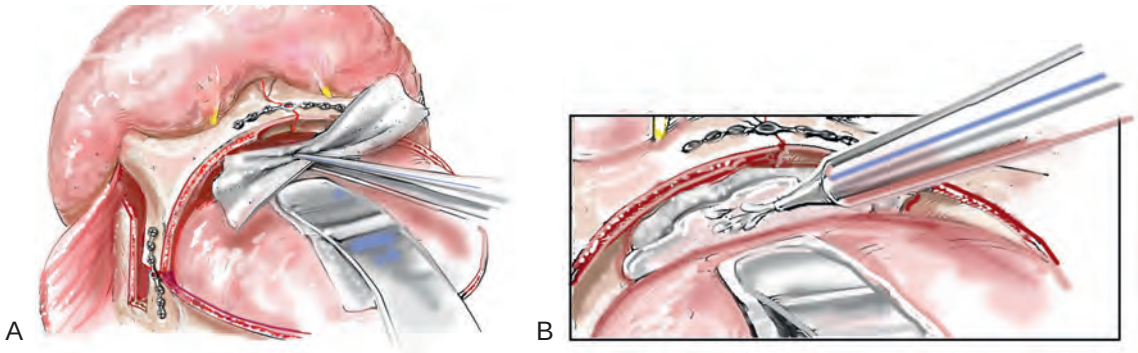
Step 27. The anterior cranial vault is reshaped, advanced, and secured in place (Fig. 30-41, *A, B,* and *C*). With the use of a reciprocating saw, osteotomies of the removed anterior cranial vault are completed (Fig. 30-41, *A*). Rotary drill recontouring is also accomplished to achieve the desired shape (Fig. 30-41, *B*). Split- or full-thickness cranial bone graft is harvested and used as needed to fill in defects, and fixation is accomplished with plates and screws. Split-thickness cranial bone graft is also placed and fixed in the midzygomatic arch segmental defects (Fig. 30-41, *C*).

Step 28. Lateral canthopexies are completed (Fig. 30-42). Two holes are drilled at each new frontozygomatic suture region. The lateral canthi are identified with a skin hook through the coronal incision. A figure-of-eight wire suture is placed through each lateral canthus through the coronal incision. Each lateral canthus is fixed by passing the wire through the drill holes in the frontozygomatic suture.

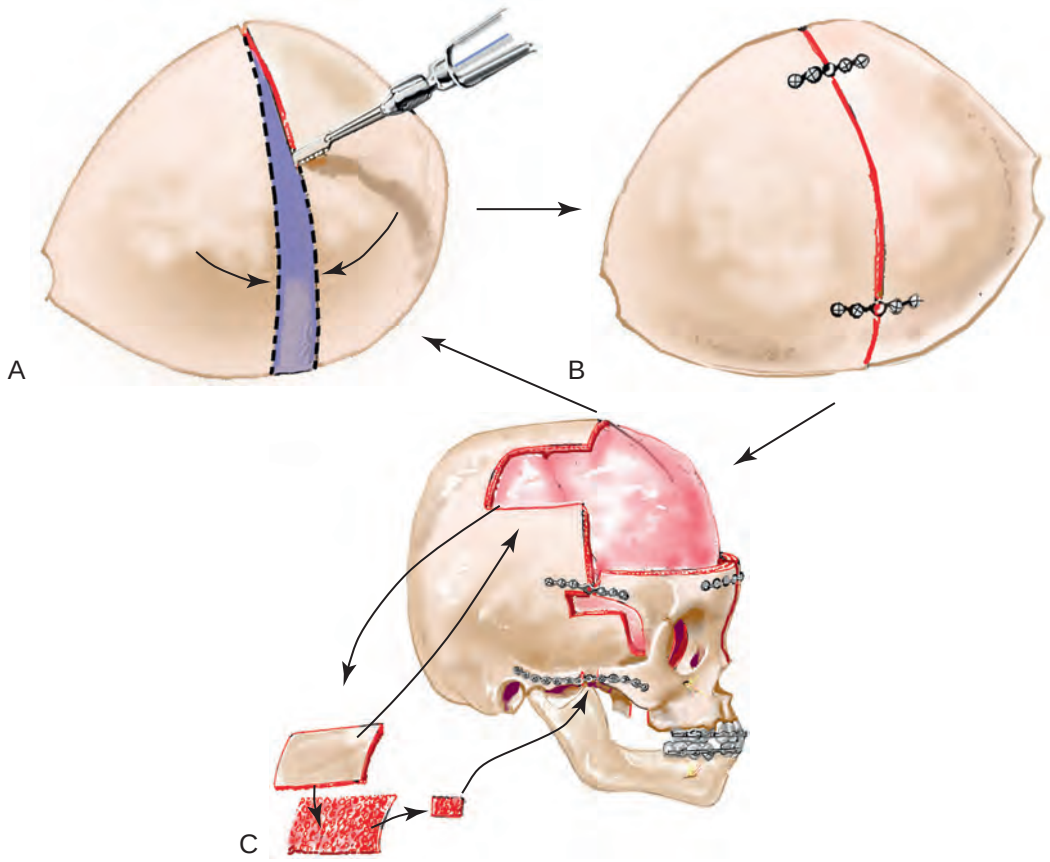
Step 29. Each temporalis muscle is resecured to bone (Fig. 30-43). The temporalis muscles are repositioned anteriorly and secured to the lateral orbital rims and the temporal bones with interrupted sutures.

Step 30. The scalp wound is closed (Fig. 30-44). Suction drains are placed through the posterior scalp flap; one is placed on each side. One drain is placed under the anterior flap, and the other is placed below the posterior flap. The galea closure is completed with interrupted sutures, and the skin layer closure is completed with staples or resorbable sutures in accordance with surgeon preference.

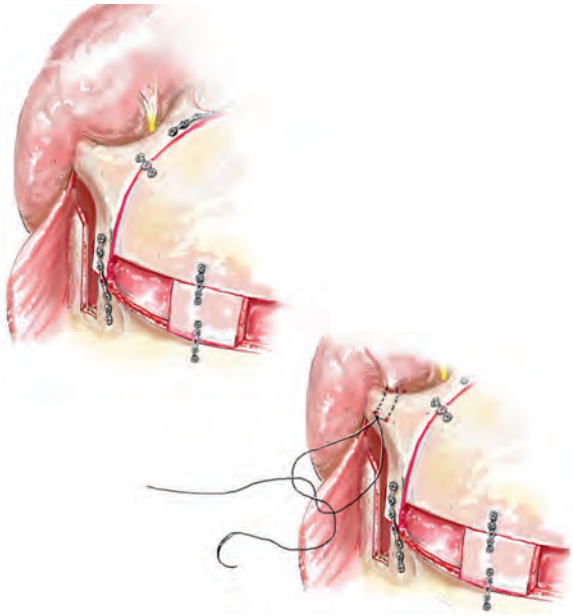
An overview of the skeletal morphology is shown before (Fig. 30-45, *A*) and after (Fig. 30-45, *B*) Facial Bipartition (FB) osteotomies and anterior cranial vault reshaping, repositioning, and stabilization. The locations of the proposed osteotomies are indicated.



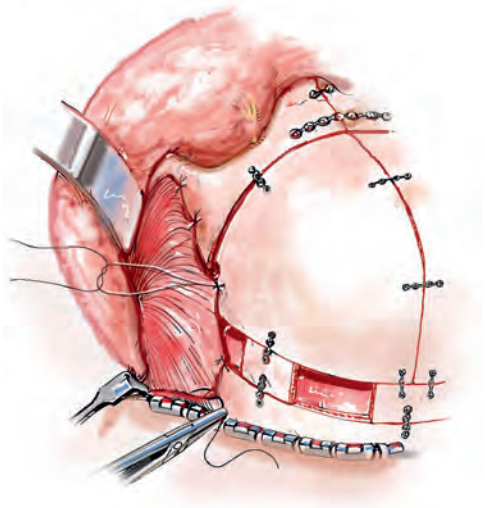
• **Figure 30-40 A** and **B,** Step 26.



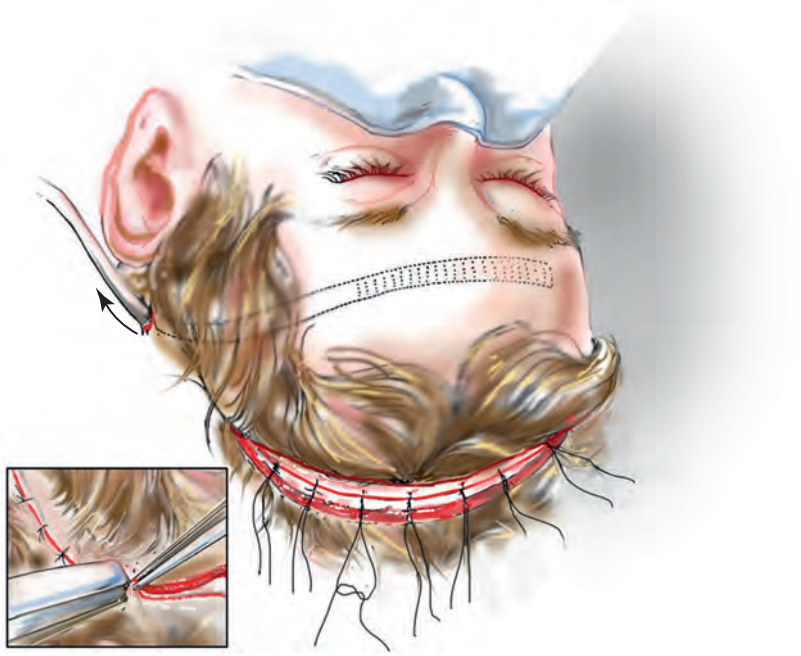
• **Figure 30-41 A, B,** and **C,** Step 27.



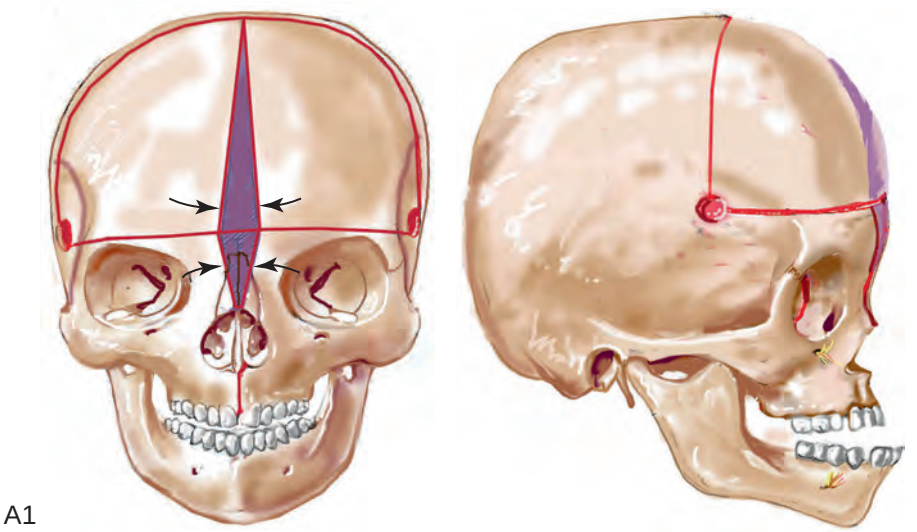
• **Figure 30-42** Step 28.



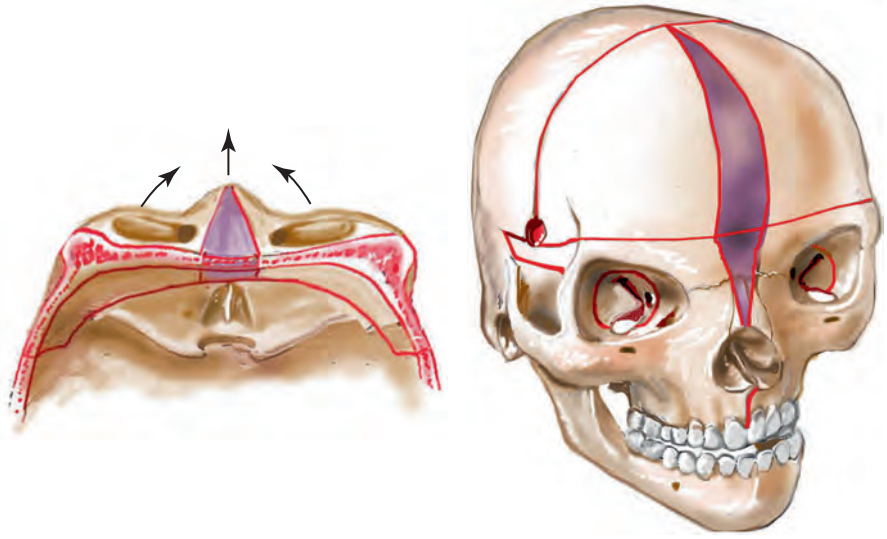
• **Figure 30-43** Step 29.



• **Figure 30-44** Step 30.

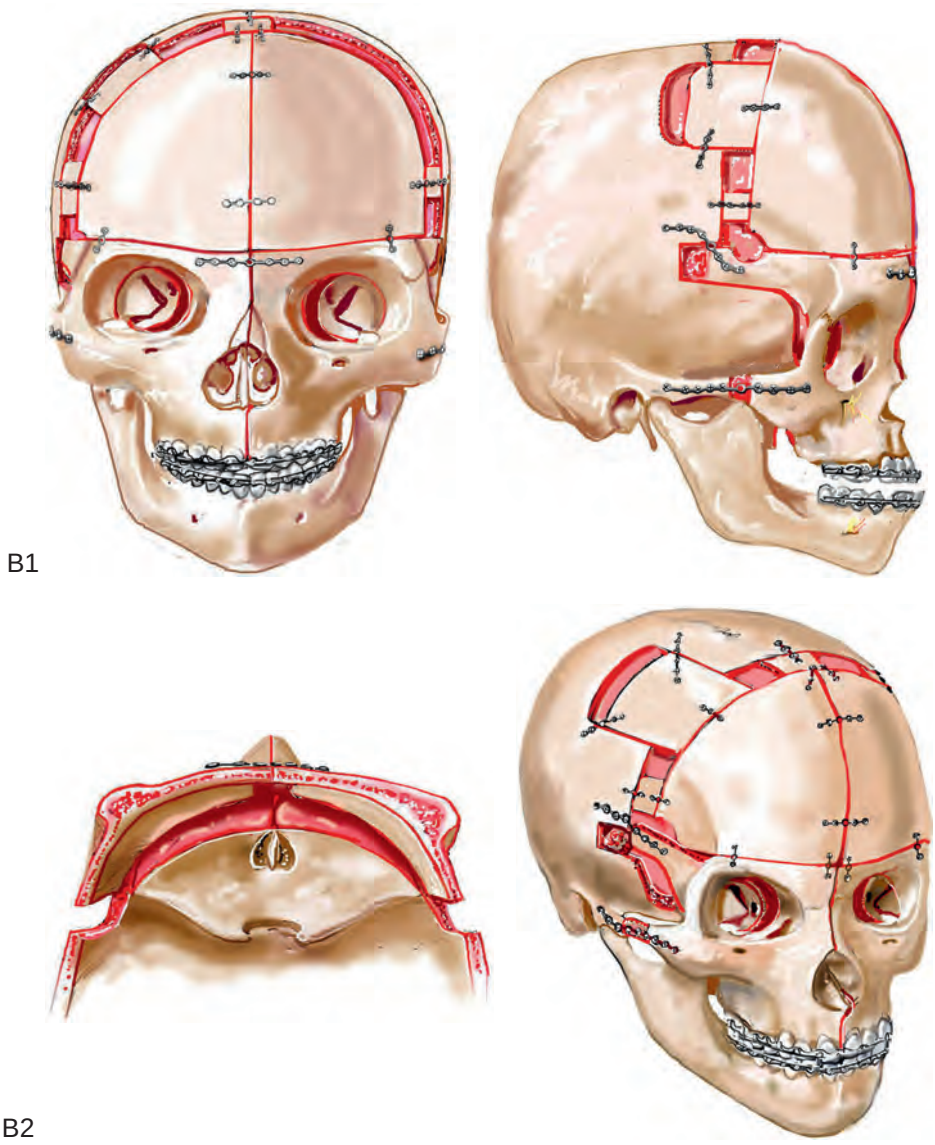


A1



A2

• **Figure 30-45** An overview of the skeletal morphology is shown **A,** before and **B,** after facial bipartition osteotomy with repositioning and stabilization.



• **Figure 30-45, cont'd**

Staging of Reconstruction for Crouzon Syndrome

Primary Brain Decompression: Cranio–Orbital Reshaping during Infancy

The initial surgery for a child with a craniosynostosis syndrome generally requires cranial suture release, simultaneous anterior cranial vault and upper orbital osteotomies, and reshaping and advancement for brain decompression.[200,203,205,216,274] There continues to be a search for minimally invasive approaches to the surgical decompression of the brain in the infant with syndromal craniosynostosis; the goal is to limit morbidity while achieving morphologic correction or at least improvement. Current minimally invasive techniques that are under consideration include endoscopic suture release; spring-mediated cranioplasty; and DO procedures. These techniques must be compared with the standard "open" approach of cranio–orbital reshaping carried out by an experienced craniofacial team; this is described later in this chapter. In addition, the preferred timing for brain decompression (i.e., <6 months; 6 to 12 months, >12 months) remains undetermined. Current studies support the 6- to 12-month time frame for first-stage brain decompression whenever feasible. Operating at an earlier age is more likely to result in the need for early reoperation and may involve increased perioperative risk.

In the infant with Crouzon syndrome who presents with bicoronal synostosis, my preference is to carry out the primary cranio–orbital reshaping when the child is 9 to 12 months old unless clear signs of increased intracranial pressure are identified earlier during his or her life.[153,200,203,205,216] The standard technique of reshaping of the upper three quarters of the orbits and the tenon extensions is geared toward decreasing the bitemporal and anterior cranial base width while achieving anteroposterior advancement. This

increases the depth of the anterior cranial base and the upper orbits with at least some improvement of ocular proptosis. The anterior cranial vault is then reconstructed in accordance with the patient's morphologic needs. The goals at this stage are to provide increased space in the anterior fossa for the brain; to increase orbital volume for better eye protection; and to improve the morphology of the forehead and the upper orbits. By allowing additional growth to occur before surgery (i.e., by waiting until the child is 9 to 12 months old), the reconstructed cranial vault and the upper orbital shape are better maintained with less need for repeat craniotomy procedures during early childhood.

Further Craniotomy for Brain Decompression during Childhood

After the initial suture release and the reshaping of the cranio–orbital region during infancy, the child is observed clinically at set intervals by the craniofacial surgeon, the pediatric neurosurgeon, the pediatric ophthalmologist, and the developmental specialist; he or she also undergoes interval CT scanning. Should signs of increased intracranial pressure develop, urgent brain decompression via further cranial vault expansion and reshaping is performed.[64,87,137,200,203,205,216] When increased intracranial pressure occurs, the suspected location of brain compression influences the region of the skull for which further expansion and reshaping is planned. If progressive brain compression is judged to be anterior, then additional forehead and upper orbital osteotomies with reshaping for expansion are carried out. The technique is similar to that described earlier in this chapter. If the problem is posterior, then expansion of the occipital region with the patient in the prone position is required. The repeat craniotomy is often complicated by brittle irregular cortical bone, which lacks a diploic space and which may contain sharp spicules that pierce the dura; by the presence of previously placed fixation hardware in the operative field; and by convoluted thin dura that is compressed against or herniated into the inner table of the skull. This results in a greater potential for dural tears during the calvarectomy than would normally occur during a primary procedure.

Management of the Upper Midface Deformity during Childhood

The approach selected to manage upper midface deficiencies and anomalies and residual cranial vault dysplasia should depend on the extent and location of dysmorphology.[200,203,205] A main objective of this phase of reconstruction is to normalize the orbits, the zygomas, and the cranial vault. The correction of the maxillomandibular deformity will require orthognathic surgery that includes a separate Le Fort I osteotomy. The selection of either an MB osteotomy (with or without additional orbital segmentation), an FB osteotomy (with or without additional orbital segmentation), or an extracranial Le Fort III osteotomy to manage the basic horizontal, transverse, and vertical upper and midface deficiencies and anomalies will depend on the original malformation, the previous procedures that have been carried out, and the effects of ongoing growth. The final reconstruction of the upper midface deformities in a patient with Crouzon syndrome can be managed when he or she is as young as 7 to 10 years old. (See previous sections in this chapter concerning upper midface reconstruction options).

Orthognathic Procedures to Achieve Facial Balance, an Improved Airway, and Definitive Occlusal Correction

Although the mandible has a normal basic growth potential in a patient with Crouzon syndrome, the maxilla does not. An Angle class III malocclusion caused by deficient maxillary growth is to be expected. A segmented Le Fort I osteotomy for maxillary horizontal advancement, transverse widening, and vertical adjustment is generally required in combination with an osseous genioplasty (vertical reduction and horizontal advancement) to further correct the lower face dysmorphology.[200,203,205] Secondary deformity of the mandible is also frequent and requires simultaneous sagittal ramus osteotomies. If correction of chronic nasal obstruction is required, then septoplasty, inferior turbinate reduction, the widening of the pyriform rims, and nasal floor recontouring are simultaneously completed (see Chapter 10). The definitive orthognathic and intranasal procedures are carried out in conjunction with orthodontic treatment that is planned for completion at the time of early skeletal maturity (i.e., ≈13 to 15 years in girls and ≈14 to 16 years in boys).

Assessment of Results in the Patient with Crouzon Syndrome

CT scans have provided the quantitative assessment of baseline craniofacial abnormalities in patients with Crouzon syndrome as well as of surgical results after first-stage cranio–orbital reconstruction.[216] The purposes of the quantitative assessment of the craniofacial complex—whether by CT scan or by anthropometric, cephalometric, or model analysis—are to help predict growth patterns, to confirm or refute clinical impressions, to help with treatment planning, and to provide a framework for the objective assessment of the immediate and long-term reconstructive results.

Quantitative Assessment of Presenting Crouzon Deformity and Surgical Results on the Basis of Computed Tomography Scan Analysis after First-Stage Cranio–Orbital Reconstruction

Waitzman and colleagues developed a method of analysis based on CT scan measurements and that allows for a quantitative assessment of the upper and midface skeleton

in both the horizontal and transverse planes.[272,273] This method of quantitative CT scan analysis was used to document the differences in the cranio–orbito–zygomatic region between children with Crouzon syndrome who had not yet undergone reconstruction and age-matched controls.[29] Morphologic results that were achieved in those children 1 year after undergoing standard suture release and anterior cranial vault and upper orbital procedures to reshape these regions were also evaluated.[216]

The preoperative CT scan measurements of the children with untreated Crouzon syndrome confirmed a widened anterior cranial vault at 108% of normal and a cranial length that averaged only 92% of normal.[29] In comparison with age-matched controls, orbital measurements revealed a widened anterior interorbital width at 122% of normal, an increased intertemporal width at 121% of normal, globe protrusion at 119% of normal, and a short medial orbital wall length at only 86% of normal. The distance between the zygomatic buttresses and the interarch widths were found to be increased at 106% and 103% of normal, respectively. The zygomatic arch lengths were substantially shortened at only 87% of the values of age-matched controls. These findings confirmed clinical observations of brachycephalic anterior cranial vaults with shallow and frequently hyperteloric orbits and globe proptosis. In general, the upper midface in patients with Crouzon syndrome is horizontally retrusive and transversely wide, which is reflected in wide and shortened zygomas. The same quantitative CT scan assessment was carried out in the children with Crouzon syndrome more than 1 year after they underwent anterior cranial vault and upper orbital osteotomies with reshaping to compare their values to the new age-matched control values.[216] No significant improvement in the cranio–orbito–zygomatic measurements was demonstrated.

Quantitative Intracranial Volume Measurements Before and After Cranio–Orbital Reshaping in Children with Crouzon Syndrome

In a previous study, the intracranial volumes in children with Crouzon syndrome before and after standard cranio–orbital reshaping procedures was documents.[209] The intracranial volumes were also compared with those of an age- and gender-matched cohort, and the rate of cranial expansion with growth was reviewed. The study included 13 children who presented sequentially with Crouzon syndrome and who subsequently underwent a standard first-stage cranio–orbital reconstruction by the author (Posnick) in conjunction with a pediatric neurosurgeon. The average age at the time of operation was 13 months (range, 6 to 46 months). Postoperative clinical follow up ranged from 12 to 60 months at the time of the study's completion. Of the children with Crouzon syndrome who were evaluated preoperatively, 12 out of 13 had intracranial volume values that were greater than the mean. When comparing postoperative volumes with the normative data, all 13 maintained volumes that were at or greater than the mean. When reviewing the cranial capacity of each patient with Crouzon syndrome over time, 5 of the 13 patients approximated the normal growth curve, whereas 6 of the 13 patients exceeded it. According to the findings of the study, for the majority of children who were born with Crouzon syndrome, the cranial capacity will exceed the mean early during life and expand at a rapid rate in conjunction with cranio–orbital decompression and reconstruction. The biologic explanation for these unexpected findings remains unclear. It should be noted that other researchers have studied the intracranial volume of children who were born with craniosynostosis.[18,20,21,24,60,64,67,73,80,85-87,93,137,140,141,210,246,247]

Quantitative Assessment of Presenting Deformity during the Mixed Dentition in Children with Crouzon Syndrome and Surgical Results after Monobloc Osteotomy on the Basis of Computed Tomography Scan Analysis

During their middle childhood years, a group of children with Crouzon syndrome were assessed via quantitative CT scan measurements. They were found to have cranial vault lengths that averaged only 87% of the values of the age-matched norms. The medial orbital walls were horizontally short at 87% of normal, whereas the extent of globe protrusion was excessive at 134% of age-matched norms. The zygomatic arch lengths averaged only 84% of normal. These findings confirmed horizontal anteroposterior deficiency of the upper face and the midface.

After they underwent MB osteotomies in combination with anterior cranial vault reshaping and advancement carried out through an intracranial approach, the children's cranio–orbito–zygomatic measurements were re-evaluated. The mean cranial length that was initially achieved after MB osteotomy was 98%, and at 1 year it was 92% of the control value. As compared with age-matched controls, the orbital measurements reflected improvements in both the midorbital hypertelorism (the mid-interorbital width was 97% initially after operation and 102% at 1 year), and the orbital proptosis (early after surgery, their values were 86% of the values for age-matched normal children; they were 92% at 1 year). The medial orbital wall length initially normalized at 101% and later at 97% of normal values. The zygomatic arch length initially corrected at 106% and later to 101% of normal.

Staging of Reconstruction for Apert Syndrome

Primary Brain Decompression: Cranio–Orbital Reshaping during Infancy

The initial craniofacial procedure for patients with Apert syndrome generally includes cranial suture release as well as simultaneous anterior cranial vault and upper three-quarter orbital osteotomies, with reshaping and advancement for brain decompression [202] This author's preference is to carry out these procedures when the child is 9 to 12 months old, unless signs of increased intracranial pressure are identified earlier during the child's life. The main goals at this stage

are to decompress the brain by providing increased space in the anterior cranial vault and to increase the orbital volume for improved eye protection. The fronto–orbital surgical technique is similar to that described for Crouzon syndrome (see the section about Crouzon syndrome earlier in this chapter).

Further Craniotomy for Brain Decompression during Childhood

As described previously for Crouzon syndrome, the initial suture release, brain decompression, and cranio–orbital reshaping are carried out during infancy (i.e., 9 to 12 months of age).[202] The child is observed clinically at intervals by the craniofacial surgeon, the pediatric neurosurgeon, the pediatric ophthalmologist, and the developmental pediatrician, and interval CT scanning is also performed. Should signs of increased intracranial pressure develop, further brain decompression with reshaping of the cranial vault to expand the intracranial volume is performed. In patients with Apert syndrome, the posterior cranial vault more commonly requires expansion at this stage. The technique is similar to what has been previously described (see the section about Crouzon syndrome earlier in this chapter).

Management of the Upper Midface Deformity during Childhood

For almost all patients with Apert syndrome, facial bipartition osteotomies in combination with further cranial vault reshaping allow for a better correction of the dysmorphology than can be achieved through any other upper midface procedure (i.e., MB or Le Fort III).[202] When using FB osteotomies, the correction of the concave midface arc of rotation is also possible. This further reduces the stigmata of the "flat, wide, and retrusive" facial appearance associated with Apert syndrome. The FB allows the orbits and the zygomas as units to shift to the midline (i.e., correction of hypertelorism) while the maxillary arch is simultaneously widened (i.e., relief of the V-shaped upper midface). Horizontal advancement of the reassembled upper midface complex is then possible to improve orbital depth and zygomatic length. The forehead is generally flat, tall, and retruded, with a constricting band just above the supraorbital ridge. The reshaping of the anterior cranial vault is also simultaneously carried out. A Le Fort III osteotomy is virtually never adequate for an ideal correction of the residual upper midface anomalies documented among patients with Apert syndrome (see earlier section in this chapter concerning upper midface reconstruction options).

Orthognathic Procedures to Achieve Facial Balance, an Improved Airway, and Definitive Occlusal Correction

The mandible has normal basic growth potential in patients with Apert syndrome, but it is generally secondarily deformed. The extent of maxillary deficiency will result in an Angle Class III anterior open-bite malocclusion. A segmented Le Fort I osteotomy is required for horizontal advancement, transverse widening, and vertical adjustment. This is often in combination with an osseous genioplasty and sagittal split ramus osteotomies of the mandible. Septoplasty and inferior turbinate reduction are generally carried out to improve nasal breathing, and the recontouring of the nasal floor and the pyriform rims is also required (see Chapter 10). The definitive orthognathic and intranasal surgery is carried out in conjunction with orthodontic treatment and planned for completion at the time of early skeletal maturity (i.e., ≈13 to 15 years of age).[202]

Assessment of Results in the Patient with Apert Syndrome

Quantitative Assessment of Presenting Apert Deformity and Surgical Results on the Basis of Computed Tomography Scan Analysis after First-Stage Cranio–Orbital Reconstruction

In a previously published study, a method of quantitative CT scan analysis was applied to document the differences in the cranio–orbito–zygomatic region between children with Apert syndrome who had not been operated on and age-matched controls.[29] Eight consecutive infants and young children with Apert syndrome who underwent a standard cranio–orbital procedure by a single craniofacial surgeon (Posnick) in conjunction with one of three pediatric neurosurgeons over a 4-year period were reviewed 1 year after surgery.[217] The series included seven girls and one boy, with an average age at surgery of 12 months (range, 9 to 23 months). The average postoperative follow-up period was 34 months (range, 12 to 48 months) at the close of the study. Preoperative and postoperative (>1 year) CT scans were compared with those of age-matched controls, and percentages of normal were then compared for significant differences. Two of the children had clear evidence of hydrocephalus and required ventriculoperitoneal shunting before the cranio–orbital reconstruction at 3 and 5 months of age. A third child had mildly increased ventricular size, but clinical correlation did not suggest the need for shunting.

Significant preoperative morphologic findings included a wide anterior cranial vault at 110% of normal, a maximum cranial length that averaged only 90% of normal, a substantially widened anterior interorbital width at 117% of normal, an increased lateral interorbital distance at 112% of normal, and a widened bitemporal width at 122% of normal.[217] Globe protrusion was significant at 121% of normal, and the medial orbital wall length was less than normal at 92%. In the upper midface zygomatic region, both the width between the zygomatic buttresses and the interarch width were found to be increased at 109% of normal, whereas the zygomatic arch lengths were substantially shortened at 79% of normal. The measurements confirmed the clinical observations of brachycephalic and

hyperteloric anterior cranial vaults, orbits, and zygomas accompanied by globe proptosis and midface deficiency. Results of surgical reconstruction as documented by CT scan measurements demonstrated that, at more than 1 year after surgery, none of the craniofacial measurements had significantly improved ($P < .05$) as compared with those of the new age-matched controls.

Quantitative CT scan measurements of the cranio–orbito–zygomatic region confirmed the clinical findings in the unoperated children to be brachycephalic anterior cranial vaults and upper face hypertelorism of the orbits and the zygomas,) with ocular proptosis and a flat midface.[217] It was found that cranial vault and upper orbital reshaping before 1 year of age does not achieve or maintain a corrected shape in the craniofacial skeleton in children with Apert syndrome. Although the cranio–orbito–zygomatic dysmorphology did not worsen when analyzed at least 1 year after surgery, values remained far from those of the new age-matched controls, thereby confirming the need for a staged reconstructive approach.

Quantitative Intracranial Volume Measurements Before and After Cranio–Orbital Reshaping in Children with Apert Syndrome

In published studies, a proven method for obtaining intracranial volumes with the use of CT scan measurement was applied to a consecutive series of children with Apert syndrome before any craniofacial procedures were performed.[209] A standard cranio–orbital operation was performed for each child, and this was followed by longitudinal follow up and the remeasurement of the intracranial volume at least 1 year later. The patients' intracranial volumes were also compared with those of an age-matched cohort, and each patient's cranial growth velocity was reviewed. The study included six girls and two boys with an average age at operation of 12 months (range, 9 to 23 months). The average postoperative follow-up duration at the close of the study was 34 months (range, 12 to 48 months).[209]

Preoperative intracranial volume in the patients with Apert syndrome ranged from 393 mL in a 2-month-old girl to 1715 mL in a 28-month-old girl.[209] A comparison of the preoperative intracranial volume in patients with Apert syndrome with those of the age- and gender-matched cohort group showed that six of the eight patients had values of at least two standard deviations above the mean. Interestingly, the other two were infants who underwent ventriculoperitoneal shunting for hydrocephalus earlier in life. Their measured preoperative intracranial volumes were two standard deviations below the mean. When the patients' postoperative intracranial volumes were compared with those of the cohort group, all eight achieved values of at least two standard deviations above the mean. The majority of the measured preoperative and postoperative intracranial volume values of the patients with Apert syndrome followed a growth curve that greatly exceeded the rate expected for normal children. In three of the patients, cranial vault growth velocity seemed to match closely that expected for a normal child but with a starting point determined by their preoperative values.

The findings confirmed that untreated patients with Apert syndrome are generally macrocephalic early during their lives, that standard cranio–orbital procedures carried out during childhood do not alter this trend, and that continued cranial volume expansion often exceeds the mean. The ability to develop "normal" intracranial volume standards and to identify variations from normal in specific syndromes and in individual patients before and after surgery continues to be elusive.[18,20,21,24,60,73,80,85,86,93,96,140,141,210,246,247]

Quantitative Assessment of Presenting Deformity during the Mixed Dentition in Children with Apert Syndrome and Surgical Results after Facial Bipartition Osteotomy on the Basis of Computed Tomography Scan Analysis

In published studies, children with Apert syndrome were assessed during their middle childhood years with the use of quantitative CT scan measurements.[220] Many of these children's measurements varied from normal as compared with those of age-matched controls. The orbital measurements showed a substantially increased anterior interorbital width (123% of normal), an increased mid-interorbital width (122% of normal), and an increased intertemporal width (126% of normal). The globe protrusion beyond the sagittal plane of the lateral orbital walls was excessive (142% of normal). There was also a short medial orbital wall length (85% of normal). The width between the lateral orbital walls was excessive at 111% of normal. Zygomatic arch lengths were substantially shortened at 83% of normal.

After undergoing FB osteotomies with three-dimensional repositioning in combination with cranial vault reshaping, CT scan measurements were again taken both early after the operation and 1 year later.[220] An analysis of the measurements showed an improvement toward the normal range. As compared with the values of age-matched controls, the orbital measurements reflected the correction of the hypertelorism; the anterior interorbital width early after operation was 106% of normal and later 105% of normal. The mid-interorbital width initially improved to 106% of normal and later to 100% of normal. The width between the lateral orbital walls stabilized at 108% of normal, and the intertemporal width was 115% of normal, which was an improvement over the preoperative value of 126% of normal. The zygomatic arch length was initially overcorrected at 110% of normal and then stabilized at 103% of normal.

Further studies were carried out to evaluate the presence of extradural (retrofrontal) dead space after the FB osteotomy to reconstruct the upper midface deformity associated with Apert syndrome.[208] Seven patients with Apert syndrome (mean age, 8 years) underwent FB osteotomies with advancement. Extradural (retrofrontal) dead space was measured from a reproducible axial CT scan slice for each patient at standard postoperative intervals (1 to 2 weeks, 6 to 8 weeks, and 1 year). An initial extradural (retrofrontal) dead space was identified early after surgery in each patient.

There was good resolution of the dead space by the 6- to 8-week postoperative interval through expansion of the dura and the frontal lobes of the brain. The dead space was confirmed to be closed in all patients at the 1-year postoperative interval. The morbidity of the same consecutive series of patients was then reviewed. For the seven children, there were no deaths, cardiopulmonary sequelae, injuries to the brain or eyes, new seizure activity, or central nervous system problems.

Conclusions

The preferred approach to the management of the individual with a craniosynostosis syndrome is to stage reconstruction to coincide with craniofacial growth patterns, visceral function (e.g., cognitive, vision, breathing, swallowing, speech, chewing, hearing), and psychosocial needs. The recognition of the advantages of a staged approach to reconstruction serves to clarify the objectives of each phase of treatment for the surgeons, the craniofacial team, and the family. By continuing to define a rationale for the timing, techniques, and extent of intervention and then objectively evaluating both head and neck function and morphologic (aesthetic) outcomes, the quality of life for children who are born with craniosynostosis syndromes will improve.

References

1. Ades LC, Mulley JC, Senga IP, et al: Jackson-Weiss syndrome: Clinical and radiological findings in a large kindred and exclusion of the gene from 7p21 and 5qter. *Am J Med Genet* 51:121, 1994.
2. Al-Saleh A, Riekstins A, Forrest CR, et al: Sleep-related disordered breathing in children with syndromic craniosynostosis. *J Craniomaxillofac Surg* 39:153–157, 2011.
3. Ahmad F, Cobb AR, Mills C, et al: Frontofacial monoblock distraction in the very young: A review of 12 consecutive cases. *Plast Reconstr Surg* 129:488E, 2012.
4. Anderson PJ, Hall CM, Evans RD, et al: The cervical spine in Saethre–Chotzen syndrome. *Cleft Palate Craniofac J* 34:79, 1997.
5. Anderson PJ, Netherway DJ, Cox TC, et al: Do craniosynostosis syndrome phenotypes with both FGFR2 and TWIST mutations have a worse clinical outcome? *J Craniofac Surg* 17:166, 2006.
6. Anderson PM, Geiger I: Craniosynostosis: A survey of 204 cases. *J Neurosurg* 22:229, 1956.
7. Apert E: De l'acrocephalosyndactlie. *Bull Mem Soc Med Hop Paris* 23:1310, 1906.
8. Arnaud E, Marchac D, Renier D: Reduction of morbidity of the frontofacial monobloc advancement in children by the use of internal distraction. *Plast Reconstr Surg* 120:1009–1026, 2007.
9. Atkins FRB: Hereditary craniofacial dysostosis or Crouzon disease. *Med Press Circ* 195:118, 1937.
10. Bachmayer DI, Ross RB: Stability of Le Fort III advancement surgery in children with Crouzon, Apert, and Pfeiffer syndromes. *Cleft Palate J* 23(Suppl 1): 69–74, 1986.
11. Bachmayer DI, Ross RB, Munro IR: Maxillary growth following Le Fort III advancement surgery in Crouzon, Apert, and Pfeiffer syndromes. *Am J Orthod Dentofacial Orthop* 90:420–430, 1986.
12. Baldwin JL: Dysostosis craniofacialis of Crouzon: A summary of recent literature and case reports with involvement of the ear. *Laryngoscope* 78:1660, 1968.
13. Barr M, Jr, Kreiborg S: The cervical spine in the Apert syndrome. *Am J Med Genet* 43:704, 1992.
14. Bernard JP, Levaillant JM: Prenatal diagnosis of craniosynostosis. *Neurochirurgie* 52:246, 2006.
15. Berrada A: Erode sur les craniostenoses en milieum marocain: A propos de 87 observations (these medicale), Paris, 1963-1964.
16. Bertelsen TI: The premature synostosis of the cranial sutures. *Acta Ophthalmol* 51(Suppl):87, 1958.
17. Bigot C: L'acrocephalo-syndactylie (these pour le doctorat en medicine). Paris, Faculte de Medicine 1922.
18. Blinkov SM, Glezer II, Haigh B: *The human brain: A quantitative handbook*, New York, 1968, Basic Books.
19. Bradley JP, Gabbay JS: Monobloc advancement by distraction osteogenesis decreases morbidity and relapse. *Plast Reconstr Surg* 118:1585, 2006.
20. Bray PF, Shields WD, Wolcott GJ, et al: Occipitofrontal head circumference: An accurate measure of intracranial volume. *J Pediatr* 75:303, 1969.
21. Breiman RS, Beck JW, Korobkin M, et al: Volume determinations using computed tomography. *Am J Roentgenol* 138:329, 1982.
22. Broder HL: Psychological research of children with craniofacial anomalies: Review, critique and implications for the future. *Cleft Palate Craniofac J* 34:402, 1997.
23. Bu BH, Kaban LB, Vargervik K: Effect of Le Fort III osteotomy on mandibular growth in patients with Crouzon and Apert syndrome. *J Oral Maxillofac Surg* 47:666, 1989.
24. Buda FB, Reed JC, Rabe EF: Skull volume in infants. *Am J Dis Child* 129:1171, 1975.
25. Camfield PR, Camfield CS, Cohen M Jr: Neurologic aspects of craniosynostosis. In Cohen MM, Jr, MacLean RE, editors: *Craniosynostosis: Diagnosis, evaluation and management*, ed 2, New York, 2000, Oxford University Press, 14, pp 177–183.
26. Campbell JW, Albright AL, Losken HW, et al: Intracranial hypertension after cranial vault decompression for craniosynostosis. *Pediatr Neurosurg* 22:270, 1995.
27. Carinci F, Pezzetti F, Locci P, et al: Apert and Crouzon syndromes: Clinical findings, genes and extracellular matrix. *J Craniofac Surg* 16:361, 2005.
28. Carpenter G: Two sisters showing malformations of the skull and other congenital abnormalities. *Rep Soc Study Dis Child (London)* 1:110, 1901.
29. Carr M, Posnick JC, Pron G, Armstrong D: Cranio-orbito-zygomatic measurements from standard CT scans in unoperated Crouzon and Apert infants: Comparison with normal controls. *Cleft Palate Craniofac J* 29(2):129–136, 1992.
30. Chen CP, Lin SP, Su YN, et al: A cloverleaf skull associated with Crouzon syndrome. *Arch Dis Child Fetal Neonatal Ed* 91:98, 2006.
31. Chen P, Por Y, Liou EJ, Chang FC: Maxillary distraction osteogenesis in the adolescent cleft patient: Three-dimensional computed tomography analysis of linear and volumetric changes over five years. *Cleft Palate Craniofac J* 40:445–454, 2011.
32. Chin M, Toth BA: Le Fort III advancement with gradual distraction using internal devices. *Plast Reconstr Surg* 100:819, 1997.
33. Chotzen F: Eine eigenartige familiare entwicklungsstorung. (Akrocephalosyndaktylie, dystosis craniofacialis und hypertelorismus). *Monatschr Kinderheilkd* 55:97, 1932.
34. Chumas PD, Cinalli G, Arnaud E, et al: Classification of previously unclassified cases of craniosynostosis. *J Neurosurg* 86:197–181, 1997.
35. Clinical practice guideline: Diagnosis and management of childhood obstructive sleep apnea syndrome. *Pediatrics* 109:704–712, 2002.

36. Coccaro PJ, McCarthy JG, Epstein FJ, et al: Early and late surgery in craniofacial dysostosis: A longitudinal cephalometric study. *Am J Orthod* 77:421–436, 1980.
37. Cohen MM, Jr: Perspectives on craniosynostosis. *West J Med* 132:508, 1980.
38. Cohen MM, Jr: Let's call it "Crouzonodermoskeletal syndrome" so we won't be prisoners of our own conventional terminology. *Am J Med Genet* 84:74, 1999.
39. Cohen MM, Jr: Carpenter syndrome. In Cohen MM, Jr, MacLean RE, editors: *Craniosynostosis: diagnosis, evaluation and management*, ed 2, New York, 2000, Oxford University Press, 29, pp 377–379.
40. Cohen MM, Jr: Saethre-Chotzen syndrome. In Cohen MM, Jr, MacLean RE, editors: *Craniosynostosis: Diagnosis, evaluation and management*, ed 2, New York, 2000, Oxford University Press, 28, pp 374–376.
41. Cohen MM, Jr: Jackson-Weiss syndrome. *Am J Med Genet* 100:325–329, 2001.
42. Cohen MM, Jr: FGFs/FGFRs and associated disorders. In Epstein CJ, Erickson RP, Wynshaw-Boris A, editors: *Inborn errors of development*, New York, 2004, Oxford University Press, Ch 33, pp 380–400.
43. Cohen MM, Jr: Invited comment: Perspectives on craniosynostosis. *Am J Med Genet* 136A:313–326, 2005.
44. Cohen MM, Jr: Craniofacial abnormalities. In Gilbert-Barness E, editor: *Potter's pathology of the fetus, infant, and child,* vol I, ed 2, Philadelphia, 2007, Mosby/Elsevier, 20, pp 885–918.
45. Cohen MM, Jr: Cloverleaf skull: Etiologic heterogeneity and pathogenetic variability. *J Craniofac Surg* 20(Suppl):652–656, 2009.
46. Cohen MM, Jr: Apert, Crouzon, and Pfeiffer syndromes. Monographs in human genetics, In Muenke M, Kress W, Collmann H, Solomon BD, editors: *Craniosynostosis: Molecular genetics, diagnosis, and treatment.* 19:67–88, 2011.
47. Cohen MM, Jr: No man's craniosynostosis: The arcane of sutural knowledge. *J Craniofac Surg* 23:338–348, 2012.
48. Cohen MM, Jr, MacLean RE: *Craniosynostosis: Diagnosis, evaluation and management*, ed 2, New York, 2000, Oxford University Press.
49. Connolly JP, Gruss J, Seto ML, et al: Progressive postnatal craniosynostosis and increased intracranial pressure. *Plast Reconstr Surg* 113:1313,2004.
50. Correa Normando AD, da Silva Filho OG, Capelozza Filho L: Influence of surgery on maxillary growth in cleft lip and/or palate patients. *J Craniomaxillofac Surg* 20:111, 1992.
51. Crouzon O: Une nouvelle famille atteinte de dysostose craniofaciale hereditaire. *Arch Med Enfant* 18:540, 1915.
52. Crouzon O: Sur la dysostose cranio-faciale hereditaire et sur les rapports avec l'acrocephalosyndactylie. *Bull Mem Soc Med Hop Paris* 48:1568, 1932.
53. Crouzon O: Les dysostose prechordales. *Bull Acad Med* 115:696, 1936.
54. Cruveiller J: La Maladie d'Apert-Crouzon (these medicale). Paris 1954.
55. Cushing H: Neurosurgery. In Keen WW, editor: *Surgery: Its principles and practice*, Philadelphia, 1908, WB Saunders, p 254.
56. David DJ, Cooter RD: Craniofacial infections in 10 years of transcranial surgery. *Plast Reconstr Surg* 80:213, 1987.
57. David DJ, Sheen R: Surgical correction of Crouzon syndrome. *Plast Reconstr Surg* 85:344, 1990.
58. David LR, Velotta E, Weaver RG, Jr, et al: Clinical findings precede objective diagnostic testing in the identification of increased ICP in syndromic craniosynostosis. *J Craniofac Surg* 13:676, 2002.
59. DeGunten P: Contribution a l'etude des malformations de la face et des maxillaires dans la dysostose craniofaciale. *Ann Otolaryngol* 57:1056, 1938.
60. Dekaban AS: Tables of cranial and orbital measurements, cranial volume and derived indexes in males and females from 7 days to 20 years of age. *Ann Neurol* 2:485, 1977.
61. Devine P, Bhan I, Feingold M, et al: Completely cartilaginous trachea in a child with Crouzon syndrome. *Am J Dis Child* 138:40, 1984.
62. Dickerman RD, Lefkowitz M, Arinsburg SA, Schneider SJ: Chiari malformation and odontoid panus causing craniovertebral stenosis in a child with Crouzon syndrome. *J Clin Neurosci* 12:963, 2005.
63. Diner PA: Le Fort III advancement with gradual distraction using internal devices [discussion]. *Plast Reconstr Surg* 100:831, 1997.
64. Disler DG, Marr DS, Rosenthal DI: Accuracy of volume measurements of computed tomography and magnetic resonance imaging phantoms by three-dimensional reconstruction and preliminary clinical application. *Invest Radiol* 29:739, 1994.
65. Farkas LG, Posnick JC: Growth and development of regional units in the head and face based on anthropometric measurements. *Cleft Palate Craniofac J* 29:301–302, 1992.
66. Farkas LG, Posnick JC, Hreczko T: Anthropometric growth study of the head. *Cleft Palate Craniofac J* 29:303–307, 1992.
67. Farkas LG, Posnick JC, Hreczko T: Growth patterns in the orbital region: A morphometric study. *Cleft Palate Craniofac J* 29:315–317, 1992.
68. Farkas LG, Posnick JC, Hreczko T: Growth patterns of the face: a morphometric study. *Cleft Palate Craniofac J* 29:308–314, 1992.
69. Fearon JA: The Le Fort III osteotomy: To distract or not to distract? *Plast Reconstr Surg* 107:1091–1103, discussion 1104-1096, 2001.
70. Fearon JA: Halo distraction of the Le Fort III in syndromic craniosynostosis: A long-term assessment. *Plast Reconstr Surg* 115:1524, 2005.
71. Fearon JA, Whitaker LA: Complications with facial advancement: A comparison between the Le Fort III and monobloc advancements. *Plast Reconstr Surg* 91:990, 1993.
72. Fearon JA, Yu J, Bartlett SP, et al: Infections in craniofacial surgery: A combined report of 567 procedures from two centers. *Plast Reconstr Surg* 100:862, 1997.
73. Fernandez F, Moule N, Singhi S: Roentgenographic skull volume in Jamaican children between the ages of 1 month and 5 years. *West Indian Med J* 33:227, 1984.
74. Ferreira JC, Carter SM: Second-trimester molecular prenatal diagnosis of sporadic Apert syndrome following suspicious ultrasound findings. *Ultrasound Obstet Gynecol* 14:426, 1999.
75. Figueroa AA, Polley JW, Friede H, et al: Long-term skeletal stability after maxillary advancement with distraction osteogenesis using a rigid external distraction device in cleft maxillary deformities. *Plast Reconstr Surg* 114:1382, 2004.
76. Figueroa AA, Polley JW, Ko EW: Maxillary distraction for the management of cleft maxillary hypoplasia with a rigid external distraction system. *Semin Orthod* 5:46, 1999.
77. Fishman MA, Hogan GR, Dodge PR: The concurrence of hydrocephalus and craniosynostosis. *J Neurosurg* 34:621, 1971.
78. Flippen JH, Jr: Craniofacial dysostosis of Crouzon: Report of a case in which the malformation occurred in four generations. *Pediatrics* 5:90, 1950.
79. Flores RL, Shetye PR, Zeitler D, et al: Airway changes following Le Fort III distraction osteogenesis for syndromic craniosynostosis: A clinical and cephalometric study. *Plast Reconstr Surg* 124:590–601, 2009.
80. Fok H, Jones BM, Gault DG, et al: Relationship between intracranial pressure and intracranial volume in craniosynostosis. *Br J Plast Surg* 45:394, 1992.
81. Freitas EC, Nascimento SR, de Mello MP, Gil-da-Silva-Lopes VL: Q289P mutation in FGFR2 gene causes Saethre-Chotzen syndrome: some considerations about familial heterogeneity. *Cleft Palate Craniofac J* 43:142, 2006.
82. Fujisawa H, Hasegawa M, Kida S, Yamashita J: A novel fibroblast growth factor receptor 2 mutation in Crouzon syndrome associated with Chiari type I malformation and syringomyelia. *J Neurosurg* 97:396, 2002.
83. Funato N, Nohtomi-Ohyama J, Ohyama K: Monozygotic twins concordant for Crouzon syndrome. *Am J Med Genet A* 133:225, 2005.
84. Garcin M, Thurel R, Rudeaux P: Sur en cas asole de dysostose craniofaciale (maladie de Crouzon) avec extradactylie. *Bull Soc Med Hop* 56:1458, 1932.

85. Gault DT, Brunelle F, Renier D, et al: The calculation of intracranial volume using CT scans. *Childs Nerv Sys* 4:271, 1988.
86. Gault DT, Renier D, Marchac D, et al: Intracranial volume in children with craniosynostosis. *J Craniofac Surg* 1:1, 1990.
87. Gault DT, Renier D, Marchac D, Jones BM: Intracranial pressure and intracranial volume in children with craniosynostosis. *Plast Reconstr Surg* 90:230–271, 1992.
88. Genest P, Mortezai MA, Tremblay M: Le syndrome d'Apert (acrocephalosyndactyly). *Arch Fr Pediatr* 23:887, 1966.
89. Gillies HD, Harrison SH: Operative correction by osteotomy of recessed malar maxillary compound in case of oxycephaly. *Br J Plast Surg* 3:123, 1950.
90. Golabi M, Chierici G, Ousterhout DK, et al: Radiographic abnormalities of Crouzon syndrome: A survey of 23 cases. *Proc Greenwood Genet Center* 3:102, 1984.
91. Golabi M, Edwards MSB, Ousterhout DK: Craniosynostosis and hydrocephalus. *Neurosurgery* 21:63, 1987.
92. Goldberg S, Shatz A, Picard E, et al: Endoscopic findings in children with obstructive sleep apnea: Effects of age and hypotonia. *Pediatr Pulmonol* 40:205–210, 2005.
93. Gordon IRS: Measurement of cranial capacity in children. *Br J Radiol* 39:377, 1966.
94. Gorlin RJ, Cohen MM, Jr, Levin LS: *Syndromes of the head and neck*, ed 3, New York, 1990, Oxford University Press, pp 524-525.
95. Gorry MC, Preston RA, White GJ, et al: Crouzon syndrome: Mutations in two splice forms of FGFR2 and a common point mutation shared with Jackson-Weiss syndrome. *Hum Mol Genet* 4:1387, 1995.
96. Gosain AK, McCarthy JG, Glatt P, et al: A study of intracranial volume in Apert syndrome. *Plast Reconstr Surg* 95:284, 1995.
97. Gosain AK, Santoro TD, Havlik RJ, et al: Midface distraction following Le Fort III and monobloc osteotomies: Problems and solutions. *Plast Reconstr Surg* 109:1797, 2002.
98. Gray TL, Casey T, Selva D, et al: Ophthalmic sequelae of Crouzon syndrome. *Ophthalmology* 112:1129, 2005.
99. Green SM: Pathological anatomy of the hands in Apert syndrome. *J Hand Surg* 7:450, 1982.
100. Grenet H, Leveuf J, Issac G: Etude anatomique de la maladie de Crouzon. *Bull Soc Pediatr* 32:343, 1934.
101. Gulleminault C, Lee JH, Chan A: Pediatric obstructive sleep apnea syndrome. *Arch Pediatr Adolesc Med* 159:775–785, 2005.
102. Harada K, Baba Y, Ohyama K, et al: Maxillary distraction osteogenesis for cleft lip and palate children using an external, adjustable, rigid distraction device: A report of 2 cases. *J Oral Maxillofac Surg* 59:1492, 2001.
103. Harada K, Sato M, Omura K: Long-term maxillomandibular skeletal and dental changes in children with cleft lip and palate after maxillary distraction. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 102:292, 2006.
104. Harris V, Beligere N, Pruzansky S: Progressive generalized bony dysplasia in Apert syndrome. *Birth Defects* 14:175, 1977.
105. Hoeve HL, Joosten KF, van den Berg S: Management of obstructive sleep apnea syndrome in children with craniofacial malformation. *Int J Pediatr Otorhinolaryngol* 49(Suppl 1):S59–S61, 1999.
106. Hoeve HL, Pijpers M, Joosten KF: OSAS in craniofacial syndromes: An unresolved problem. *Int J Pediatr Otorhinolaryngol* 67(Suppl 1):111–113, 2003.
107. Hoffman HJ, Mohr G: Lateral canthal advancement of the supraorbital margin: A new corrective technique in the treatment of coronal synostosis. *J Neurosurg* 45:376, 1976.
108. Hogan GR, Bauman ML: Hydrocephalus in Apert syndrome. *J Pediatr* 79:782, 1971.
109. Hogeman KE, Willmar K: On Le Fort III osteotomy for Crouzon disease in children: Report of a four-year follow-up in one patient. *Scand J Plast Reconstr Surg* 8:169–172, 1974.
110. Hollier L, Kelly P, Babigumira E, et al: Minimally invasive Le Fort III distraction. *J Craniofac Surg* 13:44, 2002.
111. Hopper RA: Frontofacial monobloc distraction in the very young: A review of 12 consecutive cases. *Plast Reconstr Surg* 129(3):498e–501e, 2012.
112. Huang CS, Harikrishnan P, Liao YF, et al: Long-term follow-up after maxillary distraction osteogenesis in growing children with cleft lip and palate. *Cleft Palate Craniofac J* 44:274, 2007.
113. Iannetti G, Fadda T, Agrillo A, et al: Le Fort III advancement with and without osteogenesis distraction. *J Craniofac Surg* 17:536–543, 2006.
114. Ishii K, Kaloust S, Ousterhout DK, Vargervik K: Airway changes after Le Fort III osteotomy in craniosynostosis syndromes. *J Craniofac Surg* 7:363–370, discussion 371, 1996.
115. Ito S, Sekido K, Kanno H, et al: Phenotypic diversity in patients with craniosynostoses unrelated to Apert syndrome: The role of fibroblast growth factor receptor gene mutations. *J Neurosurg* 102(1 Suppl):23, 2006.
116. Jabs EW, Li X, Scott AF, et al: Jackson-Weiss and Crouzon syndromes are allelic with mutations in fibroblast growth factor receptor 2. *Nat Genet* 8:275–279, 1994.
117. Jensen JN, McCarthy JG, Grayson BH, et al: Bone deposition/generation with Le Fort III (midface) distraction. *Plast Reconstr Surg* 119:298–307, 2007.
118. Juberg RC, Chambers SR: An autosomal recessive form of craniofacial dysostosis (the Crouzon syndrome). *J Med Genet* 10:89, 1973.
119. Kaban LB, Conover M, Mulliken J: Midface position after Le Fort III advancement: A long-term follow-up study. *Cleft Palate J* 23(Suppl):75–77, 1986.
120. Kaban LB, West B, Conover M, et al: Midface position after Le Fort III advancement. *Plast Reconstr Surg* 73:758–767, 1984.
121. Kaloust S, Ishii K, Vargervik K: Dental development in Apert syndrome. *Cleft Palate Craniofac J* 34:117, 1997.
122. Kapp-Simon KA, Figueroa A, Jocher CA, et al: Longitudinal assessment of mental development in infants with nonsyndromic craniosynostosis with and without cranial release and reconstruction. *Plast Reconstr Surg* 92:831, 1993.
123. Kasser J, Upton J: The shoulder, elbow, and forearm in Apert syndrome. *Clin Plast Surg* 18:381, 1991.
124. Khong JJ, Anderson P, Gray TL, et al: Ophthalmic findings in Apert syndrome prior to craniofacial surgery. *Am J Ophthalmol* 142:328, 2006.
125. Kirkpatrick WN, Koshy CE, Waterhouse N, et al: Pediatric transcranial surgery: A review of 114 consecutive procedures. *Br J Plast Surg* 55:561, 2002.
126. Kolar JC, Munro IR, Farkas LG: Patterns of dysmorphology in Crouzon syndrome: An anthropometric study. *Cleft Palate J* 25:235–244, 1988.
127. Kreiborg S: Crouzon syndrome: A clinical and roentgencephalometric study. *Scand J Plast Reconstr Surg* 18(Suppl):1, 1981.
128. Kreiborg S, Barr M, Jr, Cohen M Jr: Cervical spine in the Apert syndrome. *Am J Med Genet* 43:704, 1992.
129. Kreiborg S, Cohen MM, Jr: The infant Apert skull. *Neurosurg Clin North Am* 2:551–554, 1991.
130. Kreiborg S, Cohen MM, Jr: The oral manifestations of the Apert syndrome. *J Craniofac Genet Dev Biol* 12:41, 1992.
131. Kreiborg S, Marsh JL, Cohen MM, Jr, et al: Comparative three-dimensional analysis of CT scans of the calvaria and cranial base in Apert and Crouzon syndromes. *J Craniomaxillofac Surg* 21:181–188, 1993.
132. Kreiborg S, Prydsoe U, Dahl E, Fogh-Anderson P: Calvarium and cranial base in Apert syndrome: An autopsy report. *Cleft Palate J* 13:296–303, 1976.
133. Lane LC: Pioneer craniectomy for relief of mental imbecility due to premature sutural closure and microcephalus. *JAMA* 18:49, 1892.
134. Lannelongue M: De la craniectomie dans la microcephalie. *Compte Rendu Acad Sci* 110:1382, 1890.
135. Lauritzen C, Lilja J, Jarlstedt J: Airway obstruction and sleep apnea in children with craniofacial anomalies. *Plast Reconstr Surg* 77:1–6, 1986.
136. Lee Y, Kim WJ: How to make the blockage between the nasal cavity and intracranial

space using a four-layer sealing technique. *Plast Reconstr Surg* 117:233, 2006.
137. Lichtenberg R: Radiographic du crane de 226 enfants normaux de la naissance a 8 ans: Impressions digitformes, capacite: Angles et indices (thesis), Paris, 1960, University of Paris.
138. Lo LJ, Chen YR: Airway obstruction in severe syndromic craniosynostosis. *Ann Plast Surg* 43:258–264, 1999.
139. Lo LJ, Marsh JL, Yoon J, et al: Stability of fronto-orbital advancement in non-syndromic bilateral coronal synostosis: A quantitative three-dimensional computed tomographic study. *Plast Reconstr Surg* 98:393, 1996.
140. Mackinnon IL: The relation of the capacity of the human skull to its roentgenological length. *Am J Radiol* 74:1026, 1955.
141. Mackinnon IL, Kennedy JA, Davies TV: The estimation of skull capacity from roentgenological measurements. *Am J Radiol* 76:303, 1956.
142. Mah J, Kasser J, Upton J: The foot in Apert syndrome. *Clin Plast Surg* 18:391, 1991.
143. Mantilla-Capacho JM, Arnaud L, Diaz-Rodriguez M, Barros-Nunez P: Apert syndrome with preaxial polydactyly showing the typical mutation Ser252Trp in the FGFR2 gene. *Genet Couns* 16:403, 2005.
144. Marchac D: Radical forehead remodeling for craniosynostosis. *Plast Reconstr Surg* 62:335–338, 1978.
145. Marchac D, Renier D, Jones BM: Experience with the "floating forehead." *Br J Plast Surg* 41:1–15, 1988.
146. Marsh JL, Gado M: Surgical anatomy of the craniofacial dysostoses: Insights from CT scans. *Cleft Palate J* 19:212–221, 1982.
147. Marsh JL, Galic M, Vannier MW: Surgical correction of craniofacial dysmorphology of Apert syndrome. *Clin Plast Surg* 18:251, 1991.
148. Marsh JL, Vannier MW: The "third" dimension in craniofacial surgery. *Plast Reconstr Surg* 71:759–767, 1983.
149. Mathijssen I, Arnaud E, Marchac D, et al: Respiratory outcome of midface advancement with distraction: A comparison between Le Fort III and frontofacial monobloc. *J Craniofac Surg* 17:880–882, 2006.
150. Matsumoto K, Nakanishi H, Koizumi Y, et al: Segmental distraction of the midface in a patient with Crouzon syndrome. *J Craniofac Surg* 13:273, 2002.
151. Mavili ME, Tuncbilek G, Vargel I: Rigid external distraction of the midface with direct wiring of the distraction unit in patients with craniofacial dysplasia. *J Craniofac Surg* 14:783, 2003.
152. McCarthy JG, Coccaro PJ, Eptstein F, Converse JM: Early skeletal release in the infant with craniofacial dysostosis: The role of the sphenozygomatic suture. *Plast Reconstr Surg* 62:335–346, 1978.
153. McCarthy JG, Epstein FJ, Sadove M, et al: Early surgery for craniofacial synostosis: An 8-year experience. *Plast Reconstr Surg* 73:521, 1984.
154. McCarthy JG, Glasberg SB, Cutting CB, et al: Twenty-year experience with early surgery for craniosynostosis: I. Isolated craniofacial synostosis—results and unsolved problems. *Plast Reconstr Surg* 96:272, 1995.
155. McCarthy JG, Grayson B, Bookstein F, et al: Le Fort III advancement osteotomy in the growing child. *Plast Reconstr Surg* 74:343, 1984.
156. McCarthy JG, La Trenta GS, Breitbart AS, et al: The Le Fort III advancement osteotomy in the child under 7 years of age. *Plast Reconstr Surg* 86:633–646, discussion 647-649, 1990.
157. Meazzini MC, Mazzoleni F, Caronni E, Bozzetti A: Le Fort III advancement osteotomy in the growing child affected by Crouzon and Apert syndromes: Presurgical and postsurgical growth. *J Craniofac Surg* 16:369, 2005.
158. Meling TR, Hans-Erik H, Per S, Due-Tonnessen BJ: Le Fort III distraction osteogenesis in syndromal craniosynostosis. *J Craniofac Surg* 17:28, 2006.
159. Meyers GA, Day D, Goldberg R, et al: FGFR2 exon III and IIIc mutations in Crouzon, Jackson-Weiss, and Pfeiffer syndromes: Evidence for missense changes, insertions and a deletion due to alternative RNA splicing. *Am J Hum Genet* 58:491, 1996.
160. Meyers GA, Orlow SJ, Munro IR, et al: Fibroblast growth factor receptor 3 (FGFR3) transmembrane mutation in Crouzon syndrome with acanthosis nigricans. *Nat Genet* 11:462, 1995.
161. Montaut J, Stricker M: Dysmorphies craniofaciales: Les synostoses prematurees (craniostenoses et facio-stenoses). *Neurochirurgie* 23(Suppl 2):1, 1977.
162. Moore MH. Upper airway obstruction in the syndromal craniosynostoses. *Br J Plast Surg* 46:355–362, 1993.
163. Moretti G, Sraeffen J: Dysostose craniofaciale de Crouzon et syringomylie: Association chez le frere et la soeur. *Presse Med* 67:376, 1959.
164. Moss ML: The pathogenesis of premature cranial synostosis in man. *Acta Anat (Basel)* 37:351–370, 1959.
165. Muenke M, Gripp KW, McDonald-McGinn DM, et al: A unique point mutation in the fibroblast growth factor receptor 3 gene (FGFR3) defines a new craniosynostosis syndrome. *Am J Hum Genet* 60:555, 1997.
166. Muenke M, Schell U: Fibroblast-growth-factor receptor mutations in human skeletal disorders. *Trends Genet* 11:308, 1995.
167. Muenke M, Schell U, Hehr A, et al: A common mutation in the fibroblast growth factor receptor 1 gene in Pfeiffer syndrome. *Nat Genet* 8:269, 1994.
168. Muenke M, Schell U, Robin NH, et al: Variable clinical spectrum in Pfeiffer syndrome: Correlation between phenotype and genotype. *Proc Green Genet Ctr* 15:126, 1995.
169. Mulliken JB, Godwin SL, Pracharktam N, et al: The concept of the sagittal orbital-globe relationship in craniofacial surgery. *Plast Reconstr Surg* 97:700, 1996.
170. Murovic JA, Posnick JC, Drake JM, et al: Hydrocephalus in Apert syndrome: A retrospective review. *Pediatr Neurosurg* 19(3):151–155, 1993.
171. Murray JE, Swanson LT: Midface osteotomy and advancement for craniosynostosis. *Plast Reconstr Surg* 41:299–306, 1968.
172. Nelson TE, Mulliken JB, Padwa BL: Effect of midfacial distraction on the obstructed airway in patients with syndromic bilateral coronal synostosis. *J Oral Maxillofac Surg* 66:2318–2321, 2008.
173. Nixon GM, Brouillette RT: Sleep. 8: Pediatric obstructive sleep apnea. *Thorax* 60511–60516, 2005.
174. Noetzel MJ, Marsh JL, Palkes H, et al: Hydrocephalus and mental retardation in craniosynostosis. *J Pediatr* 107:885, 1985.
175. Nout E, Bouw FP, Veenland JF, et al: Three-dimensional airway changes after Le Fort III advancement in syndromic craniosynostosis patients. *J Plast Reconstr Surg* 126:564–571, 2010.
176. Nout E, Cesteleyn LL, Van der Wal KG, et al: Advancement of the midface, from conventional Le Fort III osteotomy to Le Fort III distraction: Review of the literature. *Int J Oral Maxillofac Surg* 37:781–789, 2008.
177. Oldridge M, Wilkie AOM, Slaney SF, et al: Mutations in the third immunoglobulin domain of the fibroblast growth factor receptor-2 gene in Crouzon syndrome. *Hum Mol Genet* 4:1077, 1995.
178. Ortiz-Monasterio F, Fuente del Campo A: Refinements on the monobloc orbitofacial advancement. In Caronni EP, editor: *Craniofacial surgery*, Boston, 1985, Little, Brown, p 263.
179. Ortiz-Monasterio F, Fuente del Campo A, Carillo A: Advancement of the orbits and the midface in one piece, combined with frontal repositioning for the correction of Crouzon syndrome. *Plast Reconstr Surg* 61:507–516, 1978.
180. Ousterhout DK, Vargervik K, Clark S: Stability of the maxilla after Le Fort III advancement in craniosynostosis syndromes. *Cleft Palate J* 23(Suppl 1):91–101, 1986.
181. Padwa B: Effects of midface distraction osteogenesis on obstructive sleep apnea in patients with syndromic craniosynostosis. *J Oral Maxillofac Surg* 64:49, 2006.
182. Park WJ, Meyers GA, Li X, et al: Novel FGFR2 mutations in Crouzon and Jackson-Weiss syndromes show allelic heterogeneity and phenotypic variability. *Hum Mol Genet* 4:1229, 1995.
183. Park WJ, Theda C, Maestri NE, et al: Analysis of phenotypic features and FGFR2

mutations in Apert syndrome. *Am J Hum Genet* 57:321–328, 1995.
184. Paznekas WA, Cunningham ML, Howard TD, et al: Genetic heterogeneity of Saethre-Chotzen syndrome due to TWIST and FGFR mutations. *Am J Hum Genet* 62:1370, 1998.
185. Perkins JA, Sie KC, Milczuk H, et al: Airway management in children with craniofacial anomalies. *Cleft Palate Craniofac J* 34:135, 1997.
186. Peterson SJ, Pruzansky S: Palatal anomalies in the syndromes of Apert and Crouzon. *Cleft Palate J* 11:394, 1974.
187. Peterson-Falzone SJ, Pruzansky S, Purris P, et al: Nasopharyngeal dysmorphology in the syndromes of Apert and Crouzon. *Cleft Palate J* 18:237, 1981.
188. Pfeiffer RA: Dominant erbliche akrocephalosyndaktylie. *Z Kinderheilkd* 90:301–320, 1964.
189. Phillips JH, George AK, Tompson B: Le Fort III osteotomy or distraction osteogenesis imperfecta: Your choice. *Plast Reconstr Surg* 117:1255–1260, 2006.
190. Pijpers M, Poels PJ, Vaandrager JM, et al: Undiagnosed obstructive sleep apnea syndrome in children with syndromal craniofacial synostosis. *J Craniofac Surg* 15:670–674, 2004.
191. Pollack IF, Losken HW, Biglan AW: Incidence of increased intracranial pressure after early surgical treatment of syndromic craniosynostosis. *Pediatr Neurosurg* 24:202, 1996.
192. Polley JW, Figueroa AA: Management of severe maxillary deficiency in childhood and adolescence through distraction osteogenesis with an external, adjustable, rigid distraction device. *J Craniofac Surg* 8:181–185, discussion 186, 1997.
193. Pope AW, Ward J: Self-perceived facial appearance and psychosocial adjustment in preadolescents with craniofacial anomalies. *Cleft Palate Craniofac J* 34:396, 1997.
194. Posnick JC: Craniofacial dysostosis: Staging of reconstruction and management of the midface deformity. *Neurosurg Clin North Am* 2:683–702, 1991.
195. Posnick JC: The craniofacial dysostosis syndromes: Current reconstructive strategies. *Clin Plast Surg* 21(4):585–598, 1994.
196. Posnick JC: The effects of rigid fixation on the craniofacial growth of the rhesus monkeys [discussion]. *Plast Reconstr Surg* 93:11, 1994.
197. Posnick JC: Craniofacial dysostosis syndromes: A staged reconstructive approach. In Turvey TA, Vig KWL, Fonseca RJ, editors: *Facial clefts and craniosynostosis: Principles and management*, Philadelphia, 1996, WB Saunders.
198. Posnick JC: Monobloc and facial bipartition osteotomies: A step-by-step description of the surgical technique. *J Craniofac Surg* 7(3):229–250, 1996.
199. Posnick JC: Stability of fronto-orbital advancement in non-syndromic bilateral coronal synostosis: A quantitative three-dimensional computed tomographic study [discussion]. *Plast Reconstr Surg* 98:406, 1996.
200. Posnick JC: Crouzon syndrome: Basic dysmorphology and staging of reconstruction. *Techniques in Neurosurgery* 3:216–229, 1997.
201. Posnick JC: The craniofacial dysostosis syndromes: Staging of reconstruction and management of secondary deformities. *Clin Plast Surg* 24(3):429–446, 1997.
202. Posnick JC: Apert syndrome: Evaluation and staging of reconstruction. In Posnick JC, editor: *Craniofacial and maxillofacial surgery in children and young adults*, Philadelphia, 2000, WB Saunders Co, pp 308–342.
203. Posnick JC: Brachycephaly: Bilateral coronal synostosis without midface deficiency. In Posnick JC, editor: *Craniofacial and maxillofacial surgery in children and young adults*, Philadelphia, 2000, WB Saunders Co; pp 249–268.
204. Posnick JC: Cloverleaf skull anomalies: Evaluation and staging reconstruction. In Posnick JC, editor: *Craniofacial and maxillofacial surgery in children and young adults*. Philadelphia, 2000, WB Saunders Co, 17, pp 354–366.
205. Posnick JC: Crouzon syndrome: Evaluation and staging of reconstruction. In Posnick JC, editor: *Craniofacial and maxillofacial surgery in children and young adults*. Philadelphia, 2000, WB Saunders Co, pp 271–307.
206. Posnick JC: Pfeiffer syndrome: Evaluation and staging of reconstruction. In Posnick JC, editor: *Craniofacial and maxillofacial surgery in children and young adults*. Philadelphia, 2000, WB Saunders Co, 16, pp 343–353.
207. Posnick JC: The monobloc and facial bipartition osteotomies: A step-by-step description of the surgical technique. In Posnick JC, editor: *Craniofacial and maxillofacial surgery in children and young adults*. Philadelphia, 2000, WB Saunders Co, 18, pp 367–388.
208. Posnick JC, Al-Qattan MM, Armstrong D: Monobloc and facial bipartition osteotomies reconstruction of craniofacial malformations: A study of extradural dead space. *Plast Reconstr Surg* 97(6):1118–1128, 1996.
209. Posnick JC, Armstrong D, Bite U: Crouzon and Apert syndrome: Intracranial volume measurements prior to and after cranio-orbital reshaping in childhood. *Plast Reconstr Surg* 96(3):539–548, 1995.
210. Posnick JC, Bite NP, Nakano P, Davis J, Armstrong D: Indirect intracranial volume measurements using CT scans: Clinical applications for craniosynostosis. *Plast Reconstr Surg* 89(1):34–45, 1992.
211. Posnick JC, Farkas LG. The application of anthropometric surface measurements in craniomaxillofacial surgery. In Farkas LG, editor: *Anthropometry of the head and face*, New York, 1994, Raven Press.
212. Posnick JC, Farkas LG: Anthropometric surface measurements in the analysis of craniomaxillofacial deformities: Normal values and growth trends. In Posnick JC, editor: *Craniofacial and maxillofacial surgery in children and young adults*, Philadelphia, 2000, WB Saunders Co, pp 55–79.
213. Posnick JC, Goldstein JA, Armstrong D, Rutka JT: Reconstruction of skull defects in children and adolescents by the use of fixed cranial bone grafts: Long-term results. *Neurosurgery* 32(5):785–791, 1993.
214. Posnick JC, Goldstein JA, Clokie C: Advantages of the postauricular coronal incision. *Ann Plast Surg* 29(2):114–116, 1992.
215. Posnick JC, Goldstein JA, Clokie C: Refinements in pterygomaxillary dissociation for total midface osteotomies: Instrumentation, technique and CT scan analysis. *Plast Reconstr Surg* 91(1):167–172, 1993.
216. Posnick JC, Lin KY, Jhawar BJ, Armstrong D: Crouzon syndrome: Quantitative assessment of presenting deformity and surgical results based on CT scans. *Plast Reconstr Surg* 92(6):1027–1037, 1993.
217. Posnick JC, Lin KY, Jhawar BJ, Armstrong D: Apert syndrome: Quantitative assessment in presenting deformity and surgical results after first-stage reconstruction by CT scan. *Plast Reconstr Surg* 93:489–497, 1994.
218. Posnick JC, Ruiz R: The craniofacial dysostosis syndromes: Current surgical thinking and future directions. *Cleft Palate Craniofac J* 37(5):433, 2000.
219. Posnick JC, Ruiz R, Tiwana P: The craniofacial dysostosis syndromes: Stages of reconstruction. *Oral Maxillofac Surg Clin North Am* 475–491, 2004.
220. Posnick JC, Waitzman A, Armstrong D, Pron G: Monobloc and facial bipartition osteotomies: Quantitative assessment of presenting deformity and surgical results based on computer tomography scans. *J Oral Maxillofac Surg* 53(4):358–367, 1995.
221. Posnick JC, Yaremchuk M: The effect of non-resorbable internal fixation devices placed on and within a child's cranial vault: Brain function, morbidity and growth restrictions [editorial]. *Plast Reconstr Surg* 96:966, 1995.
222. Pugeaut R: Le probleme neuro chirurgical des craniostenoses. *Cahier Med Lyon* 44:3343, 1968.
223. Reardon W, Winter RM, Rutland P, et al: Mutations in the fibroblast growth factor receptor 2 gene cause Crouzon syndrome. *Nat Genet* 8:98, 1994.
224. Reddy BSN: An unusual association of acanthosis nigricans and Crouzon disease. *J Dermatol* 12:85, 1985.
225. Regnault F, Crouzon O: Etude sur un cas de dysostose craniofaciale hereditaire. *Ann Med Enfant* 43:676, 1927.
226. Renier D: Intracranial pressure in craniosynostosis: Pre- and postoperative recordings—correlation with functional

results. In Persing JA, Edgerton MT, Jane JA, editors: *Scientific foundations and surgical treatment of craniosynostosis*, Baltimore, 1989, Williams & Wilkins, pp 263–269.
227. Renier D, Arnaud E, Cinalli G, et al: Prognosis for mental function in Apert syndrome. *J Neurosurg* 85:66, 1996.
228. Renier D, Marchac D: Intracranial pressure recordings: Analysis of 300 cases. In Marchac D, editor: *Craniofacial surgery: Proceedings of the First International Congress on Craniomaxillofacial Surgery*, Heidelberg, 1988, Springer-Verlag.
229. Renier D, Marchac D: Longitudinal assessment of mental development in infants with nonsyndromic craniosynostosis with and without cranial release and reconstruction [discussion]. *Plast Reconstr Surg* 92:840, 1993.
230. Renier D, Sainte-Rose C, Marchac D, et al: Intracranial pressure in craniosynostosis. *J Neurosurg* 57:370, 1982.
231. Robin NH, Scott JA, Arnold JE, et al: Favorable prognosis for children with Pfeiffer syndrome types 2 and 3: Implications for classification. *Am J Med Genet* 75:240, 1998.
232. Rougerie J, Derome P, Anquez L: Craniostenosis et dysmorphies craniofaciales. Principes d'un nouvelle technique de traitement et ses resultats. *Neurochirurgie* 18:429, 1972.
233. Rutland P, Pulley LJ, Reardon W: Identical mutations in the FGFR2 gene cause both Pfeiffer and Crouzon syndrome phenotypes. *Nat Genet* 9:173–176, 1995.
234. Saethre H: Ein beitrag zum turmschadelproblem (pathogenese, erblichkeit und symptomatologie). *Deutsche Zeitschrift fur Nervenheilkunde* 117:533, 1931.
235. Satoh K, Mitsukawa N, Hosaka Y: Dual midfacial distraction osteogenesis: Le Fort III minus I and Le Fort I for syndromic craniosynostosis. *Plast Reconstr Surg* 111:1019, 2003.
236. Schell U, Hehr A, Feldman GJ, et al: Mutations in FGFR1 and FGFR2 cause familial and sporadic Pfeiffer syndrome. *Hum Mol Genet* 4:323, 1995.
237. Schiller JG: Craniofacial dysostosis of Crouzon: A case report and pedigree with emphasis on hereditary. *Pediatrics* 23:107, 1959.
238. Seruya M, Oh A, Boyajian M, et al: Long-term outcomes of primary craniofacial reconstruction for craniosynostosis: A 12-year experience. *Plast Reconstr Surg* 127:2397, 2011.
239. Shetye PR, Boutros S, Grayson BH, et al: Midterm follow-up of midface distraction for syndromic craniosynostosis: A clinical and cephalometric study. *Plast Reconstr Surg* 120:1621–1632, 2007.
340. Shetye PR, Davidson EH, Sorkin M, et al: Evaluation of three surgical techniques for advancement of the midface in the growing children with syndromic craniosynostosis. *Plast Reconstr Surg* 126:982–994, 2010.
241. Shetye PR, Grayson BH, McCarthy JG: Le Fort III distraction: Controlling position and path of the osteotomized midface segment on a rigid platform. *J Craniofac Surg* 21:1118–1121, 2010.
242. Shetye PR, Kadadia H, Grayson BH, McCarthy JG: A 10-year study of skeletal stability and growth of the mid face following Le Fort III advancement in syndromic craniosynostosis. *Plast Reconstr Surg* 126:973–981, 2010.
243. Shillito J, Jr, Matson DD: Craniosynostosis: A review of 519 surgical patients. *Pediatrics* 41:829, 1968.
244. Shin JH, Duncan CC, Persing J: Monobloc distraction: Technical modification and considerations. *J Craniofac Surg* 14:763, 2003.
245. Siddiqi SN, Posnick JC, Buncic R, et al: The detection and management of intracranial hypertension after initial suture release and decompression for craniofacial dysostosis syndromes. *Neurosurgery* 36(4):703–708, 1995.
246. Singal VK, Mooney MO, Burrows AM, et al: Age-related changes in intracranial volume in rabbits with craniosynostosis. *Plast Reconstr Surg* 100:1121, 1997.
247. Singhi S, Walia BNS, Singhi P, et al: A simple nonroentgenographic alternative for skull volume measurements in children. *Ind J Med Res* 82:150, 1985.
248. Slaney SF, Oldridge M, Hurst JA, et al: Differential effects of FGFR2 mutations on syndactyly and cleft plate in Apert syndrome. *Am J Med Genet* 58:923–932, 1996.
249. Spinelli HM, Irizarry D, McCarthy JG, et al: An analysis of extradural dead space after fronto-orbital surgery. *Plast Reconstr Surg* 93:1372, 1994.
250. Steinberger D, Mulliken JB, Muller U: Predisposition for cysteine substitutions in the immunoglobulin-like chain of FGFR2 in Crouzon syndrome. *Hum Genet* 96:113, 1995.
251. Stevens CA, Roeder ER: Ser351Cys mutation in the fibroblast growth factor receptor 2 gene results in severe Pfeiffer syndrome. *Clin Dysmorphol* 15:187, 2006.
252. Tessier P: Demonstration operations at Hopital Foch, Paris-Suresnes, Nov 1967.
253. Tessier P: Total facial osteotomy. Crouzon syndrome, Apert syndrome: Oxycephaly, scaphocephaly, turricephaly [French]. *Ann Chir Plast* 12:273–286, 1967.
254. Tessier P: *Dysostosis cranio-faciales (syndromes de Crouzon et d'Apert) osteotomies totales de la face. Transactions of the Fourth International Congress of Plastic and Reconstructive Surgery*, Rome, 1969, October 1967, Excerpta Medica Foundation, Amsterdam, pp 774–783.
255. Tessier P: Autogenous bone grafts taken from the calvarium for facial and cranial applications. *Plast Reconstr Surg* 48:224, 1971.
256. Tessier P: The definitive plastic surgical treatment of the severe facial deformities of craniofacial synostosis: Crouzon and Apert diseases. *Plast Reconstr Surg* 48:419, 1971.
257. Tessier P: Total osteotomy of the middle third of the face for faciostenosis or for sequelae of the Le Fort III fractures. *Plast Reconstr Surg* 48:533, 1971.
258. Tessier P: Traitement des dysmorphies faciales propres aux dysostoses craniofaciales (DGF), maladies de Crouzon et d'Apert. *Neurochirurgie* 17:295, 1971.
259. Tessier P: Recent improvement in the treatment of facial and cranial deformities in Crouzon disease and Apert syndrome. In *Symposium of Plastic Surgery of the Orbital Region*, St Louis, MO, 1976, CV Mosby Co, p 271.
260. Tessier P: Relationship of craniosynostosis to craniofacial dysostosis and to faciosynostosis: A study with therapeutic implications. *Clin Plast Surg* 9:531, 1982.
261. Tessier P: Apert syndrome: Acrocephalosyndactyly, type I. In Caronni P, editor: *Craniofacial surgery*, Boston, 1985, Little, Brown.
262. Tessier P: *Craniofacial surgery in syndromic craniosynostosis: Craniosynostosis, diagnosis, evaluation and management*, New York, 1986, Raven Press, p 321.
263. Tessier P: The monobloc and frontofacial advancement: Do the pluses outweigh the minuses? [discussion]. *Plast Reconstr Surg* 91:988, 1993.
264. Tessier P, Guiot G, Rougerie J, et al: Osteotomies cranio-naso-orbito-faciales, hypertelorisme. *Chir Plast* 12:103, 1967.
265. Thompson DN, Harkness W, Jones B, et al: Subdural intracranial pressure monitoring in craniosynostosis: Its role in surgical management. *Childs Nerv Syst* 11:269–275, 1995.
266. Tuite GF, Chong WK, Evanson J, et al: The effectiveness of papilledema as an indicator of raised intracranial pressure in children with craniosynostosis. *Neurosurgery* 38:272, 1996.
267. Van der Meulen JC: Medial faciotomy. *Br J Plast Surg* 32:339–342, 1979.
268. Vannier MW, Pilgram TK, Marsh JL, et al: Craniosynostosis: Diagnostic imaging with three-dimensional CT presentation. *AJNR Am J Neuroradiol* 15:1861–1869, 1994.
269. Virchow R: Uber den cretinismus, nametlich in Franken, under uber pathologische: *Schadelformen Verk Phys Med Gessellsch Wurszburg* 2:230–271, 1851.
270. Vogt A: Dyskephalie (dysostosis craniofacialis, maladie de Crouzon 1912) und eine neuartige kombination dieser krankheir mit syndaktylie der 4 extremitaeten (dyskephlodaktylie). *Klin Monatsbl Augenheilk* 90:441, 1933.
271. Vuilliamy DG, Normandale PA: Craniofacial dysostosis in a Dorset family. *Arch Dis Child* 41:275, 1966.
272. Waitzman AA, Posnick JC, Armstrong D, Pron GE: Craniofacial skeletal measurements

based on computed tomography: Part I. Accuracy and reproducibility. *Cleft Palate Craniofac J* 29:112–117, 1992.

273. Waitzman AA, Posnick JC, Armstrong D, Pron GE: Craniofacial skeletal measurements based on computed tomography. Part II. Normal values and growth trends. *Cleft Palate Craniofac J* 29:118–128, 1992.
274. Walia HK, Sodhi JS, Gupta BB, et al: Roentgenologic determination of the cranial capacity in the first four years of life. *Indian J Radiol Imaging* 26:250, 1972.
275. Ward SL, Marcus CL: Obstructive sleep apnea in infants and young children. *J Clin Neurophysiol* 13:198–207, 1996.
276. Warren SM, Shetye PR, Obaid SI, et al: Long-term evaluation of midface position after Le fort III advancement: A 20-plus-year follow-up. *Plast Reconstr Surg* 129: 234–242, 2012.
277. Whitaker LA, Bartlett SP, Schut L, et al: Craniosynostosis: An analysis of the timing, treatment and complications in 164 consecutive patients. *Plast Reconstr Surg* 80:195, 1987.
278. Whitaker LA, Munro IR, Sayler KE, et al: Combined report of problems and complications in 793 craniofacial operations. *Plast Reconstr Surg* 64:198, 1979.
279. Whitaker LA, Schut L, Kerr LP: Early surgery for isolated craniofacial dysostosis. *Plast Reconstr Surg* 60:575, 1977.
280. Witherow H, Dunaway D, Evans R, et al: Functional outcomes in monobloc advancement by distraction using the rigid external distractor device. *Plast Reconstr Surg* 121:1311–1322, 2008.
281. Wolfe SA: Critical analysis of 81 monobloc frontofacial advancements over a 27-year period: Should they all be distracted, or not? Rancho Mirage, Calif, 2009, Abstract Presentation, 88th Annual Meeting, American Association of Plastic Surgeons. pp 200–201.
282. Wolfe SA, Morrison G, Page LK, et al: The monobloc and frontofacial advancement: Do the pluses outweigh the minuses? *Plast Reconstr Surg* 91:977, 1993.
283. Wong GB, Kakulis EG, Mulliken JB: Analysis of fronto-orbital advancement for Apert, Crouzon, Pfeiffer, and Saethre-Chotzen syndromes. *Plast Reconstr Surg* 105:2314, 2000.
284. Xu HS, Mu XZ, Yu ZY, et al: Changes of different section area at different parts of upper-airway after Le Fort III osteotomy [Chinese]. *Zhonghua Zheng Xing Wai Ke Za Zhi* 24:181–183, 2008.
285. Zhou G, Schwartz LT, Gopen Q: Inner ear anomalies and conductive hearing loss in children with Apert syndrome: An overlooked otologic aspect. *Otol Neurotol* 30:184–189, 2009.

31

Selected Complex Malformations that Frequently Require Maxillofacial Reconstruction: Evaluation & Treatment

JEFFREY C. POSNICK, DMD, MD

- Klippel–Feil Anomaly
- Down Syndrome (Trisomy 21 Syndrome)
- Neurofibromatosis
- Hemihyperplasia of the Face
- Vascular Malformations
- Velocardiofacial Syndrome
- Conclusions

More than 400 syndromes and anomalies produce a spectrum of head and neck dysfunctions and facial disproportions within the maxillofacial region. Each syndrome type and its presenting set of anomalies will be unique, with individual variations. Developing an appreciation for the complexity of how these patients present is essential to providing thoughtful counsel regarding the timing and techniques of maxillofacial reconstruction and orthodontic care. In this chapter, only a few of the more frequently encountered syndromes are reviewed; suggestions are given for their reconstruction.

Klippel–Feil Anomaly

Background

The Klippel–Feil anomaly (KFA) is characterized by the congenital fusion of two or more cervical vertebrae. In its most severe form, there is massive cervical vertebral fusion with a short neck, limited head movement, and a low posterior hairline.[2,12,15,21,23,29,37,39,55] Gorlin and Cohen have pointed out that the term *vertebral fusion* is not accurate because this condition results from the failure of the normal segmentation process.[24] Vertebral fusion can be traced to the third embryonic week when the segmentation of the mesodermal somites normally takes place.

KFA was described as early as the 16th century, and similar anatomic findings in the second and third cervical vertebrae have been found in an Egyptian mummy that has been dated to be from about 500 B.C. In 1912, Maurice Klippel and Andre Feil described the postmortem findings of a 46-year-old French tailor who had a short, immobile neck with massive fusion of cervical and upper thoracic vertebrae.[34] In 1919, Feil added reports of 13 additional patients, 12 of whose cases had been reported previously.

In 1967, Gunderson and colleagues categorized KFA in accordance with three morphologic types of cervical vertebral fusion.[27] Type I consists of the massive fusion of many cervical and upper thoracic vertebrae into bony locks. Type II involves fusion at only one or two interspaces. Type III comprises both cervical fusion and lower thoracic or lumbar fusion.

It is important to remember that KFA is morphologically and etiologically heterogeneous. This was further documented by Gunderson, who used Feil's classification system to review inheritance patterns.[28] Type I cases have almost all been sporadic, with a female predilection. Type II is transmitted as an autosomal dominant disorder. For Type III, an autosomal recessive mode of inheritance has been suggested.

Various authors differ with regard to their interpretation of what constitutes KFA.[9,61-63] A malformation with the triad of a short neck, a low posterior hairline, and a painless restriction of cervical motion characterizes just over 50% of patients. The functional limitation of the neck range of motion is the most consistent finding. Despite frequent

sparing of the atlanto–axial joint, rotation is typically impaired more than flexion or extension is, with the latter exhibiting a range of more than 90 degrees, even with only one open disc space.

With the severe form of KFA (Type I deformity), there is massive cervical vertebral fusion, and the head seems to sit directly on the thorax, without any interposition of the neck. The flared trapezius muscles extend from the mastoid areas to the shoulders, which produces a pterygium-like effect. Posteriorly, the hairline extends to the shoulders. Cervical foreshortening (rather than overgrowth of the scalp) displaces the hairline and is accompanied by webbing of the neck.

Diverse ocular anomalies are frequently found, with the impairment of extraocular movement being the most frequent.[57]Convergent strabismus and, less commonly, horizontal nystagmuses are seen. From 25% to as many as 50% of affected children exhibit hearing loss that may be sensorineural, mixed, or conductive.[11,16,39,51,60,64] Clefting of the secondary palate is present in 15% to 20% of patients.[10,58,59] The combination of hearing loss and cleft palate explains the hypernasality that has been documented in approximately 15% to 20% of patients with KFA. Congenital heart defects occur in 4.2% of patients, which is in stark contrast with the prevalence of 0.6% among all live births in the general population.[50] Ventricular septal defect is the most common heart anomaly documented,[43] and vascular anomalies may also occur.[5,6] Congenital urinary anomalies are a frequent finding, with unilateral renal agenesis occurring in 28% of patients.[14] Genital anomalies (e.g., vaginal agenesis) also occur at a higher frequency in these patients than they do in the general population.[40,42] Neurologic disturbances that consist of involuntary dyskinesis, spasticity or hyperreflexia, syringomyelia, syringobulbia, disc protrusion, osteophytes, and narrowing at the level of the craniovertebral junction have been reported..[3,20,22,36,44,48] Nagib and colleagues found patients with KFA with two unsegmented blocks of bone, cervical stenosis, or cranial involvement to be at highest risk for neurologic disturbances.[45-47] Those with only one unsegmented bone were found to be at low risk for cervical injuries. The flared trapezius muscles associated with KFA give the individual an appearance that may be similar to that seen in patients with Turner or Noonan syndrome.[8,13,18,38] The cervical vertebral anomalies and other defects of the spine that occur within the oculo–auriculo–vertebral spectrum may also be confused with those of KFA.[4,7,8,25,32,33,35,49,52,54,56]

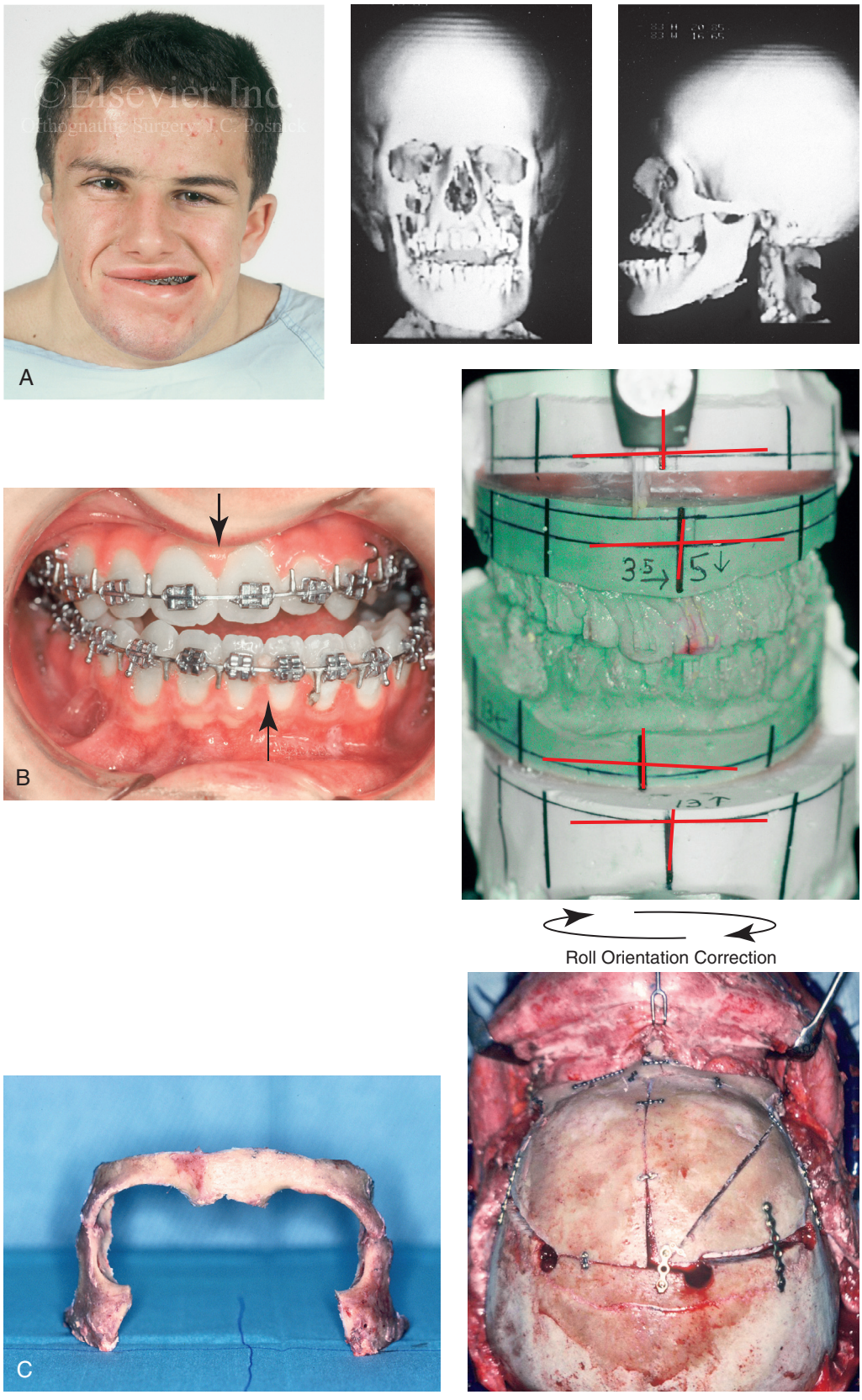
Author's Approach to Facial Reconstruction in Patients with Klippel–Feil Anomaly

The surgical management of KFA requires the accurate diagnosis of each specific problem (see Fig. 1-2).[17,26,31] Middle-ear infections should be treated promptly, and any hearing deficit should be aided whenever possible. Clefting of the secondary palate should be repaired in the usual way. Hypernasality as a result of inadequate soft palate motion after initial palate repair should be recognized and treated with a pharyngoplasty. Extraocular muscle abnormalities are managed with eye patching, spectacles, and eye-muscle surgery, as appropriate. The recognition of cardiovascular and genitourinary malformations should lead to medical treatment and surgery. Cervical spine instability requires the decompression of any nerve impingement and the fusion of unstable segments. Unfortunately, the typical fixed neck curvature observed in patients with KFA cannot safely be straightened.

The aim of the reconstructive procedures used to correct a webbed neck is to create as normal a neck contour as possible with a symmetrical posterior hairline while avoiding obvious scarring.[1] It is important to recognize that not all patients with webbed necks present with the same deformity. The clinician must assess the quality and quantity of available neck skin as well as the height and straightness of the cervical spine. Type I KFA consists of an extremely short skeletal neck that results in the increased width of the neck soft tissues. A spectrum of neck soft-tissue rearrangement procedures has been suggested to improve the webbed-neck deformity. Menick and colleagues modified the "butterfly" excision of redundant skin to avoid a midline scar.[41] They did this by extending the more traditional inferior flaps into the scalp. Unfortunately, with this design, the scars are displaced laterally, and they become visible at the hairline, close to the original site of the web. Some surgeons feel that a midline posterior scar is often preferred aesthetically to one that can be seen along the lateral neck region. The placement of tissue expanders followed by flap rearrangements has also been carried out with some success. Thus, to review, procedures designed to correct the webbed neck suffer from an inability to elevate the hairline, noticeable scarring, and the intrinsically inadequate height of the skeletal structures of the neck.

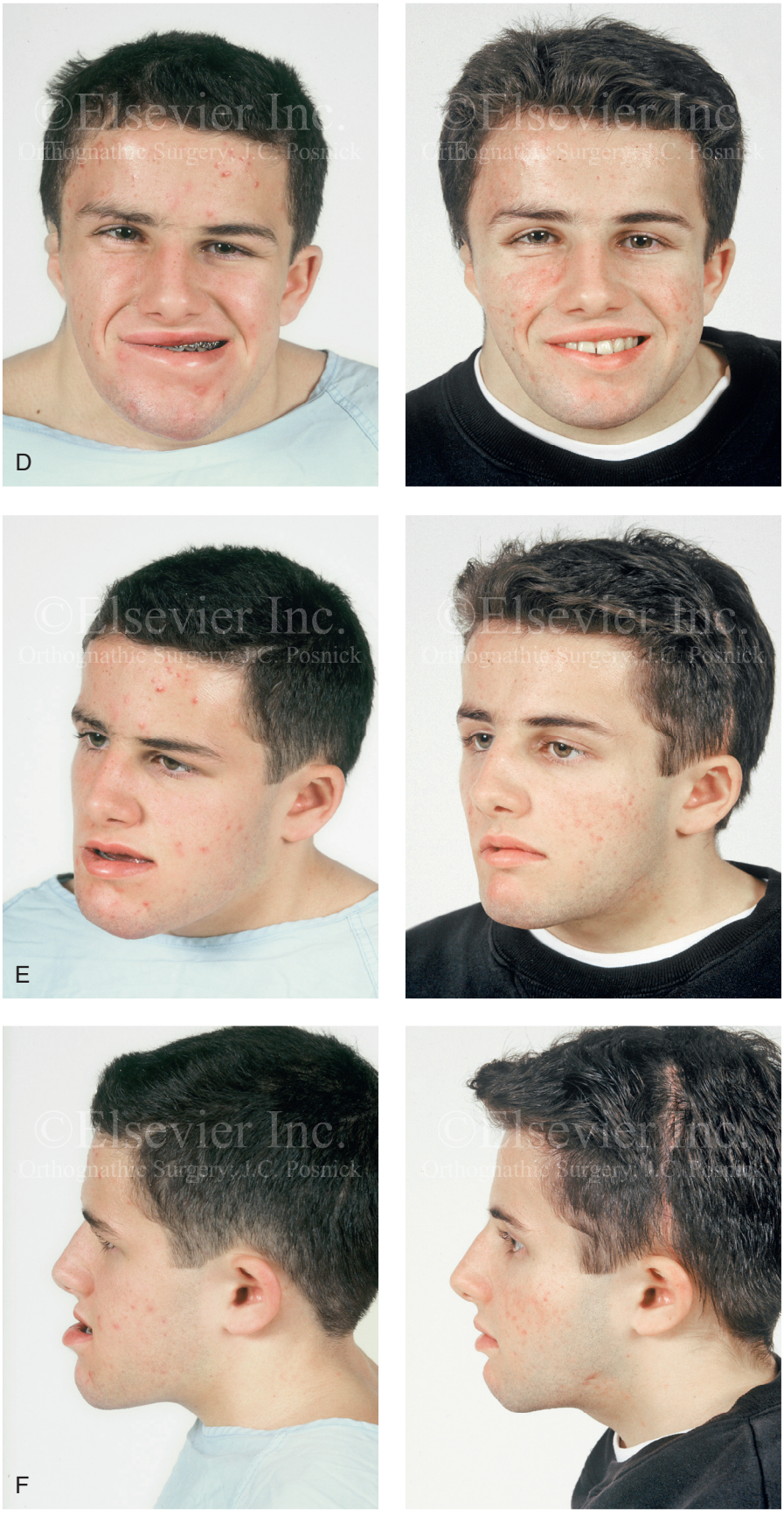
The craniofacial skeletal deformities of patients with KFA typically present in one of two patterns.[19,30,53] The first is in association with a congenital asymmetrical cervical spine fusion. In these patients, a variable degree of nonsynostotic anterior plagiocephaly is present. The ipsilateral fronto–orbito–zygomatic flattening and retrusion may be significant enough that anterior cranial vault, orbital, and zygomatic reconstruction carried out through an intracranial approach will be indicated to adequately improve morphology (Fig. 31-1). These individuals will also have an asymmetrical dentofacial deformity that requires Le Fort I (maxillary) osteotomy, bilateral sagittal split ramus osteotomies of the mandible, and an osseous genioplasty procedure for three-dimensional repositioning (Fig. 31-2). The most unsettling aspect of the craniofacial reconstruction relates to the fact that the neck cannot be straightened. It will remain crooked, and so decisions about the best aesthetic positioning of the osteotomized craniofacial skeletal units must also be adjusted.

The second pattern of presentation is in association with a congenital symmetric cervical spine fusion. The cranio–orbito–zygomatic region is symmetrical and has



• **Figure 31-1** A 17-year-old boy with Klippel–Feil anomaly has a fixed head tilt as a result of a painless cervical spine fusion and a partial facial nerve palsy on the right side. There is marked asymmetry and deformity of the craniofacial region that involves the upper facial skeleton (i.e., the cranial vault, the orbits, and the zygomas) and the lower facial skeleton (i.e., the maxilla, the mandible, and the chin). The reconstruction was compromised by the fixed head tilt and the CN VII deficit. The patient underwent cranio–orbito–zygomatic (intracranial) reconstruction through a coronal scalp incision followed by orthognathic surgery that included a Le Fort I osteotomy, bilateral sagittal split ramus osteotomies, and an osseous genioplasty. **A,** Frontal facial and computed tomography scan views before surgery. **B,** Occlusal view before surgery and articulated dental cast that indicates analytic model planning. **C,** Intraoperative view of removed orbital and zygomatic units and intraoperative bird's-eye view of the anterior cranial vault after cranio–orbito–zygomatic reconstruction.

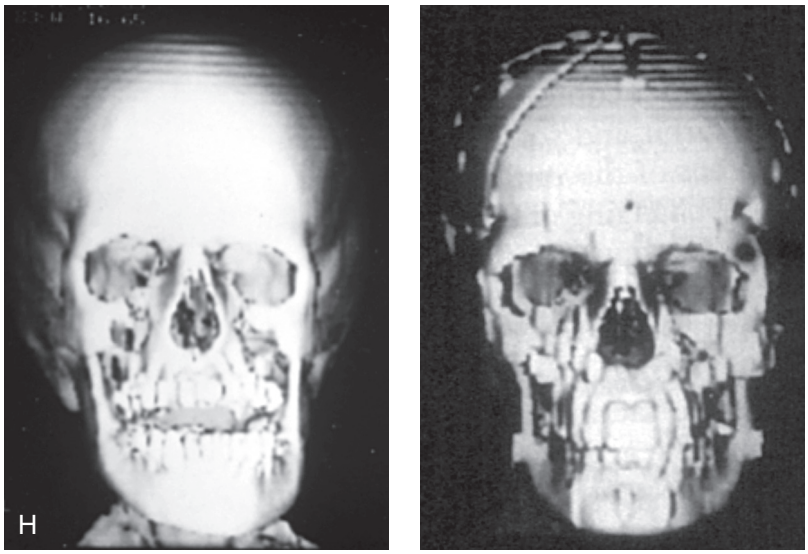
Continued



• **Figure 31-1, cont'd** **D,** Frontal views with smile before and after reconstruction. Note that the fixed head tilt limited the the correction of facial symmetry. **E,** Oblique views before and after reconstruction. **F,** Profile views before and after reconstruction.

Orthodontics in progress with maxillary bicuspid extractions

After treatment



• **Figure 31-1, cont'd G,** Occlusal views with orthodontics in progress (including upper bicuspid extractions) and then after reconstruction. **H,** Three-dimensional computed tomography scan views before and after reconstruction.

relatively good proportions.[53] No reconstruction procedures are generally required in the upper facial skeleton. With reference to the maxillomandibular region, the constant open mouth posture of the mandible generally results in vertical maxillary excess and a constricted maxillary arch width. The mandible is usually retrognathic, with a vertically long retrusive chin and an open-bite malocclusion. A degree of maxillomandibular asymmetry is expected. The neck–chin angle is markedly obtuse. These individuals will benefit from orthognathic surgery in combination with orthodontic treatment. The orthognathic surgery will generally require a Le Fort I osteotomy in segments to intrude the maxilla vertically, advance it horizontally, and widen it transversely. The preferred approach also involves bilateral sagittal split ramus osteotomies of the mandible with horizontal advancement and counterclockwise rotation to match the new maxillary position in combination with a vertical reduction and advancement genioplasty (see Chapter 15).

Down Syndrome (Trisomy 21 Syndrome)

Background

Speculation about the historic prevalence of Down syndrome has included references to depictions of the syndrome in 15th- and 16th-century paintings.[69,81]

• **Figure 31-2** A teenage girl with the Klippel–Feil and Poland congenital anomalies has a fixed head tilt as a result of a painless cervical spine fusion. At birth, she was noted to have a significant diaphragmatic herniation of the stomach and spleen into the thoracic cavity. She underwent procedures for the correction of the diaphragmatic hernia and then required esophageal dilatations. She also required decompression and fusion for the cervical anomalies to limit injury to the upper spinal cord. She was referred to this surgeon as a teenager and underwent evaluations that included restorative dentistry, orthodontics, speech pathology, otolaryngology, pediatric surgery, pediatric orthopedic spine surgery, and pediatrics professionals. The assessment of pulmonary capacity, swallowing and dysphagia, and safe range of motion of the neck were of particular importance. There was marked asymmetry and deformity of the maxillomandibular region. The plans for reconstruction were affected by the fixed head tilt. The patient underwent a combined orthodontic and surgical approach, and her procedures included: maxillary Le Fort I osteotomy (horizontal advancement and cant correction); sagittal split ramus osteotomies (horizontal advancement and asymmetry improvement); osseous genioplasty (vertical shortening and horizontal advancement); and septoplasty and inferior turbinate reduction. Intraoperative cervical monitoring was carried out to limit the chance of spinal cord injury. **A,** Frontal views in repose before and after reconstruction. **B,** Frontal views with smile before and after reconstruction.

©Elsevier Inc.
Orthognathic Surgery; J.C. Posnick

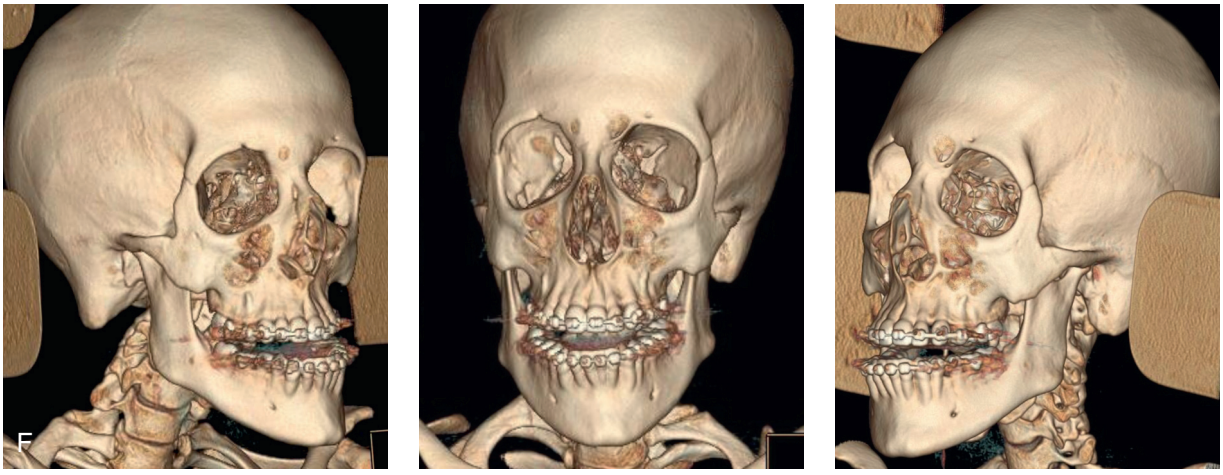
• **Figure 31-2, cont'd** **C,** Oblique facial views before and after reconstruction. **D,** Profile views before and after reconstruction.

Continued

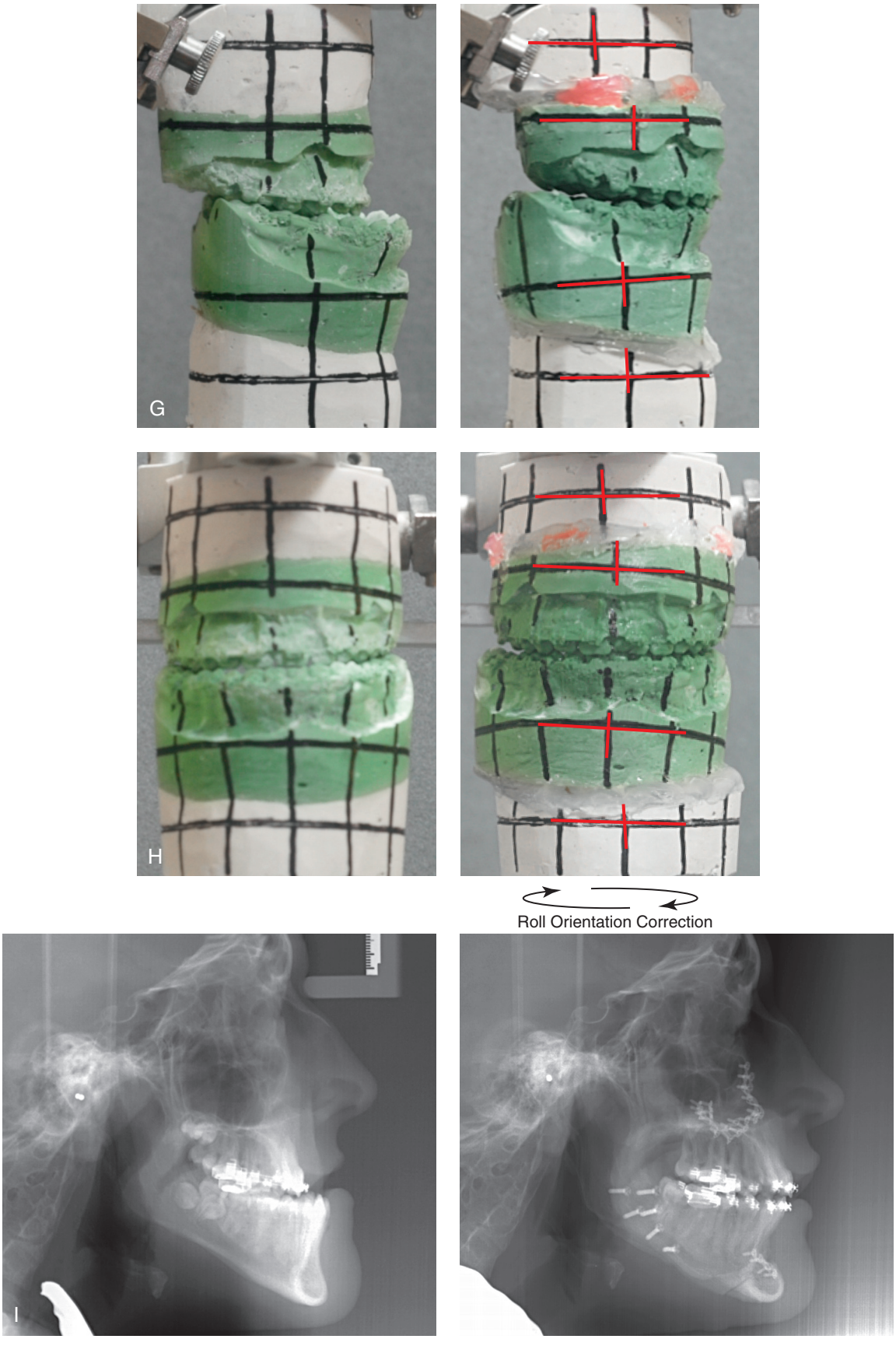
Early orthodontics in progress

Pre surgery with upper bicuspid extractions

After treatment



• **Figure 31-2, cont'd E,** Occlusal views before treatment, with orthodontics in progress and with upper bicuspid extractions, and then after reconstruction. **F,** Computed tomography scan views before reconstruction.



• **Figure 31-2, cont'd** **G** and **H,** Articulated dental models that indicate analytic model planning. **I,** Cephalometric radiographs before and after reconstruction.

Martínez-Frías described what seems to be the earliest observation of the syndrome in a sculpted terracotta head from 580 A.D.[89] The terracotta piece comes from the Toltec culture of Mexico, and its facial features, which include macroglossia, help to define its subject as a person with Down syndrome. The features of this Down syndrome were initially described by Langdon Down in 1866, and the condition represents a combination of phenotypic features that includes a degree of mental deficiency and characteristic facial features.[72]

The birth prevalence of trisomy 21 syndrome is generally stated to be 1 in 650 live births, but it is known to vary in different populations from 1 in 600 to 1 in 2000 live births. During the 1970s, 15% of patients who had been institutionalized for mental deficiency were found to have trisomy 21 syndrome.[75]

About 95% of all cases of Down syndrome arise from non-disjunction. Approximately 80% are of maternal origin, with 20% being of paternal origin. The risk increases with increasing maternal age as well as with paternal age and particularly when the parents are more than 35 years old. It has been estimated that approximately 4.8% of Down syndrome cases arise from an unbalanced translocation, which may occur de novo or by parental transmission. Inherited D/G translocations occur in 93% of maternal cases and 7% of paternal cases; however, for G/G translocations, 50% are of maternal origin, and 50% are of paternal origin. It has been estimated that 65% to 80% of trisomy 21 conceptuses result in spontaneous abortions.[75]

Detectable mosaicism (found in about 3% of trisomy 21 cases) and the Down syndrome phenotype may only be partially expressed.[75] The use of the DNA samples from individuals who have partial trisomy 21 with or without features of the phenotype has been helpful for understanding the nature of the disorder. The area between loci D21S58 and D21S42 has been identified as being associated with mental retardation and with most of the facial features of Down syndrome. The facial features that have been identified in this region include oblique palpebral fissures; epicanthal folds; a flat nasal bridge; a protruding tongue; short, broad hands with clinodactyly of the fifth finger; a gap between first and second toes; hypotonia; short stature; Brushfield spots; and characteristic dermatoglyphic findings, including a single palmar crease and an increased number of ulnar loops.[88]

Individuals with Down syndrome have a specific set of major congenital malformations, including heart anomalies (30% to 40%) and gastrointestinal tract anomalies such as duodenal stenosis or atresia, imperforate anus, and Hirschsprung disease.[75] The development of Alzheimer disease occurs at a much earlier age in individuals with trisomy 21 than it does in the general population. The frequency of symptomatic gallbladder disease is 25% higher among adults with Down syndrome as compared with the general population.

Down syndrome is characterized by extensive phenotypic variability.[65,67,71,98] Although cognitive impairment, muscle hypotonia at birth, and dysmorphic features occur to some extent in all individuals, most associated traits occur in only a fraction of affected individuals. In the patient with Down syndrome, a number of characteristic facial features that vary from patient to patient are frequently recognized:

1. Epicanthal folds
2. Oblique (anti-mongoloid) slanting of the eyelids
3. Strabismus as a result of extraocular muscle dysfunction
4. Flatness of the dorsum of the nose
5. Hypoplasia of the upper jaw that results in an Angle Class III malocclusion
6. Macroglossia
7. Microgenia

In 1967, Otermin Aguirre presented a series of patients with Down syndrome who underwent reconstructive surgery in an attempt to normalize their facial morphology.[93] During the same year, Hohler attempted to normalize the face of a young girl with Down syndrome by augmenting the hypoplastic dorsum of the nose and the receding chin with alloplastic implants.[77] In 1977, Lemperle and Spitalny systematically tried to normalize the faces of children with Down syndrome.[82-85] Similar results were published by Olbrisch,[91,92] Patterson and colleagues,[94] Wexler and Peled,[103,104] and Rozner,[99] in addition to several others.[76,90,94,97]

Despite the initial enthusiasm for facial reconstruction in children with Down syndrome during the late 1960s through the mid 1980s, a critical review of the procedures used to normalize the face was not carried out until the mid to late 1980s. In 1989, Katz and Kravetz presented a balanced, thoughtful review and a discussion of the literature regarding the "effectiveness of facial surgery for persons with Down syndrome."[79] The research of Katz and Kravetz may be summarized as follows: If improved functioning, appearance, and social acceptability are measured by parent and treating physician satisfaction, then the outcome of facial plastic surgery for people with Down syndrome seems positive. If function, appearance, and social acceptability are evaluated by people who were not involved in making the decision to conduct the facial plastic surgery—and particularly when control subjects are included in the evaluation—then the outcome of facial plastic surgery for people with Down syndrome is not so positive. For most patients with Down syndrome, the surgeons' and parents' well-intentioned attempts to normalize the child's facial morphology could not be realized.[96]

Macroglossia with protrusion of the large tongue is a frequent feature of Down syndrome. Dystonia or hypotonia of the tongue is also a factor.[66,74] During the 1970s and the early 1980s, several investigators suggested that partial glossectomy in the patient with Down syndrome could improve the clarity of speech.[68,70] However, studies that used objective criteria to document speech results after partial

glossectomy in patients with Down syndrome showed no significant differences in the number of articulation errors between audiotape recordings made preoperatively and 6 months postoperatively in a series of patients or between the recordings of the same patients and recordings of an additional series of Down syndrome control patients who did not undergo surgery. A further study by Margar-Bacal and colleagues looked at changes in the aesthetic appearance and intelligibility of speech after partial glossectomy in patients with Down syndrome.[87] The aesthetic appearance of speech (i.e., the visual acceptability of the patient while he or she is speaking) was judged from visual information only. Judgments of speech intelligibility were made separately from the auditory portion of the videotapes. The acceptability and intelligibility of the children were also judged together during audiovisual presentation. Statistical analysis showed that speech was significantly more acceptable aesthetically after surgery. No significant difference was found in speech intelligibility postoperatively as compared with preoperatively.

An Angle Class III malocclusion as a result of the midface hypoplasia with overclosure of the mandible is a frequent finding in patients with Down syndrome.[95] This was objectively studied by Farkas with the use of anthropometric measurements in which he documented that 60% of children with Down syndrome showed a disproportionately small depth to the middle third of the face (i.e., the anthropometric measurement from the subnasal point to the tragus).[73] The congenital absence of permanent teeth, poor root formation, and hypoplasia of the enamel are frequent complicating factors.

Author's Approach to Facial Reconstruction in Individual's with Down Syndrome

By the time that a individual with Down syndrome is 5 years old (with a formal speech and language assessment if the tongue is confirmed to be excessive in volume and if he or she is unable to comfortably maintain the tongue in the oral cavity), a conservative partial glossectomy may be useful to limit secondary skeletal deformities, improve breathing, and improve the aesthetic appearance of the child while he or she is eating or speaking and when his or her lower jaw is at rest. The basic procedure is a closing anterior wedge resection of the tongue that is carried out with the patient under general anesthesia. A variety of other techniques have also been described to conservatively reduce the size of the tongue.

When a child with Down syndrome approaches skeletal maturity (i.e., 14 to 16 years of age for girls, 16 to 18 years of age for boys) and a significant jaw deformity with malocclusion is present, then a comprehensive approach of orthodontic treatment and orthognathic surgery can effectively alter the facial proportions (aesthetics) and improve head and neck functions (i.e., lip posture, chewing, speech articulation, and breathing) (Fig. 31-3). A formal sleep study is indicated before surgery to clarify the extent of upper airway obstruction and the degree of maxillomandibular advancement required to manage it.[1,78,80,86,100-102] The orthodontic treatment may be compromised as a result of poor dental root formation, congenitally missing teeth, or limited compliance. The coordination of care with an appropriate dental team (e.g., a restorative dentist, a periodontist, an orthodontist) is useful before the initiation of any treatment.

The correction of the dentofacial deformity and the management of the airway frequently requires maxillary Le Fort I osteotomy (horizontal advancement and vertical lengthening), often with sagittal split ramus osteotomies for alignment to the new maxillary position (i.e., advancement but not setback) and an osseous genioplasty (horizontal advancement and vertical lengthening). Septoplasty and inferior turbinate reduction should be simultaneously performed if required to improve nasal airflow (see Chapter 10).

Unfortunately, the numerous adnexal (eyelid) soft-tissue procedures described in the literature (e.g., lateral canthopexy) in an attempt to normalize the faces of patients with Down syndrome have mostly proven to be suboptimal.

Neurofibromatosis

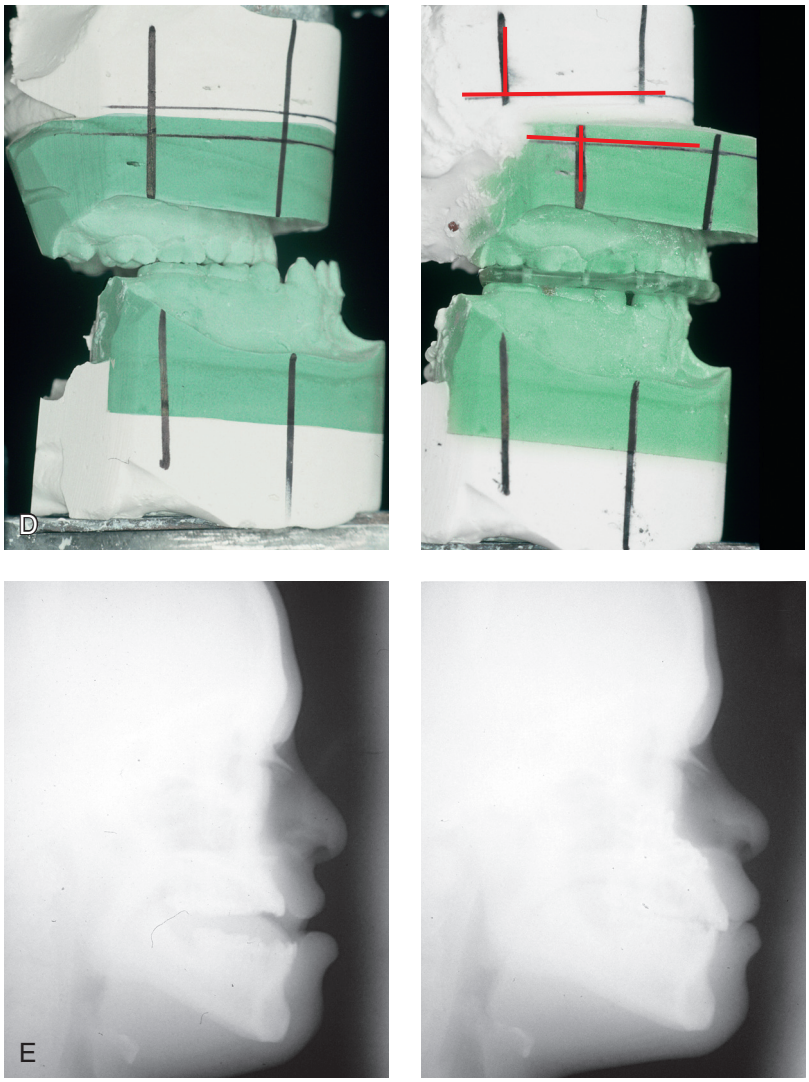
Background

In 1849, Robert Smith, a professor of surgery at Dublin Medical School, reported clinical and necropsy findings in two cases of individuals who were presumed to have neurofibromatosis; he cited 75 references to similar patients from the earlier medical literature.[130] Unfortunately, he did not recognize that the tumors (neurofibromas) contained neural elements. In 1882, von Recklinghausen published his findings, which convinced the medical world that neurofibromatosis was a distinct entity of neural origin.[165] Today, neurofibromatosis is known to be etiologically heterogeneous. Cohen and Hayden were the first to differentiate Proteus syndrome from neurofibromatosis in 1979.[119,162] Types I and II are the forms that are frequently encountered by the craniomaxillofacial surgeon.

Neurofibromatosis Type II (Acoustic Type)

The hallmark of Type II neurofibromatosis is the presence of bilateral acoustic neuromas.[147,150] Symptoms are usually caused by pressure on the vestibulocochlear and facial nerve complex. The first symptom is usually hearing loss that often begins during the teenage years or the early 20s. Occasionally, hearing loss may occur as early as the first decade or as late as the seventh decade of life. Cafe-au-lait spots and cutaneous neurofibromas are also present, but they are seen less frequently than they are among patients with neurofibromatosis Type I.[121] Tumors of the central nervous system are especially common, with Schwann cell tumors occurring most frequently. Multiple tumors of meningeal or glial origin may also occur.[147] Neurofibromatosis Type II has autosomal dominant inheritance with penetrance of more than 95%. The responsible gene has been mapped to chromosome 22.

• **Figure 31-3** A 16-year-old boy with Down syndrome presented with a jaw deformity and malocclusion. He underwent a combined orthodontic and surgical approach. The surgery was limited to a Le Fort I osteotomy (horizontal advancement and vertical lengthening). **A,** Frontal views before and after reconstruction. **B,** Profile views before and after reconstruction. **C,** Occlusal views before and after reconstruction. Note that the orthodontic and restorative dental objectives were limited by congenital malformations of the dental crowns and dental roots.



• **Figure 31-3, cont'd D,** Articulated dental casts that indicate analytic model planning. **E,** Lateral cephalometric radiographs before and after reconstruction.

Neurofibromatosis Type I (von Recklinghausen Type)

Joseph Merrick, the Elephant Man, has often been thought to have had von Recklinghausen disease. However, after considering several diagnostic possibilities, Cohen concluded that Merrick's skeletal findings are most consistent with Proteus syndrome.[119,130,162]

This classic form of neurofibromatosis accounts for approximately 90% of all affected cases. Major features include six or more cafe-au-lait spots, cutaneous neurofibromas, and Lisch nodules.[130] Axillary freckling develops in about 66% of all patients. Neurofibromatosis type I, which is the classic form of neurofibromatosis, is the one described by von Recklinghausen; it is also the most frequent form, occurring in 1 of every 2500 to 3000 births.[146,160,165,166] Inheritance is autosomal dominant, with almost 100% penetrance. Approximately 50% of cases represent new mutations, and an increase in paternal age at the time of conception has been found to be an associated feature. Although most evidence points to neurofibromatosis as a disorder of neural crest derivation, some controversy remains about whether the neural and mesenchymal components are interrelated or if they may arise independent of each other.[110,116,153]

Type I is caused by a mutation of the neurofibromin 1 (NF1) gene. Approximately 5% to 20% of all patients with Type I neurofibromatosis patients carry a heterozygous deletion of approximately 1.5 Mb that involves the NF1 gene and the contiguous change line in its flanking region.[157] Miller and Hall found that patients born to affected mothers have more severe disease than those born to affected fathers.[152] The mutation rate in the NF1 gene is one of the highest known in humans, with approximately 50% of all patients with NF1 presenting with novel mutations.[124]

The natural history of neurofibromatosis Type I has been reviewed by several authors.[138] More than 40% of patients have some clinical manifestations at birth, and more than 60% have manifestations by the second year of life. Cafe-au-lait spots usually develop first, with multiple lesions

present within the first year of life. In approximately 50% of patients, axillary freckling appears later. Cutaneous neurofibromas appear around the onset of puberty and increase in number throughout life.[138] Lisch nodules begin to appear during early childhood and have been observed in almost all affected adults. Plexiform neurofibromas occur in 30% of these patients.

Approximately 33% of all patients develop one or more complications in association with the disease.[105-108,115] It has been estimated that some form of malignancy develops in 6% of patients with neurofibromatosis type I who are more than 18 years old.[109,117,118] Other important complications that may occur are neurologic and include epilepsy, aqueduct stenosis, and spinal neurofibromas. Learning disabilities of various kinds affect 25% of patients.[137,154,163]

Any part of the eye may be involved.[111,126,128,132] Lisch nodules of various sizes may be found anywhere in the iris. These lesions are melanocytic hamartomas, and they are found only in patients with neurofibromatosis. Phakoma, congenital glaucoma, corneal opacity, detached retina, optic atrophy, and congenital ptosis of the eyelids have all been reported. Intraorbital lesions may also produce proptosis and eye-muscle palsies.

The literature concerning the presenting head and neck dysmorphology and the suggested treatment of orbitotemporal neurofibromatosis is extensive.* The plexiform neurofibroma that involves the orbit, the eyelid, and the temporal region may also have an intracranial component. It generally appears during childhood as a swelling of the upper eyelid, and it slowly and progressively worsens (Fig. 31-4). Further involvement of the subcutaneous tissues of the eyelids with varying degrees of ptosis and proptosis of the globe also occurs. For patients in whom progression continues, there is further plexiform neurofibromatosis of the temporal area, pulsating proptosis, continued enlargement of the eyelids with extensive mechanical ptosis, and the inability to open the eye, which results in severely diminished visual acuity or blindness. The globe itself is involved with neurofibroma, and buphthalmos may be present. The eye may be painful as a result of infection and epiphora.

The primary skeletal abnormalities associated with type I neurofibromatosis include long bone dysplasia, sphenoid wing dysplasia, and scoliosis. The unilateral nature of the skeletal involvement suggests a random molecular event. The classic upper face skeletal malformations and deformities include an absence of the greater wing of the sphenoid that may be either partial or complete. As part of the sphenoid wing malformation, there may be a defect of the posterolateral orbital wall and enlargement of the superior orbital fissure with prolapse of the temporal lobe of the brain into the orbit and the temporal fossa region. This further pushes the globe anteriorly and inferiorly, and it may cause pulsatile exophthalmos. The intraorbital volume may be greatly enlarged, and the orbit may be abnormally shaped. The lateral and infraorbital rims are displaced outwardly and inferiorly in addition to being thinned. The orbital floor is depressed, and the orbital roof and the supraorbital rim are elevated. The zygoma is often dysplastic and inferiorly displaced.

*References 112, 113, 120, 122, 123, 125, 129, 131, 133, 134, 139-145, 148, 149, 151, 156, 164, 167

Author's Approach to Reconstruction in Patients with Orbitotemporal Neurofibromatosis

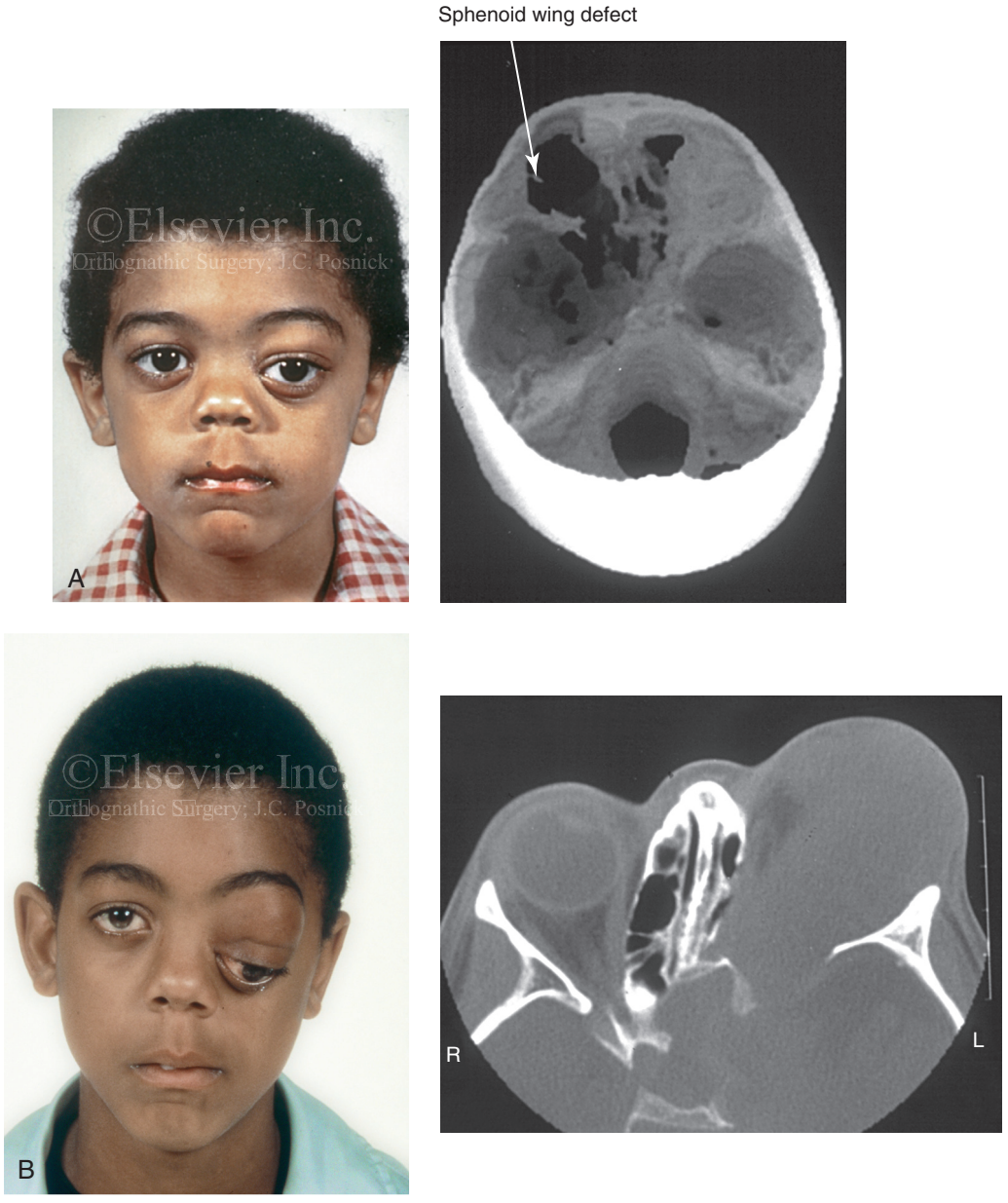
It is recognized that the facial reconstruction of patients with this condition is often difficult as a result of the multilevel involvement of the skeleton, the soft tissues, and the viscera (i.e., brain, eyes, tongue, soft palate, sinus, and teeth). A basic premise of treatment is to preserve the seeing eye whenever feasible.[114,127,135,136,155,158,159,161] The prevention of brain injury and the improvement of the tongue, lips, soft palate, and jaw function are high priorities. Jackson and colleagues have suggested a classification system for orbitotemporal neurofibromatosis that they have found useful for treatment planning[139]:

1. Orbital soft-tissue involvement with a seeing eye
2. Orbital soft-tissue and significant bony involvement with a seeing eye
3. Orbital soft-tissue and significant bony involvement with a blind or absent eye

It has been pointed out by Jackson and others that conservative surgical debulking procedures that leave considerable amounts of the plexiform neurofibroma leave patients prone to continued growth and the need for further surgery.[141] Whenever feasible, more complete resection of the tumor with immediate reconstruction is preferred.

When the temporal–cheek region is extensively involved, the facial nerve presents an extra challenge. If possible, the facial nerve is dissected free, the tumor is debulked, and the soft-tissue flaps are redraped (see Fig. 31-4). When the facial nerve is extensively infiltrated by a tumor that grotesquely deforms the face, it may be necessary or preferable to resect the nerve as part of the debulking procedure. Occasionally we have used an approach that involves the radical debulking of the tumor, the planned sacrifice of the involved facial nerve, and the simultaneous reconstruction of the cheek–facial nerve defect with a reinnervated free muscle transfer.

Despite the less than ideal restoration of function and enhancement of facial aesthetics that is often achieved with the presenting orbitotemporal, temporal–cheek, and maxillomandibular neurofibromatosis deformities, patients and families are generally pleased with the efforts (see Fig. 31-4). Longitudinal computed tomography scanning and magnetic resonance imaging reassessment are useful to follow the disease process, because residual neurofibromatosis generally remains, and malignant transformation may occur.



• **Figure 31-4** A child with neurofibromatosis involving the left cranio–orbital region. Aplasia of the sphenoid wing and the gradual growth of plexiform neurofibroma occurred, which extended from the cavernous sinus through the widened superior orbital fissure, through the cone of the eye, and into the upper eyelid. Despite an intracranial debulking procedure carried out by another surgeon during the patient's middle childhood years, the boy presented to this surgeon with residual and recurrent plexiform neurofibroma that resulted in the distortion and proptosis of the left eye. Vision remained in the affected eye, and the decision was made to preserve it. The patient underwent intracranial reconstruction of the cranio–orbito–zygomatic skeleton, including the debulking of the soft-tissue tumor and the vertical and horizontal debulking of the upper eyelid. Intracranial debulking with eye presentation was accomplished. **A,** Frontal view during early childhood and a computed tomography scan of the anterior skull base indicating the defect in the sphenoid wing. **B,** Frontal view at 10 years of age. Despite a debulking procedure carried out by another surgeon, residual growth has reoccurred. An axial computed tomography scan view through the midorbits indicates the extent of plexiform neurofibromatosis from the cavernous sinus through the upper eyelid.

Continued

• **Figure 31-4, cont'd C,** Frontal views before and after reconstruction. Note that the surgical correction of the upper eyelid ptosis was simultaneously carried out but it was limited to maintain adequate corneal protection. **D,** Profile views before and after reconstruction.

Hemihyperplasia of the Face

Background

Hemihyperplasia was first described by Meckel in 1822.[192] Many comprehensive reviews have been published since that time.[173,174,181,182] In patients with hemihyperplasia, the enlarged area may run the gamut from a single digit to a single limb to unilateral facial enlargement to involvement of half of the body.[168-170,179,183,187,189,198,199,203,206-208] In some patients, the defect is limited to a single tissue type (e.g., muscular, vascular, skeletal, nervous system).[204]

The etiology and pathogenesis of hemihyperplasia are poorly understood, with a variable range of clinical abnormalities having been described.[171,175,178] This fact—in combination with the large number of reported sporadic cases—is suggestive of etiologic heterogeneity. When the face is involved, asymmetry is usually evident at birth, and it may become accentuated or at least more of a concern with age, especially at puberty.[180,188,190,192,196,200-202] The involved bones have been found to be unilaterally enlarged, often with accelerated bone age on the affected side. A variety of non-neoplastic abnormalities of the limbs, teeth, skin, central nervous system, cardiovascular system, liver, kidneys, and genitalia have also been observed.

Various neoplasms have been reported in association with hemihyperplasia, including Wilms tumor, adrenal cortical carcinoma, and hepatoblastoma to name but a few.[172,176,177,184-186,191] In the study by Hoyme and colleagues of 104 cases of isolated hemihyperplasia, tumors developed

in 3.8% of patients.[186] The neoplasms associated with hemihyperplasia have been found to have an embryonal origin. Patients with hemihyperplasia should be screened with ultrasound for tumors every 6 months for the first 4 years of life and probably less frequently thereafter until the age of 7 years.[176,184]

Any and all ipsilateral facial structures may be involved, including the bones of the cranium, the orbit, the zygoma, the maxilla, and the mandible.[193-195] Premature development and eruption of the teeth occur, as does macrodontia of varying degrees. Unilateral enlargement of the tongue and its papillae is also a frequently observed feature. The overall facial soft tissues are increased in thickness, size, and volume. The eyeball and even the brain may be enlarged on the affected side.

Hemihyperplasia of the face should be distinguished from hemimandibular hyperplasia (HMH), which is a three-dimensional enlargement of the whole hemimandible but not of the other facial structures.[193-195] Furthermore, in patients with HMH, no other aspects of the body are enlarged, and no predisposition to tumors has been identified. HMH was first reported by Adams in 1836.[182] Epidemiologic data suggest that there is equal incidence of HMH in both sexes and in all ethnic groups. Obwegeser drew the distinction between HMH and hemimandibular elongation.[193-195] He also clearly differentiated these conditions from hemihyperplasia of the face (see Chapter 22).

Author's Approach to Reconstruction in Patients with Hemihyperplasia

Procedures to improve facial symmetry and Euclidian proportions for the individual with hemihyperplasia of the face are offered when feasible (Fig. 31-5).[197] Overzealous attempts to debulk the soft-tissue enlargement (e.g., resection of the parotid gland, resection or liposuction of the deep layer soft tissues of the cheek) may result in damage to the facial nerve or introduce an "operated" look and are thus generally discouraged.[205] Surgery should be directed at regions and tissue layers that are most amenable to safe improvement. Orthodontic treatment in combination with orthognathic surgery is often a fruitful approach. A maxillary Le Fort I osteotomy to bring the maxillary dental midline back to the facial midline; to correct canting; and to adjust the vertical dimension, horizontal projection, and arch width is frequently useful. This is combined with bilateral sagittal split ramus osteotomies of the mandible to reposition the jaw and also to recontour the inferior border. An oblique osteotomy of the chin for repositioning and also to recontour the inferior border is carried out (see Fig. 31-5).

Vascular Malformations

Background

Vascular anomalies fall into two groups: hemangiomas and vascular malformations. Mulliken and Glowacki were the first to clarify a biologic classification of vascular anomalies on the basis of endothelial properties.[226,231,232] Those authors described how hemangiomas have a proliferative endothelium whereas vascular malformations have a stable endothelium.[218] Hemangiomas have a natural history that is characterized by initial proliferation, stability, and then at least partial involution (Figs. 31-6 and 31-7). Vascular malformations are present at birth, although they may not be clinically evident until sometime later during childhood. In general, vascular malformations grow commensurately with the child, but they may expand suddenly as a result of trauma, infection, or hormonal influences (Figs. 31-8 through 31-11). Vascular malformations generally occur sporadically, although inherited autosomal dominant entities do exist. The International Society for the Study of Vascular Anomalies classification system is based on Mulliken and Glowacki's work.[226] Vascular tumors include hemangiomas, Kaposiform hemangioendotheliomas, tufted angiomas, pyogenic granulomas, and hemangiopericytomas. Vascular malformations include capillary, venous, lymphatic, and arteriovenous conditions; malformations can also be mixed and include combinations of these. From a clinical perspective, it is useful to think in terms of the flow characteristics of the malformations (i.e., either slow flow or fast flow) and the type of vessel drainage.[213-215,217,223-228]

Complications from vascular malformations include venostasis, ischemia, coagulopathy, disseminated intravascular coagulopathy, heart failure, and even death.[209] Skeletal anomalies can occur as a result of intraosseous involvement, skeletal expansion, or skeletal compression (erosion). Any head and neck function can be affected by the presence of a vascular anomaly's tumor-like mass.

The diagnostic imaging of vascular malformations primarily relies on radiographic modalities.[212] Phleboliths are generally pathognomonic for venous malformations. Color Doppler ultrasound is an invaluable tool for the assessment of blood flow velocities and patterns of vascular lesions. Computed tomography plays an important role in the understanding of skeletal involvement. Magnetic resonance imaging is a useful non-invasive and non-ionizing test for the accurate depiction of soft-tissue relationships and allows slow-flow malformations to be accurately differentiated from high-flow malformations. Angiography and venography can play important diagnostic and therapeutic roles in the management of specific vascular malformations. Advantages of venography include the clarification of lesion size and extent and of venous drainage patterns. Angiography can clarify arteriovenous shunting, the presence of macrofistulae and microfistulae, and the location of the lesion's nidus. Therapeutic embolization via angiography with the use of particular materials or coils can be carried out.

Lymphatic Malformations

Lymphatic malformations are slow-flow anomalies.[223,225,226,228] Macrocystic lesions are easier to deal with than microcysts. Microcystic lesions do not resolve

• **Figure 31-5** A 16-year-old girl with left hemihyperplasia of the face that affects both the soft tissues and the skeletal structures. There were distortions of the maxilla and the mandible that were treated through a combined orthodontic and surgical approach. The patient underwent a Le Fort I osteotomy (vertical intrusion and improvement of canting and asymmetry), bilateral sagittal split ramus osteotomies (improvement of asymmetry), and an osseous genioplasty (vertical reduction and horizontal advancement). Note that improvements in the facial skeleton were possible but that the soft-tissue fullness remains. **A,** Frontal views before and after reconstruction. **B,** Profile views before and after reconstruction.

• **Figure 31-5, cont'd C,** Worm's-eye views before and after reconstruction confirm residual soft-tissue fullness. **D,** Occlusal views before and after reconstruction. Note: Maxillo-mandibular reconstruction does not address the soft tissue enlargement.

spontaneously, and they are often harder to manage surgically as a result of their close integration within the normal head and neck soft tissues that must be preserved. Only half of lymphatic lesions are recognized at birth, but the majority are evident by approximately 2 years of age. Skeletal and soft-tissue overgrowth are typical in the craniomaxillofacial region. Cervicofacial lymphatic malformations tend to present with skeletal distortions (e.g., mandibular overgrowth, malocclusion). There may be intraosseous involvement, but skeletal expansion and erosion (remodeling) as a result of mass effect are typical. Symptoms of localized swelling, pain, and erythema associated with infection are common. Significant complications include intralesional bleeding and infection that are often associated with rapid expansion and then moderate regression. Rapid swelling can lead to airway obstruction. Macrocysts may undergo involution if they are ruptured by trauma, or they may be treated via the injection of sclerosing agents or aspiration. However, surgical removal should be considered, when feasible. Complete resection is seldom possible because lymphatic malformations often surround and incorporate normal anatomy that is not deemed appropriate for resection (see Figs. 31-8 and 31-9).[220-222,229,230] Orthognathic procedures would frequently be beneficial but are not often carried out.

Venous Malformations

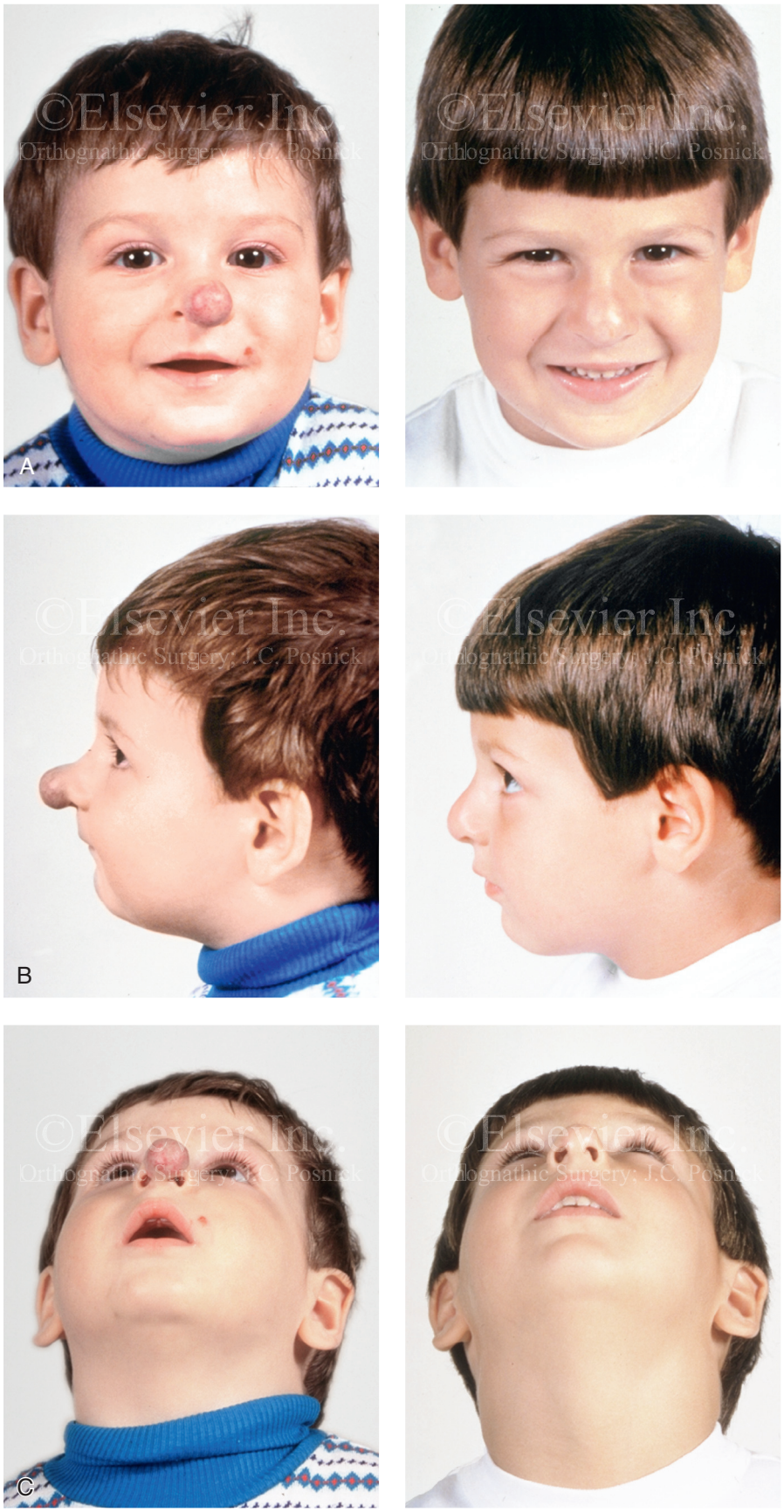
Venous malformations are the most common form seen in the head and neck region.[223,225,226,228] They are present at the time of birth, but they may not have been recognized, or they may have been misdiagnosed.[210,219] Histologically, they are composed of variably sized, thickened, dilated, spongy channels that are devoid of smooth muscle and lined by endothelial cells. Clinically, they are compressible subcutaneous masses that are often deep blue in color. They generally grow commensurate with the child but, as with lymphatic malformations, trauma, infection, and hormonal

• **Figure 31-6** A 5-year-old boy with a hemangioma that involves the soft tissues of the forehead and the upper nose (glabella region) is shown before and 2 years after undergoing a direct vertical midline excision. Note that the soft-tissue growth did not significantly resorb (compress) the underlying skeleton. **A,** Frontal views before and 4 years after reconstruction. **B,** Oblique views before and 4 years after reconstruction.

changes can cause rapid expansion. Venous malformations can involve any tissue layer (i.e., skin, mucosa, muscle, brain, bone, or viscera). Thrombosis within the malformation commonly leads to phlebolith formation. When present, phleboliths are pathognomonic and assist with the accurate diagnosis. Venous malformations are usually unilateral and result in significant facial asymmetry and dysfunction. They may affect speech, swallowing, chewing, breathing, vision, hearing, cognition, and jaw or neck range of motion. They may be intraosseous or have secondary effects on the bone (i.e., expansion, remodeling, or resorption). Treatment to reduce the size of venous malformations includes the injection of sclerosing agents in an attempt to damage the endothelial cells via intravascular thrombosis and fibrosis as well as partial surgical excision (see Figs. 31-8, 31-9, and 31-10).[220,221,222,229]

Arteriovenous Malformations

Arteriovenous malformations are high-flow malformations with a direct connection between an artery and a vein, with no intervening capillary bed.[223,225,226,228] These malformations are also present at birth, but they may have gone unrecognized or have been misdiagnosed as a more



• **Figure 31-7** A 5-year-old boy with a hemangioma that involves the soft tissues of the tip of the nose. He is shown before and after undergoing surgical debulking through a columella-splitting open rhinoplasty technique. The soft-tissue growth displaced but did not destroy the lower lateral cartilages (LLC) or septum; the LLC were reconstructed with the use of a suture pexy technique after tumor debulking. **A,** Frontal views before and after reconstruction. **B,** Profile views before and after reconstruction. **C,** Worm's-eye views before and after reconstruction.

• **Figure 31-8** A newborn child with a massive low-flow mixed (lymphatic–venous) vascular malformation that involved the right side of the face, neck, chest wall, upper arm, and upper back. The malformation was recognized through ultrasonic evaluation during fetal development, and the child was delivered by Cesarean section with a good airway. At 6 weeks of age, he underwent a planned single staged surgical debulking procedure. He retains good cranial nerve and brachial plexus function. Portions of the sternocleidomastoid, trapezius, and platysma muscles were sacrificed in the process. No significant deformation of the craniofacial skeleton resulted. **A,** Frontal facial views before surgery, at 7 months of age, and then at 5 years after resection. **B,** Posterior facial views before surgery, at 7 months of age, and then 5 years after resection. The patient is shown with both arms extended, which indicates satisfactory neuromotor control.

straightforward hemangioma. The confirmation of clinical diagnosis is helpful with color Doppler examination, which can determine flow rate, volume, and reversal of flow. Magnetic resonance imaging can determine the extent of the lesion and its relationship with the surrounding anatomy. Angiography can further clarify the lesion and be used for the therapeutic embolization of an arteriovenous malformations. If an arteriovenous malformation is well localized and can be reliably excised with satisfactory reconstruction, this is always a first choice (see Fig. 31-11). Unfortunately, larger, more extensive lesions are not surgically resectable. Superselective embolization performed by an experienced interventional radiologist with the placement of an ablative agent (e.g., absolute alcohol, sodium tetradecyl sulfate) to induce the destruction of the endothelium may be useful. Embolization that involves coils or particles is another technique for temporarily occluding flow. The ligation of feeding vessels or proximal embolization is generally avoided, because rapid collateralization tends to occur.[220,221,222,229]

Capillary Malformations

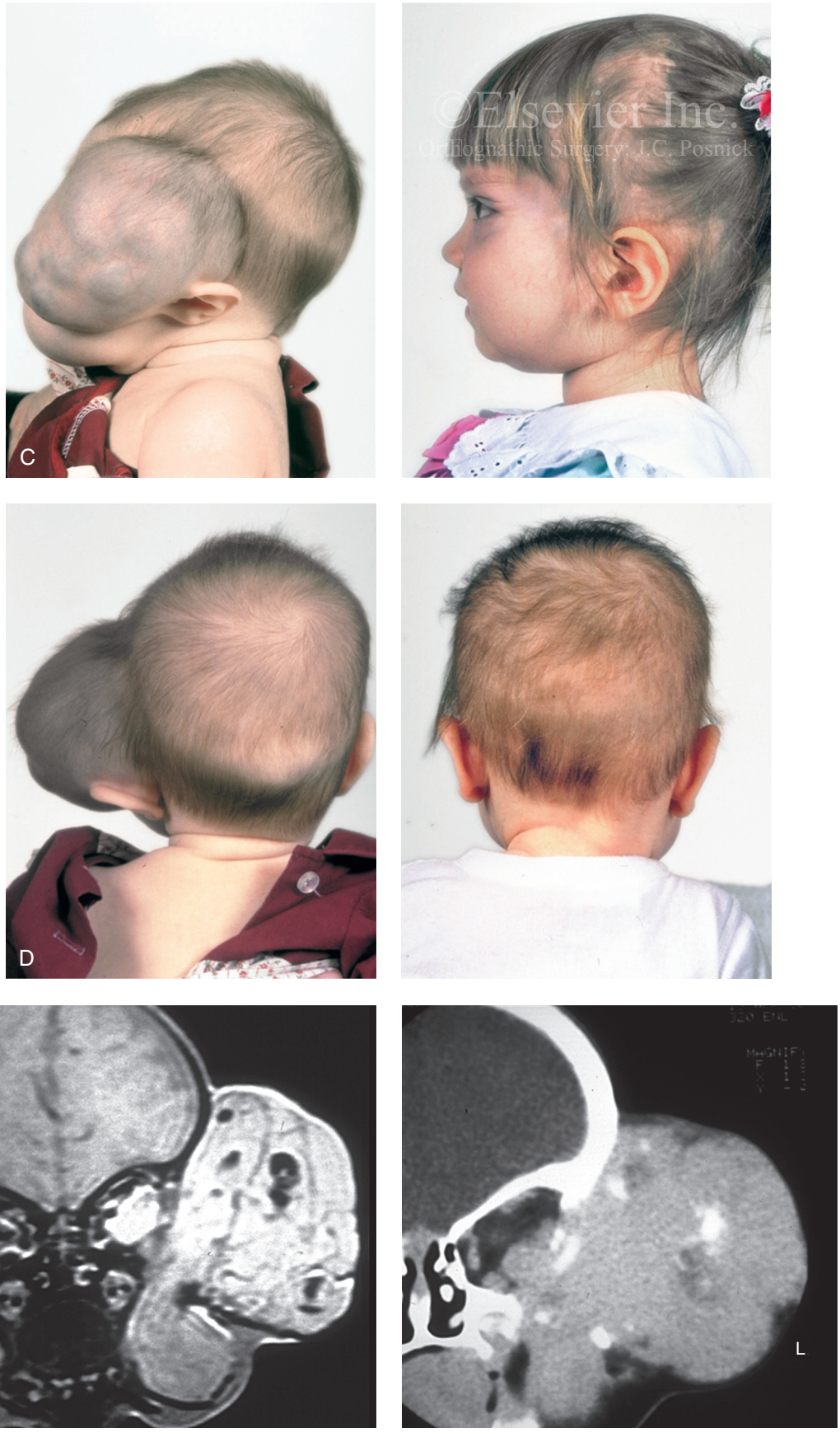
Capillary malformations, which were previously referred to as *port-wine stains,* are intradermal vascular anomalies.[223,225,226,228] They occur in less than 1% of newborns, and they have an equal distribution in male and female individuals.[216] They typically present as either pink or red cutaneous discolorations. They have a tendency to darken, thicken, and become nodular with age. Several conditions are associated with capillary malformations, including Sturge–Weber syndrome, which presents with capillary malformations in the trigeminal nerve distribution in combination with vascular anomalies of the leptomeninges,

Text continued on p. 1288

©Elsevier Inc.
Orthognathic Surgery; J.C. Posnick

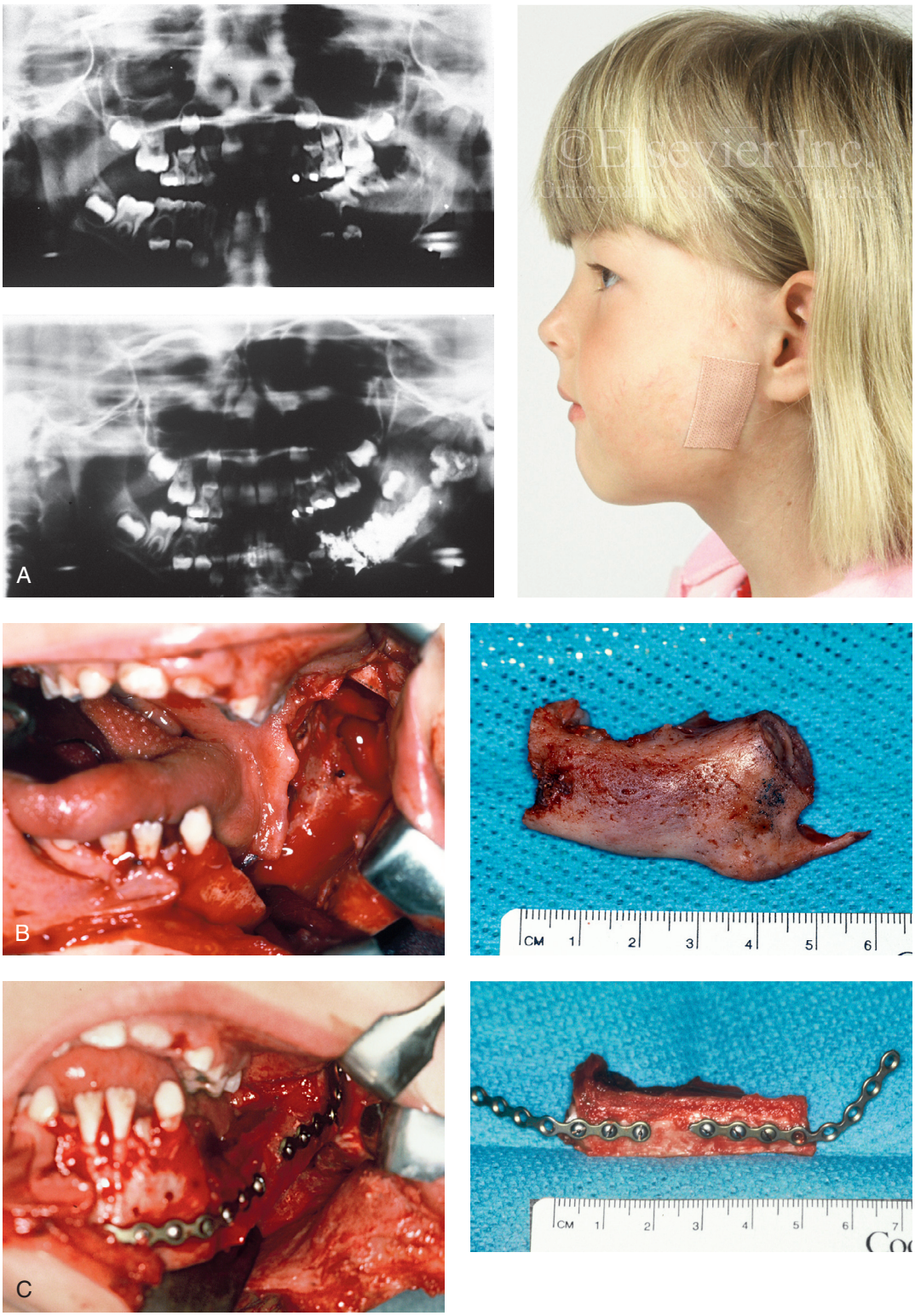
• **Figure 31-9** A child was born with a massive mixed low-flow (venous–lymphatic) vascular malformation of both sides of the neck and the floor of the mouth. He is shown before and after single staged surgical debulking through a bilateral neck (apron) incision. With debulking of the mass, a tracheostomy could be placed to improve oxygenation. Note that the postoperative residual venous engorgement of the tongue remains problematic. Severe deformities of the maxillomandibular skeleton are expected. **A,** Frontal views before and after debulking and tracheostomy. **B,** Oblique views before and after surgery.

• **Figure 31-10** A child who was born with a massive low-flow (venous) vascular malformation that involved the left side of the face underwent single staged resection/reconstruction when she was 6 months old. She is shown before and 1 year after surgical debulking, with facial nerve preservation carried out through a preauricular and coronal scalp incision. A degree of temporal bone compression resulted from the tumor mass. **A,** Frontal views before and after debulking and reconstruction. **B,** Oblique views before and after reconstruction.



©Elsevier Inc.
Orthognathic Surgery, J.C. Posnick

• **Figure 31-10, cont'd C,** Profile views before and after reconstruction. **D,** Posterior views before and after reconstruction. Note that the external ear was repositioned after the tumor was resected. **E,** Preoperative radiographic studies are shown. A magnetic resonance image and a computed tomography scan demonstrate the vascular malformations of the left face and orbit.



• **Figure 31-11** A 6-year-old girl had a high-flow (arteriovenous) vascular malformation that involved the left mandible. She had suffered multiple bleeding episodes before and after embolization and the direct injection of "glue" into the medullary cavity of the left mandible. She is shown before and 2 years after the surgical excision of the left mandible and associated soft tissues with immediate reconstruction involving a vascularized fibula bone flap. **A,** Profile view before surgery with bandage covering the chronic fistula tract. Preoperative Panorex radiographs before and after direct (glue) injection into the medullary cavity of the mandible. **B,** Involved left body–ascending ramus of the mandible excised as a specimen. Intraoral view of the mandible after resection and specimen removal is shown. **C,** View of autogenous fibular bone flap just before and after placement into the mandibular defect. Extended miniplates are attached to the graft and then secured to the native mandible. The vessels were then anastomosed for revascularization.

• **Figure 31-11, cont'd D,** Frontal views 2 years after excision and immediate reconstruction demonstrating satisfactory mouth opening, good facial symmetry, and facial nerve function. Occlusal view after reconstruction. **E,** Anteroposterior and Panorex facial radiographs after reconstruction. *A (photo), B (right), C (right), D (top), From Posnick JC, Wells MD, Zuker RM: Use of the free fibula flap in the immediate reconstruction of pediatric mandibular tumors: report of cases,* J Oral Maxillofac Surg *51:189–196, 1993.*

often with a seizure disorder, developmental delay, and eye involvement (i.e., glaucoma and retinal detachment).[211] Proteus syndrome may also be associated with cutaneous vascular malformations.[215] When feasible, the treatment of capillary malformations with a pulsed dye laser should be considered.

Velocardiofacial Syndrome

Background

Velocardiofacial syndrome (VCFS) is a genetic condition that is characterized by abnormal pharyngeal arch development that results in defective development of the thymus, the parathyroid glands, and the conotruncal region of the heart. Shprintzen and colleagues first described this syndrome in1978.[249] Since then, almost 200 different individual clinical features have been associated with VCFS.[233,240,247] No single individual has been found to have all of the described features. Affected individuals will present with unique facial features, and they may also have cardiac defects, palatal abnormalities, hypernasal speech, hypotonia, defective thymic development, and a variety of learning disabilities.[248]

Approximately 75% of individuals with VCFS have cardiac anomalies. The cardiac defects are usually of the conotruncal type.[234,242] The most common variations include an interrupted aortic arch (50%), truncus arteriosus (35%), and tetralogy of Fallot (16%). Palatal abnormalities predispose these individuals to speech difficulties. Defective thymic development is associated with impaired immune function. Learning disabilities include overt developmental delay, psychiatric disorders, and musculoskeletal defects.[238] Interestingly, ophthalmologic abnormalities occur in approximately 70% of individuals with VCFS; these may include bilateral cataracts, tortuous retinal vessels, and small optic discs.

Approximately 10% of individuals with VCFS will have DiGeorge syndrome, which consists of at least two of the three following features[239]:

1. Thymic aplasia (immune deficiency)
2. Hypoparathyroidism (hypocalcemia)
3. Conotruncal cardiac anomalies

Approximately 15% to 20% of patients with VCFS will be born with Pierre Robin sequence (see Chapter 4). Many individuals are mistakenly categorized as having CHARGE syndrome.

Pathophysiology

VCFS is now known to be a congenital disorder that is caused by a microdeletion at the Q11.2 band, which is located on the long arm (Q) of chromosome 22.[235,243] The microdeletion is known to cause an abnormality of morphogenesis that affects the migration of the neural crest cells and therefore the early development of branchial arches. In approximately 90% of affected individuals, the disorder occurs as a result of a new mutation. In the other 10% of cases, the disorder is inherited from a parent in an autosomal dominant fashion. Affected individuals have a 50% chance of passing VCSF to each offspring.[244]

Epidemiology

The prevalence of VCFS is the United States is approximately 1 in 2000 live births. Internationally, the syndrome occurs in 1 in 4000 live births. The frequency of VCFS in newborns with isolated cleft palate is approximately 8%. There seems to be no gender predilection. The syndrome is present at birth, but it is often unrecognized until childhood or later.[237] Although the heart defect or the cleft palate is generally detected soon after birth, the learning disorders and psychiatric illnesses may not become apparent until the school-aged years.[236] Even then the correct diagnosis is not always made.

DiGeorge syndrome, which involves a total absence of the thymus and a severe T-cell immunodeficiency, is found in less than 0.5% of patients with VCFS.[236,241,245] Most affected individuals have only partial defects, with impaired thymic development and variable defects in their T-cell numbers.

Author's Approach to Reconstruction in Patients with Velocardiofacial Syndrome

Evaluation by clinicians who have experience with clefting usually centers around the issue of hypernasal speech and whether or not a palatal procedure is indicated. Concern about the location of the carotid arteries (when considering the use of a pharyngeal flap) is always part of the discussion.[246]

A percentage of teenagers with VCFS will present with a jaw deformity and malocclusion. These patients will benefit from a comprehensive orthodontic and surgical approach. They generally have a long face growth pattern (see Chapter 21) and require osteotomies of the maxilla, the mandible, and the chin region (Fig. 31-12).

Conclusions

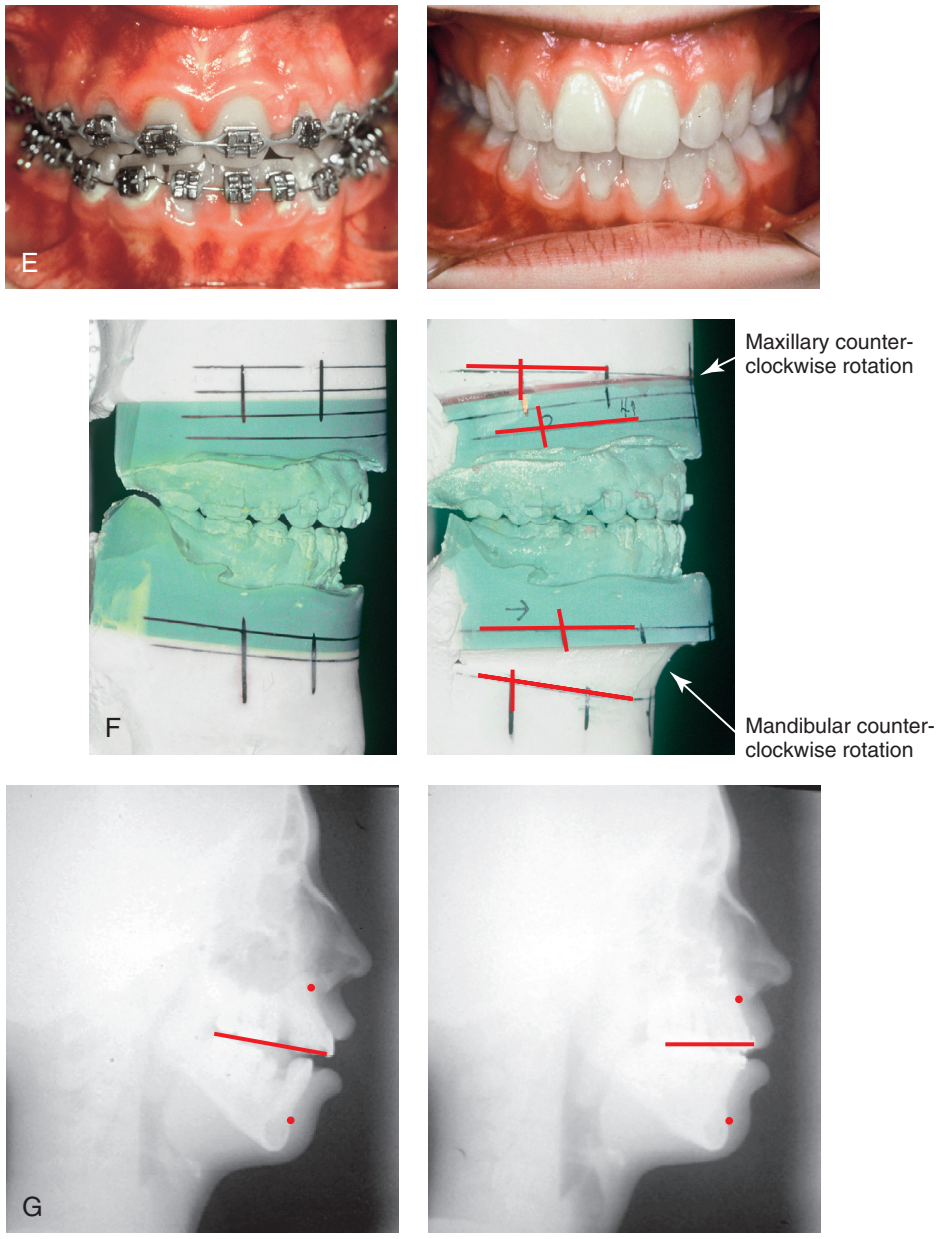
Many hundreds of syndromes and anomalies produce a spectrum of head and neck dysfunction and facial disproportion within the maxillofacial region. Each syndrome type and its presenting set of anomalies will be unique, with individual variations that must considered.

©Elsevier Inc.
Orthognathic Surgery; J.C. Posnick

• **Figure 31-12** A 14-year-old girl who was born with velocardiofacial syndrome. She had mild learning disabilities, an adequately functioning velopharyngeal valve, and mild sibilant distortions with articulation as a result of malocclusion. She had a long face growth pattern that included a skeletal Class II anterior open-bite malocclusion. She underwent orthodontic treatment that included four bicuspid extractions and orthognathic surgery. The patient's procedures included Le Fort I osteotomy (anterior vertical intrusion with counterclockwise rotation) and bilateral sagittal split ramus osteotomies (horizontal advancement with counterclockwise rotation). **A,** Frontal views in repose before and after reconstruction. **B,** Frontal views with smile before and after reconstruction.

Continued

• **Figure 31-12, cont'd C,** Oblique views before and after reconstruction. **D,** Profile views before and after reconstruction.



• **Figure 31-12, cont'd E,** Occlusal views before and after reconstruction. **F,** Articulated dental casts that indicate analytic model planning. **G,** Lateral cephalometric radiographs before and after reconstruction.

References

Klippel–Feil Anomaly

1. Agris J, Dingman RO, Varon J: Correction of webbed neck defects. *Ann Plast Surg* 2:299, 1983.
2. Baba H, Maezawa Y, Furusawa N, et al: The cervical spine in the Klippel–Feil syndrome: A report of 57 cases. *Int Orthop* 19:204, 1995.
3. Baird PA, Robinson GC, Buckler WS: Klippel–Feil syndrome: A study of mirror movements detected by electromyography. *Am J Dis Child* 113:546, 1967.
4. Bauman GI: Absence of the cervical spine: Klippel–Feil syndrome. *JAMA* 98:129, 1932.
5. Bavinck JN, Weaver DD: Subclavian artery supply disruption sequence: Hypothesis of a vascular etiology for Poland, Klippel–Feil, and Moebius anomalies. *Am J Med Genet* 23:903, 1986.
6. Brill CB, Peyster RG, Keller MS, et al: Isolation of the right subclavian artery with subclavian steal in a child with Klippel–Feil anomaly: An example of the subclavian artery supply disruption sequence. *Am J Med Genet* 26:933–940, 1987.
7. Brown MW, Templeton AW, Hodges FJ, 3rd: The incidence of acquired and congenital fusions in the cervical spine. *Am J Roentgenol* 92:1255, 1964.
8. Chandler FA: Webbed neck (pterygium colli). *Am J Dis Child* 53:798, 1937.
9. Chemke J, Nisani R, Fischel RE: Absent ulna in the Klippel–Feil syndrome: An unusual associated malformation. *Clin Genet* 17:167, 1980.
10. Cohney BC: The association of cleft palate with the Klippel–Feil syndrome. *Plast Reconstr Surg* 31:179, 1963.

11. Daniilidis J, Maganaris T, Dimitriadis A, et al: Stapes gusher and Klippel–Feil syndrome. *Laryngoscopy* 88:1178, 1978.
12. Da Silva EO: Autosomal recessive Klippel–Feil syndrome. *J Med Genet* 19:130, 1982.
13. De Bruin M: Pterygium colli congenitum (congenital webbing). *Am J Dis Child* 36:333, 1928.
14. Duncan PA: Embryologic pathogenesis of renal agenesis associated with cervical vertebral anomalies (Klippel–Feil phenotype). *Birth Defects Orig Artic Ser* 13:91, 1977.
15. Erskine CA: An analysis of the Klippel–Feil syndrome. *Arch Pathol* 41:269, 1946.
16. Everberg G, et al: Wildervanck syndrome: Klippel–Feil syndrome associated with deafness and retraction of the eyeball. *Br J Radiol* 36:562, 1963.
17. Fietti VG, Jr, Fielding W: The Klippel–Feil syndrome: Early roentgenographic appearance and progression of the deformity: A report of two cases. *J Bone Joint Surg Am* 58:891, 1976.
18. Foucar HO: Pterygium colli and allied conditions. *Can Med Assoc J* 59:251, 1948.
19. Fragoso R, Cid-Garcia A, Hernandez A, et al: Frontonasal dysplasia in the Klippel–Feil syndrome: A new associated malformation. *Clin Genet* 22:270, 1982.
20. Gardner WJ: Klippel–Feil syndrome, iniencephalus, anencephalus, hindbrain hernia, and mirror movements: Overdistention of the neural tube. *Childs Brain* 5:361, 1979.
21. Gienapp R von: Zur erbbiologie der Klippel–Feilschen krankheit. *Nervenarzt* 21:74, 1950.
22. Gilmour JR: The essential identity of the Klippel–Feil syndrome and iniencephaly. *J Pathol Bacteriol* 53:117, 1941.
23. Ginrup PA, Ginrup L: Klippel–Feil syndrome. *Danish Med Bull* 11:50, 1964.
24. Gorlin RJ, Cohen MM, Jr, Levin LS: *Syndromes of the head and neck*, ed 3, New York, 1990, Oxford University Press.
25. Gray SW, Romaine CB, Skandalakis JE: Congenital fusion of the cervical vertebrae. *Surg Gynecol Obstet* 118:373, 1964.
26. Guille JT, Miller A, Bowen JR, et al: The natural history of Klippel–Feil syndrome: Clinical, roentgenographic, and magnetic resonance imaging findings at adulthood. *J Pediatr Orthop* 15:17, 1995.
27. Gunderson CH, Greenspan RH, Glaser GH, et al: The Klippel–Feil syndrome: Genetic and clinical evaluation of cervical fusion. *Medicine* 46:491, 1967.
28. Gunderson CH, Solitaire GB: Mirror movements in patients with the Klippel–Feil syndrome. *Arch Neurol* 18:675, 1968.
29. Heiner H: Uber die kombination von medianer gaumenspalte und Klippel–Feil syndrom. *Dtsch Stomatol* 10:92, 1960.
30. Helmi C, Pruzansky S: Craniofacial and extracranial malformations in the Klippel–Feil syndrome. *Cleft Palate J* 17:65, 1980.
31. Hensinger RN, Lang JE, MacEwen GD: Klippel–Feil syndrome: A constellation of associated anomalies. *J Bone Joint Surg Am* 56:1246, 1974.
32. Jarcho S, Levin PM: Hereditary malformation of the vertebral bodies. *Bull Johns Hopkins Hosp* 62:216, 1938.
33. Juberg RC, Gershank JJ: Cervical vertebral fusion (Klippel–Feil) syndrome with consanguineous parents. *J Med Genet* 13:246, 1976.
34. Klippel M, Feil A: Un cas d'absence des vertebres cervicales. *Nouv Iconogr Salpet* 25:223, 1912.
35. Lauerman WC: Maurice Klippel. *Spine* 20:157, 1995.
36. Lowry RB: The Klippel–Feil anomalad as part of the fetal alcohol syndrome. *Teratology* 16:53, 1977.
37. Luftman II, Weintraub S: Klippel–Feil syndrome in a full-term newborn infant. *NY State J Med* 51:2035, 1951.
38. Mahajan PV, Bharucha BA: Evaluation of short neck: New neck length percentiles and linear correlations with height and sitting height. *Indian Pediatr* 31:1193, 1994.
39. McLay K, Maran AGD: Deafness and the Klippel–Feil syndrome. *J Otolaryngol* 83:175, 1969.
40. Mecklenburg RS, Krueger PM: Extensive genitourinary anomalies associated with Klippel–Feil syndrome. *Am J Dis Child* 128:92, 1974.
41. Menick FJ, Furnas DW, Achauer BM: Lateral cervical advancement flaps for the correction of webbed-neck deformity. *Plast Reconstr Surg* 73:223, 1984.
42. Moore WB, Matthews TJ, Rabinowitz R: Genitourinary anomalies associated with Klippel–Feil syndrome. *J Bone Joint Surg Am* 57:355, 1975.
43. Morrison SG, Perry LW, Scott LP, 3rd: Congenital torticollis (Klippel–Feil syndrome) and cardiovascular anomalies. *Am J Dis Child* 115:614, 1968.
44. Mosberg WH: The Klippel–Feil syndrome: Etiology and treatment of neurologic signs. *J Nerv Ment Dis* 117:479, 1953.
45. Nagib MG, Larson DA, Maxwell RE, et al: Neuroschisis of the cervical spinal cord in a patient with Klippel–Feil syndrome. *Neurosurgery* 20:629, 1987.
46. Nagib MG, Maxwell RE, Chou SN: Identification and management of high-risk patients with Klippel–Feil syndrome. *J Neurosurg* 61:523, 1984.
47. Nagib MG, Maxwell RE, Chou SN: Klippel–Feil syndrome in children: Clinical features and management. *Childs Nerv Syst* 1:255, 1985.
48. Neidengard L, Carter TE, Smith DW: Klippel–Feil malformation complex in fetal alcohol syndrome. *Am J Dis Child* 132:929, 1978.
49. Nobel TP, Frawley JM: Klippel–Feil syndrome: Numerical reduction of cervical vertebrae. *Ann Surg* 82:728, 1925.
50. Nora JJ, et al: Klippel–Feil syndrome with congenital heart disease. *Am J Dis Child* 102:858, 1961.
51. Palant DI, Carter BL: Klippel–Feil syndrome and deafness: A study with polytomography. *Am J Dis Child* 123:218, 1972.
52. Pizzutillo PD, Woods M, Nicholson L, et al: Risk factors in Klippel–Feil syndrome. *Spine* 19:2110, 1994.
53. Posnick JC: Other craniofacial syndromes: Klippel–Feil anomaly, neurofibromatosis, Down syndrome, micro-ophthalmia, hemifacial hyperplasia (hemi-hypertrophy). In Posnick JC, editor: *Craniofacial and maxillofacial surgery in children and young adults*, Philadelphia, 2000, WB Saunders Co, 24, pp 503–527.
54. Raas-Rothschild A, Goodman RM, Grunbaum M, et al: Klippel–Feil anomaly with sacral agenesis: Additional subtype, type IV. *J Craniofac Genet Dev Biol* 8:297, 1988.
55. Schwarze K: Zur frage des Klippel–Feilschen fehlers der wirbelsule. *Arch Orthop Unfallchir* 41:47, 1941.
56. Shoul MJ, Ritvo M: Clinical and roentgenological manifestations of Klippel–Feil syndrome (congenital fusion of the cervical vertebrae brevicollis): Report of 8 additional cases and review of the literature. *Am J Roentgenol* 68:369, 1952.
57. Slate RK, Posnick JC, Armstrong DC, Buncic JR: Cervical spine subluxation associated with congenital muscular torticollis and craniofacial asymmetry. *Plast Reconstr Surg* 91(7):1187–1195, 1993.
58. Sommerfeld RM, Schweiger JW: Cleft palate with Klippel–Feil *syndrome. Oral Surg* 27:737, 1969.
59. Stadnicki G, Rassumowski D: The association of cleft palate with the Klippel–Feil *syndrome. Oral Surg* 33:335, 1972.
60. Stewart EJ, O'Reilly BF: Klippel–Feil syndrome and conductive deafness. *J Laryngol Otol* 103:947, 1989.
61. Tveter KJ, Kluge T: Cor triloculare biatrium associated with Klippel–Feil syndrome. *Acta Paediatr Scand* 54:489, 1965.
62. Weber V, Schneider HJ, Haberlandt W: Pathogenetische und genetische fragen beim Klippel–Feil syndrom–vergleichende betrachtungen bei zwei vettern. *Fortschr Geb/ Roentgenstr/Nuklearmed* 119:209, 1973.
63. Willemsen WN: Combination of the Mayer–Rokitansky–Kuster and Klippel–Feil syndrome—A case report and literature review. *Eur J Obstet Gynecol Reprod Biol* 13:229, 1982.
64. Windle-Taylor PC, Emery PJ, Phelps PD: Ear deformities associated with the Klippel–Feil syndrome. *Ann Otol Rhinol Laryngol* 90:210, 1981.

Down Syndrome

65. Adams MM, Erickson JD, Layde PM, et al: Down syndrome: Recent trends in the United States. *JAMA* 246:758, 1981.
66. Ardran GM, Harker P, Kemp FH: Tongue size in Down's syndrome. *J Ment Defic Res* 16:160, 1972.

67. Balkany TJ, Downs MP, Jafek BW, et al: Hearing loss in Down syndrome. *Clin Pediatr* 18:116, 1979.
68. Bjuggren G, Jensen R, Strombeck JO: Macroglossia and its surgical treatment: Indications and postoperative experiences from the orthodontic, phoniatric, and surgical points of view. *Scand J Plast Reconstr Surg* 2:116, 1968.
69. Brousseau K: *Mongolism*, Baltimore, Md, 1928, Williams & Wilkins.
70. Dios PD, Feijoo JF, Ferreiro MC, et al: Functional consequences of partial glossectomy. *J Oral Maxillofac Surg* 52:12, 1994.
71. Donaldson JD, Redmond WM: Surgical management of obstructive sleep apnea in children with Down syndrome. *J Otolaryngol* 17:398, 1988.
72. Down JL: Observation on an ethnic classification of idiots. *Ment Retard* 33(1):54–56, 1866.
73. Farkas LG, Munro IR, Kolar JC: Abnormal measurements and disproportions in the face of Down syndrome patients: Preliminary report of an anthropometric study. *Plast Reconstr Surg* 75:159, 1985.
74. Gisel EG, Lange LJ, Niman CW: Tongue movements in 4 and 5 year old Down syndrome children during eating: A comparison with normal children. *Am J Occup Ther* 38:660, 1984.
75. Gorlin RJ, Cohen MM, Jr, Levin LS: *Syndromes of the head and neck*, ed 3, New York, 1990, Oxford University Press.
76. Hinderer U: Malar implants for improvement of the facial appearance. *Plast Reconstr Surg* 56:157, 1975.
77. Hohler H: Changes in facial expression as a result of plastic surgery in mongoloid children. *Aesthetic Plast Surg* 1:245, 1977.
78. Hultcrantz E, Svanholm H: Down syndrome and sleep apnea—a therapeutic challenge. *Int J Pediatr Otorhinolaryngol* 21:263, 1991.
79. Katz S, Kravetz S: Facial plastic surgery for persons with Down syndrome: research finding and their professional such implications. *Am J Ment Retard* 94(2):101–110, 1989.
80. Lefavre J-F, Cohen SR, Burstein FD, et al: Down syndrome: Identification and surgical management of obstructive sleep apnea. *Plast Reconstr Surg* 99:629, 1997.
81. Lejeune J, Turpin R, Gautier M: Le mongolisme, maladie chromosomique (trisomie). *Bull Acad Natl Med* 143:256, 1959.
82. Lemperle G: Plastic surgery in children with Down syndrome. In Lane D, Stratford B, editors: *Current approaches to Down syndrome*, Eastbourne, UK, 1985, Holt, Rinehart & Winston, p 131.
83. Lemperle G, Nievergelt J: Plastisch–chirurgische korrekturen im geicht von kindern mit Down–syndrom. In Plastische Chirurgie bei Menschen mit Down Syndrom. *Lebenshilfe Marburg* 9:19, 1983.
84. Lemperle G, Radu D: Facial plastic surgery in children with Down syndrome. *Plast Reconstr Surg* 66:337, 1980.
85. Lemperle G, Spitalny HH: Long-term experience with silicone implants in the face. In Caronni EP, editor: *Craniofacial surgery*, Boston, 1985, Little, Brown, p 490.
86. Marcus CL, Keens TG, Bautista DB, et al: Obstructive sleep apnea in children with Down syndrome. *Pediatrics* 88:132, 1991.
87. Margar-Bacal F, Witzel MA, Munro IR: Speech intelligibility after partial glossectomy in children with Down syndrome. *Plast Reconstr Surg* 79:44, 1987.
88. Michejda M, Menolascino FJ: Skull base abnormalities in Down syndrome. *Ment Retard* 13:24,1975.
89. Martinez-Frias ML: The real earliest historical evidence of Down syndrome. *Am J Med Genet A* 132A(2):231, 2005.
90. Mearig JS: Facial surgery and an active modification approach for children with Down syndrome: Some psychological and ethical issues. *Rehabil Lit* 46:72, 1985.
91. Olbrisch RR: Plastic surgical management of children with Down syndrome: Indications and results. *Br J Plast Surg* 35:195, 1982.
92. Olbrisch RR: *Plastic surgery in 250 children with Down syndrome: Indications and results. Transactions of the Eighth International Congress on Plastic and Reconstructive Surgery*, Montreal, 1983, International Plastic Surgery Society, p 702.
93. Otermin A: Mongolism (Down syndrome) and plastic surgery. *Prensa Meica Argentina* 60:27, 970–972, 1967.
94. Patterson RS, Munro IR, Farkas LG: Transconjunctival lateral canthopexy in Down syndrome patients: A nonstigmatizing approach. *Plast Reconstr Surg* 79:714, 1987.
95. Posnick JC: Other craniofacial syndromes: Klippel–Feil anomaly, neurofibromatosis, Down syndrome, micro-ophthalmia, hemifacial hyperplasia (hemi-hypertrophy). In Posnick JC, editor: *Craniofacial and maxillofacial surgery in children and young adults*, Philadelphia, 2000, WB Saunders Co, 24, pp 503–527.
96. Pueschel S, Rynders J, editors: *Down syndrome: Advances in biomedicine and the behavioral sciences*, Cambridge, Mass, 1982, Ware Press.
97. Rand Y, Mintsker Y, Feuerstein R: Reconstructive plastic surgery in Down syndrome children and adults: Parents' evaluations—follow-up data. *Jerusalem, Hadassah-Wizo-Canada Research Institute*, 1984.
98. Rolfe CR, Montague JC, Jr, Tirman RM, et al: Pilot perceptual and physiological investigation of hypernasality in Down syndrome adults. *Folia Phoniatr* 31:177, 1979.
99. Rozner L: Facial plastic surgery for Down syndrome. *Lancet* 1:132, 1983.
100. Silverman M: Airway obstruction and sleep disruption in Down syndrome. *Br Med J* 296:1618, 1988.
101. Strome M: Down syndrome: A modern otorhinolaryngological perspective. *Laryngoscope* 91:1581, 1981.
102. Strome M: Obstructive sleep apnea in Down syndrome children: A surgical approach. *Laryngoscope* 96:1340, 1986.
103. Wexler MR, Peled IJ: Plastic surgery in Down syndrome. *Down Syndrome* 6:7, 1983.
104. Wexler MR, Peled IJ, Rand Y, et al: Rehabilitation of the face in patients with Down syndrome. *Plast Reconstr Surg* 77:383, 1986.

Neurofibromatosis

105. Abuelo DN, Meryash DL: Neurofibromatosis with fully expressed Noonan syndrome. *Am J Med Genet* 29:937, 1988.
106. Afifi AK, Dolan KD, Van Gilder JC, et al: Ventriculomegaly in neurofibromatosis. *Neurofibromatosis* 1:299–305, 1988.
107. Baden E, Pierce HE, Jackson WF: Multiple neurofibromatosis with oral lesions: Review of the literature and report of a case. *Oral Surg Oral Med Oral Pathol* 8:268, 1955.
108. Baden E, Jones JR, Khedekar R, et al: Neurofibromatosis of the tongue: A light and electronmicroscopic study with review of the literature from 1849 to 1981. *J Oral Med* 39:157, 1984.
109. Bader JL: Neurofibromatosis and cancer. *Ann NY Acad Sci* 486:57, 1986.
110. Barker D, Wright E, Nguyen K, et al: Gene for von Recklinghausen neurofibromatosis is in the pericentric region of chromosome 17. *Science* 236:1100, 1987.
111. Benedict PH, Szabo G, Fitzpatrick TB, et al: Melanotic macules in Albright syndrome and in neurofibromatosis. *JAMA* 205:618, 1968.
112. Binet FF, Kieffer SA, Martin SH, et al: Orbital dysplasia in neurofibromatosis. *Radiology* 93:829, 1969.
113. Bloem JJ, van der Meulen JC: Neurofibromatosis in plastic surgery. *Br J Plast Surg* 31:50, 1978.
114. Brooks B, Lehman EP: The bone changes in Recklinghausen's neurofibromatosis. *Surg Gynecol Obstet* 38:587, 1924.
115. Buntin PT, Fitzgerald JF: Gastrointestinal neurofibromatosis. *Am J Dis Child* 119:521, 1970.
116. Carey JC, Laub JM, Hall BD: Penetrance and variability in neurofibromatosis: A genetic study of 60 families. *Birth Defects Orig Artic Ser* 15:271, 1979.
117. Chao DH-C: Congenital neurocutaneous syndromes in childhood: I. Neurofibromatosis. *J Pediatr* 55:189, 1959.
118. Clark RD, Hutter JJ, Jr: Familial neurofibromatosis and juvenile chronic myelogenous leukemia. *Hum Genet* 60:230, 1982.
119. Cohen MM, Jr, Hayden PW: A newly recognized hamartomatous syndrome. *Birth Defects Orig Artic Ser* 15:291, 1979.
120. Crawford AH: Neurofibromatosis in children. *Acta Orthop Scand Suppl* 218:1, 1986.

121. Crowe FW, Schull WJ: Diagnostic importance of cafe-au-lait spot in neurofibromatosis. *Arch Intern Med* 91:758, 1953.
122. D'Ambrosio JA, Langlais RP, Young RS: Jaw and skull changes in neurofibromatosis. *Oral Surg* 66:391, 1988.
123. Davis WB, Edgerton MT, Hoffmeister SF: Neurofibromatosis of the head and neck. *Plast Reconstr Surg* 14:186, 1954.
124. Diehl SR, Boehnke M, Erickson RP, et al: A refined genetic map of the region of chromosome 17 surrounding the von Recklinghausen neurofibromatosis (NFI) gene. *Am J Hum Genet* 44:33, 1989.
125. Dreyfuss U, Ben-Arieh JY, Hirshowitz B: Liposarcoma: A rare complication in neurofibromatosis: Case report. *Plast Reconstr Surg* 61:287, 1978.
126. Dunn DW: Neurofibromatosis in childhood. *Curr Probl Pediatr* 17:445, 1987.
127. Farag MZ: Vascular malformation of the parotid gland in von Recklinghausen's disease. *J Laryngol Otol* 97:571, 1983.
128. Fienman NL, Yakovac W: Neurofibromatosis in childhood. *J Pediatr* 76:339, 1970.
129. Freeman AG: Proptosis and neurofibromatosis. *Lancet* 1:1032, 1987.
130. Gorlin RJ, Cohen MM, Jr, Levin LS: *Syndromes of the head and neck*, ed 3, New York, 1990, Oxford University Press.
131. Grabb WC, Reed O, Dingman RMO, et al: Facial hamartomas in children: Neurofibroma, lymphangioma, and hemangioma. *Plast Reconstr Surg* 66:509, 1980.
132. Grant WM, Walton DS: Distinctive gonioscopic findings in glaucoma due to neurofibromatosis. *Arch Ophthalmol* 79:127, 1968.
133. Griffith BH, Lewis YL, Jr, McKinney P: Neurofibromas of the head and neck. *Surg Gynecol Obstet* 160:534, 1985.
134. Hall BD: Congenital lid ptosis associated with neurofibromatosis [letter to the editor]. *Am J Med Genet* 25:595, 1986.
135. Holt JF: Neurofibromatosis in children. *Am J Roentgenol* 130:615, 1978.
136. Holt JF, Wright EM: The radiologic features of neurofibromatosis. *Radiology* 51:647, 1948.
137. Horwich A, Riccardi VM, Francke U: Brief clinical report: Aqueductal stenosis leading to hydrocephalus—an unusual manifestation of neurofibromatosis. *J Med Genet* 14:577, 1983.
138. Hunt JC, Pugh DG: Skeletal lesions in neurofibromatosis. *Radiology* 76:1, 1961.
139. Jackson IT, Carbonnel A, Potparic Z, et al: Orbitotemporal neurofibromatosis: Classification and treatment. *Plast Reconstr Surg* 92:1, 1993.
140. Jackson IT, Laws ER, Martin RD: The surgical management of orbital neurofibromatosis. *Plast Reconstr Surg* 71:751, 1983.
141. Jacobs MH: Oral manifestations in von Recklinghausen's disease (neurofibromatosis). *Am J Orthod Oral Surg* 32:28, 1946.
142. Janecka IP: Correction of ocular dystopia. *Plast Reconstr Surg* 97:892, 1996.
143. Kaplan P, Rosenblatt B: A distinctive facial appearance in neurofibromatosis von Recklinghausen. *Am J Med Genet* 21:463, 1985.
144. Koblin I, Reil B: Changes in the facial skeleton in cases of neurofibromatosis. *J Maxillofac Surg* 3:23, 1975.
145. Kragh LV, Soule EH, Masson JK: Neurofibromatosis (von Recklinghausen's disease) of the head and neck: cosmetic and reconstructive aspects. *Plast Reconstr Surg Transplant Bull* 25:565, 1960.
146. Laue L, Comite F, Hench K, et al: Precocious puberty associated with neurofibromatosis and optic gliomas. *Am J Dis Child* 139:1097, 1985.
147. Lewis RA, Gerson LP, Axelson KA, et al: Von Recklinghausen neurofibromatosis: II. Incidence of optic gliomata. *Ophthalmology* 91:929, 1984.
148. Maceri DS, Saxon KG: Neurofibromatosis of the head and neck. *Head Neck Surg* 6:842, 1984.
149. Marchac D: Intracranial enlargement of the orbital cavity and palpebral remodelling for orbitopalpebral neurofibromatosis. *Plast Reconstr Surg* 73:534, 1984.
150. Martuza RL, Eldridge R: Neurofibromatosis 2 (bilateral acoustic neurofibromatosis). *N Engl J Med* 318:684, 1988.
151. Miller M, Hall JG: Possible maternal effect in severity of neurofibromatosis. *Lancet* 2(8099):1071–1073, 1978.
152. Munro IR, Martin RD: The management of gigantic benign craniofacial tumors: The reverse facial osteotomy. *Plast Reconstr Surg* 65:777, 1980.
153. Opitz JM, Weaver DD: The neurofibromatosis-Noonan syndrome. *Am J Med Genet* 21:477, 1985.
154. Parker DA, Skalko RG: Congenital asymmetry: Report of 10 cases with associated developmental abnormalities. *Pediatrics* 44:584, 1969.
155. Pollack MA, Shprintzen RJ: Velopharyngeal insufficiency in neurofibromatosis. *Int J Pediatr Otorhinolaryngol* 3:257, 1981.
156. Posnick JC: Other craniofacial syndromes: Klippel–Feil anomaly, neurofibromatosis, Down syndrome, micro-ophthalmia, hemifacial hyperplasia (hemi-hypertrophy). In Posnick JC, editor: *Craniofacial and maxillofacial surgery in children and young adults*, Philadelphia, 2000, WB Saunders Co, 24, pp 503–527.
157. Riccardi VM, Kleiner B, Lubs ML: Neurofibromatosis: Variable expression is not intrinsic to the mutant gene. *Birth Defects Orig Artic Ser* 15:283, 1979.
158. Rittersma J, ten Kate LP, Westerink P: Neurofibromatosis with mandibular deformities. *Oral Surg Oral Med Oral Pathol* 33:718, 1972.
159. Shapiro SD, Abramovitch K, Van Dis ML, et al: Neurofibromatosis: Oral and radiographic manifestations. *Oral Surg* 58:493, 1984.
160. Sorensen SA, Mulvihill JJ, Nielsen A: On the natural history of von Recklinghausen neurofibromatosis. *Ann N Y Acad Sci* 486:30, 1986.
161. Spencer WG, Shattock SG: A case of macroglossia neurofibromatosa. *Proc R Soc Med* 1:8, 1908.
162. Tibbles JA, Cohen MM, Jr: The Proteus syndrome: The Elephant Man diagnosed. *Br Med J (Clin Res Ed)* 293:683, 1986.
163. Van Damme PA, Freihofer HPM, De Wilde CM: Neurofibroma in the articular disc of the temporomandibular joint: A case report. *J Craniomaxillofac Surg* 24:310, 1996.
164. Van der Meulen JC: Orbital neurofibromatosis. *Clin Plast Surg* 14:123, 1987.
165. Von Recklinghausen FD: *Uber die multiplen fibrome der haut und ihre Bezichung zu den multiplen neuromen: festschrift fur Rudolph Virchow*, Berlin, 1882, Hirschwald.
166. Weber FP: Neurofibromatosis of the tongue in a child, together with a note on the classification of incomplete and anomalous cases of Recklinghausen disease. *Br J Child Dis* 7:13, 1910.
167. Westerhof W, Delleman JW, Wolters E, et al: Neurofibromatosis and hypertelorism. *Arch Dermatol* 120:1579, 1984.

Hemihypertrophy of the Face

168. Andermann E, et al: A syndrome of hemihypertrophy, hemihypesthesia, hemiareflexia and scoliosis in three unrelated girls. Fifth International Conference on Birth Defects, Montreal, Quebec, Canada, August 21–27, 1977.
169. Arnold EB: Case of hemiacromegaly. *Int J Orthodont* 22:1228, 1933.
170. Bell RA, McTigue DJ: Complex congenital hemihypertrophy: A case report and literature review. *J Pedod* 8:300, 1984.
171. Bergman JA: Primary hemifacial hypertrophy: Review and report of a case. *Arch Otolaryngol* 97:490, 1973.
172. Boxer LA, Smith DL: Wilm's tumor prior to onset of hemihypertrophy. *Am J Dis Child* 120:564, 1970.
173. Cohen MM, Jr: Overgrowth syndromes. In El-Shafie M, Klippel CH, editors: *Associated congenital malformations*, Baltimore, Md, 1981, Williams & Wilkins, pp 71–104.
174. Cohen MM, Jr: A comprehensive and critical assessment of overgrowth and overgrowth syndromes. *Adv Hum Genet* 18:181, 1989.
175. Eisenberg RL, Pfister RC: Medullary sponge kidney associated with congenital hemihypertrophy (asymmetry). *Am J Roentgenol* 116:773, 1972.
176. Fraumeni JF, Jr, Geiser CF, Manning MD: Wilms' tumor and congenital hemihypertrophy: Report of five new cases

and review of literature. *Pediatrics* 40:886, 1967.
177. Fraumeni JF, Miller RW: Adrenocortical neoplasms with hemihypertrophy, brain tumors, and other disorders. *J Pediatr* 70:129, 1967.
178. Furnas DW, Soper RT, Nickman NJ, et al: Congenital hemihypertrophy of the face: Impersonator of childhood facial tumors. *J Pediatr Surg* 5:344, 1970.
179. Gesell A: Hemihypertrophy and twinning: Further study of the nature of hemihypertrophy with report of a new case. *Am J Med Sci* 173:542, 1927.
180. Gonzalez-Crussi F, Lee SC, McKinney M: The pathology of congenital localized gigantism. *Plast Reconstr Surg* 59:411, 1977.
181. Gorlin RJ, Cohen MM, Jr, Levin LS: *Syndromes of the head and neck*, ed 3, New York, 1990, Oxford University Press.
182. Gorlin RJ, Meskin LH: Congenital hemihypertrophy. *J Pediatr* 61:870, 1962.
183. Haicken BN: Congenital hemihypertrophy. *Am J Dis Child* 120:373, 1970.
184. Harkin JC, Reed RJ: Tumors of the peripheral nervous system. In *Atlas of Tumor Pathology, series 2, fascicle 3*, Washington, DC, 1969, Armed Forces Institute of Pathology.
185. Hennessy WT, Cromie WJ, Duckett JW: Congenital hemihypertrophy and associated abdominal lesions. *Urology* 18:576, 1981.
186. Hoyme HE, et al: The incidence of neoplasia in children with isolated congenital hemihypertrophy. David Smith Meeting on Malformations and Morphogenesis, Burlington, Vt, August 11–14, 1986.
187. Khanna JN, Andrade NN: Hemifacial hypertrophy: Report of two cases. *Int J Oral Maxillofac Surg* 18:294, 1989.
188. Kogon SL, Jarvis AM, Daley TD, et al: Hemifacial hypertrophy affecting the maxillary dentition. *Oral Surg Oral Med Oral Pathol* 58:549, 1984.
189. Lenstrup E: Eight cases of hemihypertrophy. *Acta Pediatr Scand* 6:205, 1926.
190. Loh HS: Congenital hemifacial hypertrophy. *Br Dent J* 153:111, 1982.
191. Meadows AT: Wilms' tumor in three children of a woman with hemihypertrophy. *N Engl J Med* 291:23, 1974.
192. Meckel JF: *Uber die seitliche asymmetrie im tierichen korper, anatomische physiologische beobachtungen und untersuchungen*, Renger, 1822, Haile, p 147.
193. Obwegeser HL: Descriptive terminology for jaw anomalies. *Oral Surg Oral Med Oral Pathol* 75:138–140, 1993.
194. Obwegeser HL: *Mandibular growth anomalies: Terminology-aetiology-diagnosis-treatment*, Berlin, Heidelberg, NY, 2001, Springer-Verlag.
195. Obwegeser HL, Mekek M: Hemimandibular hyperplasia—hemimandibular elongation. *J Maxillofac Surg* 14:183–208, 1986.
196. Pollock RA, Newman MH, Burdi AR, et al: Congenital hemifacial hyperplasia: An embryologic hypothesis and case report. *Cleft Palate J* 22:173, 1985.
197. Posnick JC: Other craniofacial syndromes: Klippel–Feil anomaly, neurofibromatosis, Down syndrome, micro-ophthalmia, hemifacial hyperplasia (hemi-hypertrophy). In Posnick JC, editor: *Craniofacial and maxillofacial surgery in children and young adults*, Philadelphia, 2000, WB Saunders Co, 24, pp 503–527.
198. Reed EA: Congenital total hemihypertrophy. *Arch Neurol Psychiatry* 14:824, 1925.
199. Ringrose RE, Jabbour JT, Keele DK: Hemihypertrophy. *Pediatrics* 36:434, 1965.
200. Rudolph DC, Norvold RW: Congenital partial hemihypertrophy involving marked malocclusions. *J Dent Res* 23:133, 1944.
201. Sculerati N, Jacobs JB: Congenital facial hemihypertrophy: Report of a case with airway compromise. *Head Neck Surg* 8:124, 1985.
202. Spielman WR, Marano PD, Kolodny SC, et al: True hemifacial hypertrophy: Report of case. *J Oral Surg* 29:592, 1971.
203. Tommasi AF, Jitomirski F: Crossed congenital hemifacial hemihyperplasia. *Oral Surg Oral Med Oral Pathol* 67:190, 1989.
204. Viljoen D, Pearn J, Beighton P: Manifestations and natural history of idiopathic hemihypertrophy: A review of eleven cases. *Clin Genet* 26:81, 1984.
205. Viteporn S: Surgical-orthodontic treatment in hemifacial hypertrophy: A case report. *Int J Adult Orthodon Orthognath Surg* 8:55, 1993.
206. Wagner R, see Kottmeier HL: Uber hemihypertrophia und heniatrophia corporis totalis nebst spontane extremitatenganrane bei saugliegen im anschluss zu einem ungewohnlichen fall. *Acta Paediatr Scand* 20:543, 1938.
207. Wakefield EG, Hines EA, Jr: Congenital hemihypertrophy: A report of eight cases. *Am J Med Sci* 195:493, 1933.
208. Ward J, Lerner HH: A review of the subject of congenital hemihypertrophy and a complete case report. *J Pediatr* 31:403, 1947.

Vascular Malformations

209. Arneja JS, Gosain AK: Vascular malformations. *Plast Reconstr Surg* 121:195e–206e, 2008.
210. Berenguer B, Burrows PE, Zurakowski D, et al: Sclerotherapy of craniofacial venous malformations: Complications and results. *Plast Reconstr Surg* 104:1, 1999.
211. Burns AJ, Kaplan LC, Mulliken JB: Is there association between hemangioma and syndromes with dysmorphic features? *Pediatrics* 88:1257–1267, 1991.
212. Burrows PE, Mulliken JB, Fellows KE, et al: Childhood hemangiomas and vascular malformations: Angiographic differentiation. *AJR Am J Roentgenol* 141:483, 1983.
213. Cohen MM, Jr: Vasculogenesis, angiogenesis, hemangiomas, and vascular malformations. *Am J Med Genet* 108:265–274, 2002.
214. Cohen MM, Jr: Vascular update: Morphogenesis, tumors, malformations, and molecular dimensions. *Am J Med Genet* 140A:2013–2038, 2006.
215. Cohen MM, Jr, Neri G, Weksberg R: *Overgrowth syndromes*, New York, 2002, Oxford University Press.
216. Enjolras O, Mulliken JB: The current management of vascular birthmarks. *Pediatr Dermatol* 10:311, 1993.
217. Enjolras O, Mulliken JB: Vascular tumors and vascular malformations. *Adv Dermatol* 13:375–423, 1998.
218. Folkman J, D'Amore PA: Blood vessel formation: What is its molecular basis? *Cell* 87:1153, 1996.
219. Hein KD, Mulliken JB, Kozakewich HPW, et al: Venous malformations of skeletal muscle. *Plast Reconstr Surg* 110:1625, 2002.
220. Jackson IT, Carreno R, Potparic Z, et al: Hemangiomas, vascular malformations, and lymphovenous malformations: Classification and methods of treatment. *Plast Reconstr Surg* 91:1216, 1993.
221. Jackson IT, Keskin M, Yavuzer R, et al: Compartmentalization of massive vascular malformations. *Plast Reconstr Surg* 115:10, 2005.
222. Marler JJ, Mulliken JB: Current management of hemangiomas and vascular malformations. *Clin Plast Surg* 32:99, 2005.
223. Mulliken JB: Cutaneous vascular anomalies. *Semin Vasc Surg* 6:204–218, 1993.
224. Mulliken JB: Vascular anomalies. In Aston SJ, Beasley RW, Thorne CHM, editors: *Grabb and Smith's plastic surgery*, ed 5, Philadelphia, 1997, Lippincott-Raven, p 191.
225. Mulliken JB, Fishman SJ, Burrows PE: Vascular anomalies. *Curr Probl Surg* 37:517, 2000.
226. Mulliken JB, Glowacki J: Hemangiomas and vascular malformations in infants and children: A classification based on endothelial characteristics. *Plast Reconstr Surg* 69:412–422, 1982.
227. Mulliken JB, Murray JE, Castaneda AR, et al: Management of a vascular malformation of the face using total circulatory arrest. *Surg Gynecol Obstet* 146:168, 1978.
228. Mulliken JB, Young AE: *Vascular birthmarks: Hemangiomas and malformations*, Philadelphia, 1998, Saunders.
229. Posnick JC: Hemangiomas and vascular malformations of the head and neck: Evaluation and management. In Posnick JC, editor: *Craniofacial and maxillofacial surgery in children and young adults*, Philadelphia, 2000, WB Saunders Co, 26, pp 564–596.
230. Pribaz JJ, Morris DJ, Mulliken JB: Three-dimensional folded free-flap reconstruction of complex facial defects using intraoperative modelling. *Plast Reconstr Surg* 93:285, 1994.
231. Takahashi K, Mulliken JB, Kozakewich HPW, et al: Cellular markers that distinguish

the phases of hemangioma during infancy and childhood. *J Clin Invest* 93:2357, 1994.
232. Vikkula M, Boon LM, Mulliken JB: Molecular genetics of vascular malformations. *Matrix Biol* 20:327, 2001.

Velocardiofacial Syndrome

233. Antshel KM, Fremont W, Kates WR: The neurocognitive phenotype in velo-cardio-facial syndrome: a developmental perspective. *Dev Disabil Res Rev* 14:43–51, 2008.
234. Carotti A, Digilio MC, Piacentini G, et al: Cardiac defects and results of cardiac surgery in 22q11.2 deletion syndrome. *Dev Disabil Res Rev* 14:35–42, 2008.
235. Cuneo BF: 22q11.2 deletion syndrome: DiGeorge, velocardiofacial, and conotruncal anomaly face syndromes. *Curr Opin Pediatr* 13:465–472, 2001.
236. De Smedt B, Swillen A, Ghesquiere P, et al: Pre-academic and early academic achievement in children with velocardiofacial syndrome (del22q11.2) of borderline or normal intelligence. *Genet Couns* 14:15–29, 2003.
237. Digilio MC, Angioni A, De Santis M, et al: Spectrum of clinical variability in familial deletion 22q11.2: from full manifestation to extremely mild clinical anomalies. *Clin Genet* 63:308–313, 2003.
238. Dyce O, McDonald-McGinn D, Kirschner RE, et al: Otolaryngologic manifestations of the 22q11.2 deletion syndrome. *Arch Otolaryngol Head Neck Surg* 128:1408–1412, 2002.
239. Eberle P, Berger C, Junge S, et al: Persistent low thymic activity and non-cardiac mortality in children with chromosome 22q11.2 microdeletion and partial DiGeorge syndrome. *Clin Exp Immunol* 155:189–198, 2009.
240. Goldberg R, Motzkin B, Marion R, et al: Velo-cardio-facial syndrome: a review of 120 patients. *Am J Med Genet* 45:313–319, 1993.
241. Goldmuntz E: DiGeorge syndrome: new insights. *Clin Perinatol* 32:963–978, 2005.
242. Goldmuntz E, Clark BJ, Mitchell LE, et al: Frequency of 22q11 deletions in patients with conotruncal defects. *J Am Coll Cardiol* 32:492–498, 1998.
243. Kelly D, Goldberg R, Wilson D, et al: Confirmation that the velo-cardio-facial syndrome is associated with haplo-insufficiency of genes at chromosome 22q11. *Am J Med Genet* 45:308–312, 1993.
244. McDonald-McGinn DM, Zackai EH: Genetic counseling for the 22q11.2 deletion. *Dev Disabil Res Rev* 14:69–74, 2008.
245. McLean-Tooke A, Spickett GP, Gennery AR: Immunodeficiency and autoimmunity in 22q11.2 deletion syndrome. *Scand J Immunol* 66:1–7, 2007.
246. Mehendale FV, Sommerlad BC: Surgical significance of abnormal internal carotid arteries in velocardiofacial syndrome in 43 consecutive hynes pharyngoplasties. *Cleft Palate Craniofac J* 41:368–674, 2004.
247. Ryan AK, Goodship JA, Wilson DL, et al: Spectrum of clinical features associated with interstitial chromosome 22q11 deletions: a European collaborative study. *J Med Genet* 34:798–804, 1997.
248. Shprintzen RJ: Velo-cardio-facial syndrome: 30 years of study. *Dev Disabil Res Rev* 14:3–10, 2008.
249. Shprintzen RJ, Goldberg RB, Lewin ML, et al: A new syndrome involving cleft palate, cardiac anomalies, typical facies, and learning disabilities: velo-cardio-facial syndrome. *Cleft Palate J* 15:56–62, 1978.

32

Cleft–Orthognathic Surgery: The Unilateral Cleft Lip and Palate Deformity

JEFFREY C. POSNICK, DMD, MD

- Facial Growth Implications of Cleft Palate Repair in the Infant with Unilateral Cleft Lip and Palate
- Coordinated Team Approach
- Treatment Protocol
- Timing of Orthognathic Surgery
- Residual Deformities in the Adolescent with Unilateral Cleft Lip and Palate
- Orthodontic Considerations in the Patient with Unilateral Cleft Lip and Palate with a Jaw Deformity
- Immediate Presurgical Re-Assessment
- Orthognathic Approach for Unilateral Cleft Lip and Palate Deformities
- Avoiding Pitfalls
- Clinical Management after Initial Surgical Healing
- Orthognathic Surgery for Unilateral Cleft Lip and Palate: Review of Study
- Skeletal Stability after Modified Le Fort I for Unilateral Cleft Lip and Palate: Review of Study
- Controversies and Unresolved Issues
- Conclusions

The successful reconstruction of cleft lip and palate is inextricably connected with orthodontic care and skeletal surgery. The negative effects of the primary surgical repair of cleft palate during childhood have been known for the past 200 years.

In 1880, the pioneer orthodontist Normal W. Kingsley stated the following in his remarks about surgical operations as a means of correcting cleft palate: "Although the practice [of cleft palate repair] has been tested in thousands of cases by the most eminent surgeons of their time, it has resulted in such uniformity of failure [i.e., severe jaw deformities], that it should have been utterly abandoned years ago."[85]

In 1920, another preeminent orthodontist of his day, Calvin Case, voiced a similar sentiment: "[L]et us hope that the proportion of [cleft palate] surgical failures will be greatly lessened in the future and that well-informed, honest surgeons … accept only the most favorable cases with a determination to follow them through with proper interest."[16]

In 1965, Vilray Kazanjian, who was a leading maxillofacial surgeon of the first half of the twentieth century, reminded us that "the relative value of surgery to repair a cleft palate has been intelligently questioned for over 150 years."[83]

Facial Growth Implications of Cleft Palate Repair in the Infant with Unilateral Cleft Lip and Palate

Effects of Cleft Palate Repair in Infancy

The management of individuals with unilateral cleft lip and palate (UCLP) presents specific clinical challenges for the maxillofacial surgeon, the orthodontist, and the restorative dental team. Orthognathic surgery is a procedure that should be considered as part of the treatment algorithm for patients with UCLP. Ross completed a multicenter, long-term facial growth study to assess the need for orthognathic surgery among individuals born with complete UCLP who had undergone primary lip and palate repair during

childhood. He concluded that, even by the most conservative standards and in conjunction with maximum compensating orthodontic camouflage maneuvers, at least 25% of adolescents with UCLP required orthognathic surgery to achieve even the limited objective of a neutralized occlusion.[148] His research indicated that only 25% of adolescents with UCLP had near-normal maxillary growth and that another 50% were in a borderline category with some degree of maxillary hypoplasia. Ross stated that individuals who were born with a cleft lip and palate have an intrinsic deficiency in the midfacial skeleton that is made worse by operations. More recently, Mulliken and colleagues reviewed the prevalence of Le Fort I osteotomies among patients with cleft lip and palate who were treated at Boston Children's Hospital.[55] They found that 48% of UCLP patients who underwent repair in infancy later required orthognathic surgery. The study also showed that the need for orthognathic surgery is dependent on the severity of the cleft type as well as the number and extent of previous operative procedures. Similarly, The Hospital for Sick Children in Toronto, Ontario, Canada, found that 48.3% of their patients (i.e. treated since infancy) with complete UCLP required orthognathic surgery. When they looked at all patients with UCLP who were referred to their center, they found that 59.4% needed jaw surgery.[27] A retrospective cohort study of five prominent cleft palate centers in North America compared the maxillomandibular relationships of individuals with non-syndromal complete UCLP (n = 169).[63] The one center that incorporated primary alveolar bone grafting showed especially poor maxillary growth, with 66% of its patients requiring orthognathic surgery. Interestingly, at the one center in which a single surgeon performed all of the surgeries with the use of a more delayed approach to cleft palate repair and whose patients underwent no revisions until they were 14 years old showed the lowest need for orthognathic surgery at less than 25%.

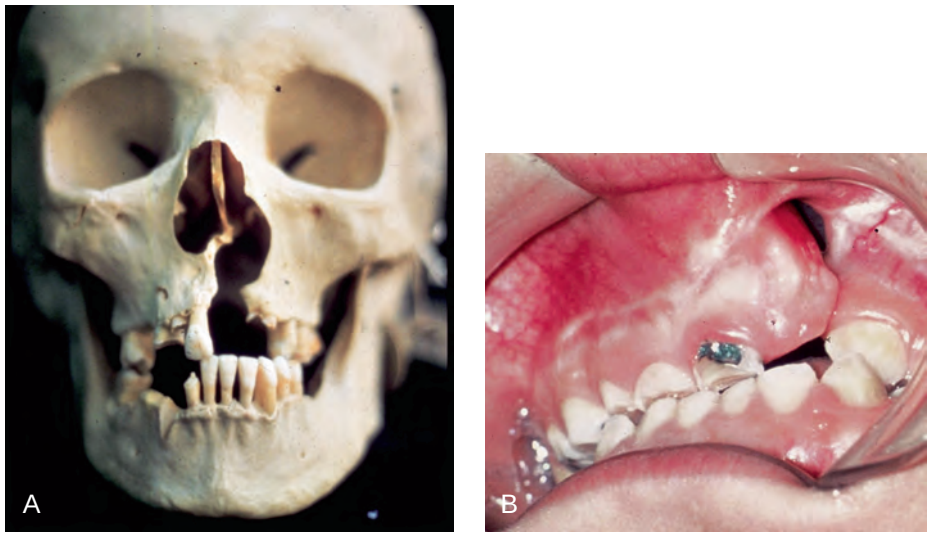
Saperstein and colleagues described the facial growth of children with complete clefting of the primary palate (i.e., the lip through the incisal foramen) but with an intact secondary palate (i.e., the incisal foramen through the uvula).[151] This was a retrospective, cross-sectional analysis of non-syndromal patients with unilateral complete clefting of the primary palate as compared with those with unilateral complete clefting of both the primary and secondary palates. Angular and linear measurements of the midfacial region were made on lateral cephalograms. The study groups included those with unilateral complete clefting of the primary palate (n = 25) and those with unilateral complete clefting of the primary and secondary palate (n = 18). The study documented that individuals with a cleft of only the primary palate who underwent lip repair during infancy typically had a normal or even slightly forward maxillary position as compared with age-matched controls. This was in contrast with children with a cleft of both the primary and secondary palates who underwent lip repair followed by palate repair before they were 1 year old; this group showed a high incidence of maxillary deficiency. The study clarified that the cleft palate repair carried out before 1 year of age—and not the cleft lip repair itself—was responsible for the high incidence of midface hypoplasia.

Effects of Achieving Bone Fusion Across the Cleft Alveolus during Infancy

Reconstruction of the alveolar process in patients with UCLP is an essential part of cleft care. Accomplishing this goal provides support for the alar base of the nose; the teeth in the cleft region; and the periodontium that surrounds those teeth (Figs. 32-1 and 32-2). There are three basic methods that have been described for the closure of the cleft alveolus: 1) primary bone grafting; 2) secondary bone grafting; and 3) gingival periosteoplasty (GPP).[56] The method of primary bone grafting is now generally recognized to result in severe midface growth disturbance and has therefore been universally abandoned throughout the world.[63,148] The use of secondary (mixed dentition) bone grafting is generally recognized as an effective method to avoid the problem of additional midface growth disturbance and to successfully achieve support for the alar base; to provide bone for the eruption of the canine through the grafted cleft site; and to establish effective periodontal support (Figs. 32-3 through 32-6).[1]

The technique of GPP was first described by Skoog in 1965 as a method to achieve fusion across the cleft alveolus at the time of lip repair. The goal was to "remove" the cleft during infancy with the hope that no harm would result.[156] Millard included the use of the Latham device to position the alveolar ridges close to one another before going forward with the GPP procedure at the time of lip repair.[105,106] Grayson and Cutting later proposed the use of a nasoalveolar molding device before the GPP and primary lip repair to accomplish the same alveolar ridge fusion. Several studies confirm that the GPP procedure has a high osteogenic potential that results in the deposition of bone to achieve successful fusion of the alveolar ridge. Unfortunately, clinical studies do not confirm sufficient bone fill to consistently allow for the eruption of the canine through the ridge and to provide ideal periodontal support. Since the late 1970s, clinical studies have been carried out to assess the long-term effects of GPP on midfacial growth in cleft patients.

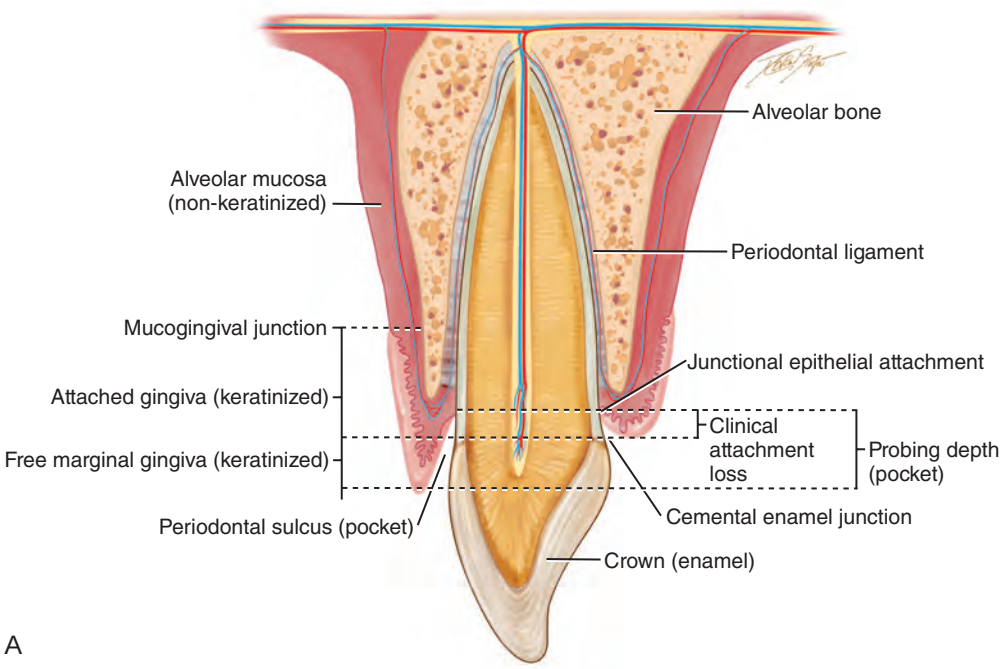
In 1999, Millard and colleagues used serial dental casts to evaluate the effects of GPP on maxillomandibular relationships.[106] They found a greater frequency of anterior crossbite in the GPP group than in the non-GPP group. They also confirmed that the GPP group had a shorter anteroposterior length of the maxilla as compared with the non-GPP group at 6 years of age. Similar findings of poor midface growth have been documented by Berkowitz and colleagues,[9] Matic and Powers,[99] and others.[70,169] In 2005, Renkielska and colleagues evaluated the impact of GPP on occlusal relationships with the use of the Goslon Yardstick occlusal grading system.[143] They found that patients treated with GPP had a poor occlusal relationship and a Goslon Yardstick score of 4 and 5, thus indicating need for

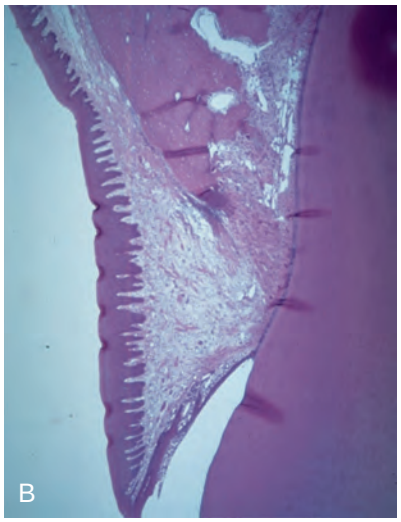


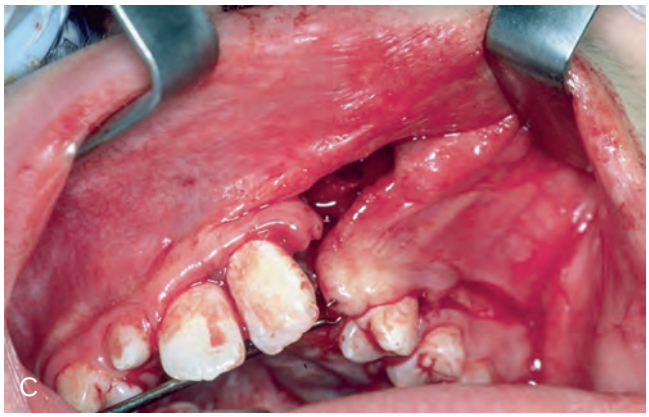
• **Figure 32-1 A,** The maxillofacial skeleton of an adult with unrepaired unilateral cleft lip and palate (UCLP) is shown. Typical UCLP deformities of the hard palate, the alveolus, the nasal septum, the floor of the nose, the nasal spine, and the pyriform rims are demonstrated. **B,** An occlusal view of a child born with UCLP who presented during the mixed dentition with a residual alveolar/palatal skeletal defect and a labial and palatal oronasal fistula is also shown.

• **Figure 32-2** A 6-year-old boy who was born with a UCLP is shown after soft tissue lip and palate repair in infancy. **A,** Frontal and **B,** oblique facial and computed tomography scan views during the mixed dentition demonstrate residual cleft skeletal defects and deformities that were present before grafting and fistula closure.

• **Figure 32-3** An example of the suboptimal management of a unilateral alveolar/palatal cleft. A child who was born with UCLP underwent soft tissue lip and palate repair during infancy. She underwent orthodontic treatment and achieved a satisfactory overjet and overbite. The alveolar/palatal cleft skeletal defects and the residual labial and palatal oronasal fistula were ignored. The maxillary lateral incisor tooth was inadequate and required extraction. The canine erupted adjacent to the cleft, and the dental gap was orthodontically retained. Dental rehabilitation was accomplished with a three-unit bridge to manage the cleft–dental gap. Limited periodontal (bone) support of the cleft-adjacent teeth (the canine and the central incisor) resulted in a periodontic–endodontic problem with a loss of the canine and poor support of the central incisor. These problems would not have occurred had effective bone grafting and fistula closure been carried out during the mixed dentition. **A,** Occlusal and palatal views during the mixed dentition before the full eruption of the canine through the non-grafted alveolar cleft. **B,** Occlusal and palatal views are also shown during orthodontic alignment (extraction of right bicuspid was carried out). **C,** Occlusal view at the completion of orthodontic alignment. A periapical radiograph indicates limited bony support of the cleft-adjacent teeth. **D,** An occlusal view is next shown after the placement of a three-unit bridge across the cleft–dental gap. A periapical radiograph confirms that, after several years, a periodontal–endodontic problem resulted in irreversible injury to the cleft-adjacent canine.

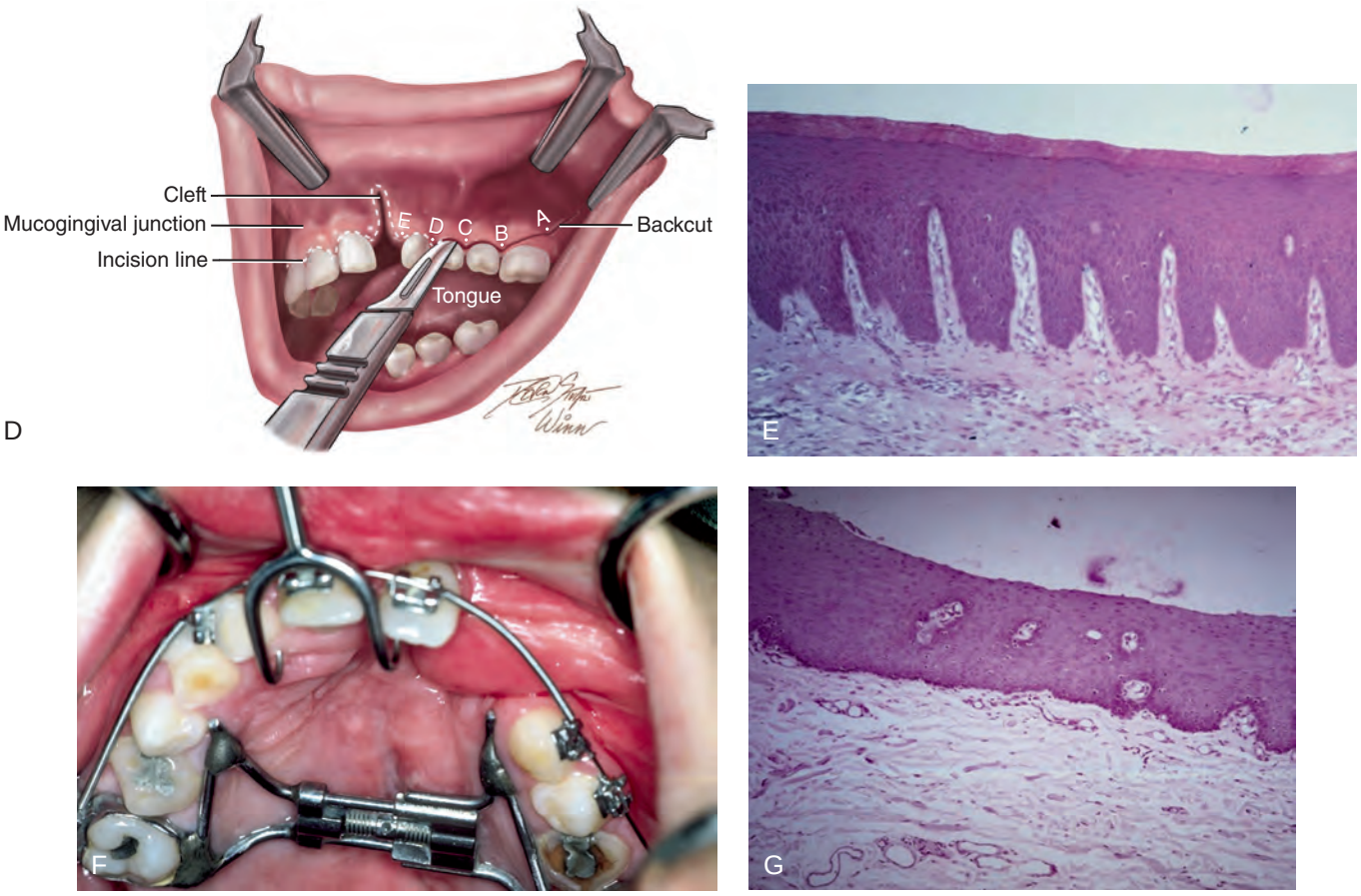






• **Figure 32-4** The importance of maintaining keratinized tissue adjacent to the cervical margin of each tooth for long-term periodontal health cannot be overemphasized (see Chapter 6). **A,** Illustration of a cross-section of dentoalveolar anatomy that indicates the location of the gingiva (keratinized mucosa) adjacent to the tooth surface. **B,** Low-power histomicrograph cross-section of the dentoalveolar anatomy that indicates the microscopic character of the keratinized mucosa adjacent to the tooth surface. Just deep to the keratinized epithelium rete pegs, organized fibrous tissue can be seen. **C,** Intraoperative view of an elevated mucogingival flap advanced anteriorly for the closure of the labial aspect of the cleft oronasal fistula. The flap brings the gingiva into the region where the canine will erupt.

Continued



• **Figure 32-4, cont'd** **D,** Illustration of incision placement for the elevation of the mucogingival flap. The flap splits the attached gingiva posteriorly adjacent to the first molar so that the advancing flap brings keratinized tissue into the cleft site without denuding the attached gingiva from the posterior teeth. **E,** Low-power histomicrograph that demonstrates keratinized epithelium, organized fibrous tissue, and rete pegs. **F,** Palatal view of a patient with UCLP during the mixed dentition. This patient underwent the closure of an oronasal fistula using a buccal "finger" flap by another surgeon. This is *not* the preferred way to rearrange the intraoral tissue for fistula closure; the labial vestibule is destroyed, and non-keratinized buccal mucosa is brought into the region where the canine will erupt. **G,** Low-power histomicrograph of non-keratinized buccal mucosa. Note the thin layer of non-keratinized epithelium with the underlying loose areolar connective tissue. *Part D modified from an original illustration by Bill Winn.*

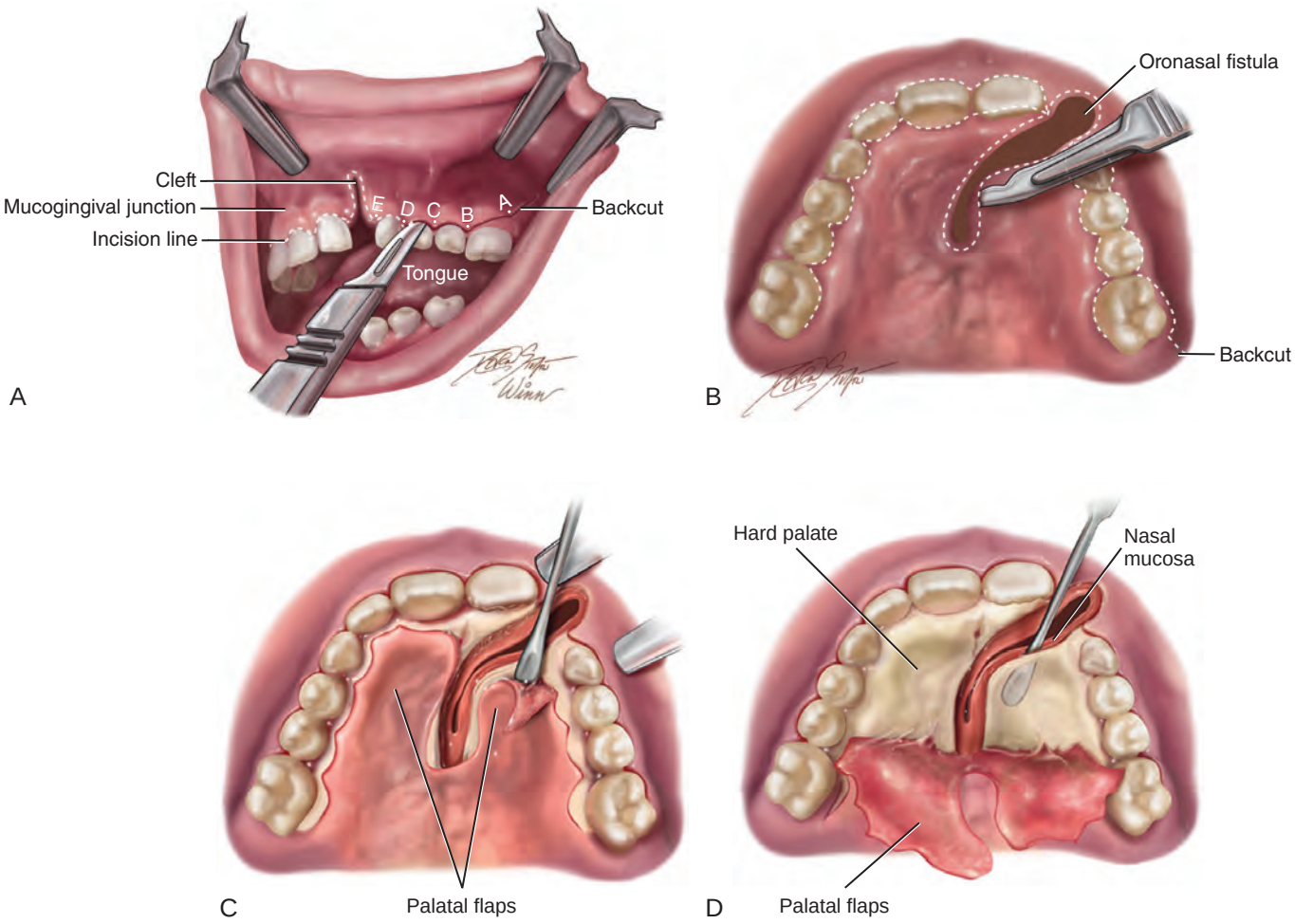
orthognathic surgery. They reaffirmed that the inclusion of the alveolar process in the primary lip repair increased severe occlusal maldevelopment.

More recently, Hsieh and colleagues completed a retrospective clinical study to evaluate the effects of GPP on facial growth in patients with UCLP.[74] Sixty-two consecutive patients with non-syndromal complete UCLP with records from when they were 5 years old were included in the study. All of the patients had received nasoalveolar molding treatment before primary lip repair. Those individuals who underwent GPP at the time of lip repair (n = 26) were placed in one group. Those that did not undergo GPP at the time of lip repair (n = 36) were placed in another group. Cephalometry was used to evaluate facial growth at 5 years of age in the two treatment groups. GPP was found to have significant negative effects on the maxillary position (i.e., diminished horizontal projection), the intermaxillary position (i.e., negative overjet), the maxillary length, and the maxillary alveolar length at the age of 5 years. The authors concluded that, in patients with UCLP, the sagittal growth of the maxilla was significantly adversely affected by the GPP procedure. As a result of their study, the Chang Gung Cleft Center no longer uses the GPP technique.

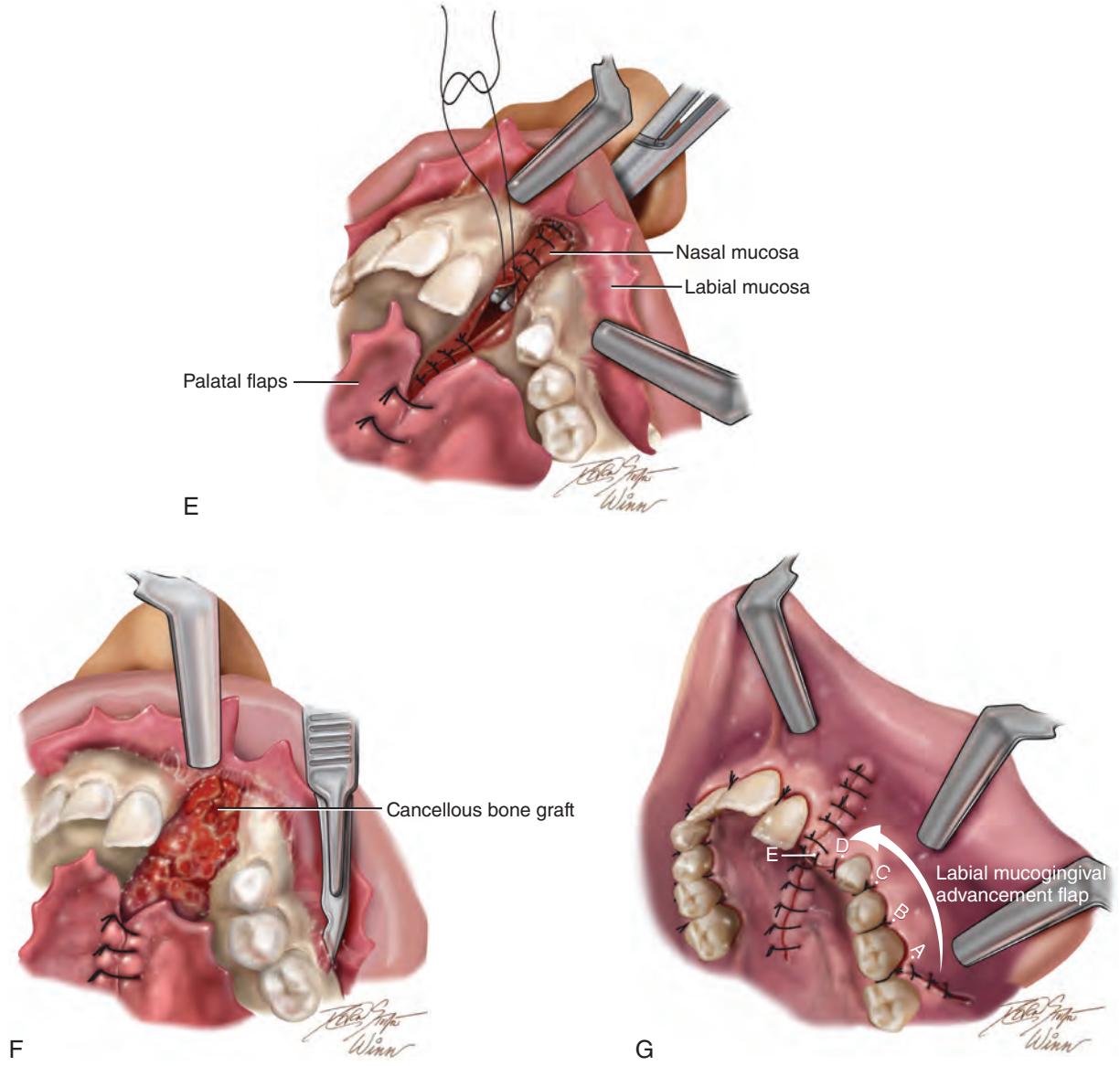
Coordinated Team Approach

Care of the patient with UCLP is best delivered by an integrated group of specialists who evaluate and provide comprehensive definitive care. It is no longer acceptable for individual practitioners (e.g., surgeons, orthodontists, restorative dentists, speech pathologists, otolaryngologists) to carry out extended treatment without considering all aspects of the patient's care and without discussing options with members of the team.[82,90]

A frequent road block to successful reconstruction and dental rehabilitation of the midface-deficient adolescent with UCLP—especially in those who present with other residual clefting problems (see the section about residual deformities later in this chapter)—is disagreement between clinicians about the indications, the most effective techniques, and the timing of intervention.[8,104,123] The surgeon, the orthodontist, the dental and medical team, and the



• **Figure 32-5** Illustrations of basic technique for bone grafting and the management of residual oronasal fistula during the mixed dentition of a child with a UCLP. If required, orthodontic expansion to correct the arch width precedes mixed-dentition bone grafting. **A,** A view of the left labial mucogingival incision in progress. **B,** Attention is turned to the sharp palate for the separation of the oral and nasal mucosa at the site of the fistula and then the elevation of the flaps. **C,** Subperiosteal elevation of the palatal flaps. **D,** With the palatal flaps elevated, the nasal mucosa is separated from the floor of the nose on each side. Left and right labial flaps have already been elevated. The nasal mucosa is further separated from the bony surface along the distal aspect of the central incision for later closure. *Part A modified from an original illustration by Bill Winn.* *Continued*



• **Figure 32-5, cont'd** **E,** Suturing of the nasal flaps for watertight closure is in progress. A hemostat is placed through the left nostril to demonstrate the partially sutured nasal floor. The palatal flaps have been sutured together for fistula closure. **F,** After the nasal flaps are sutured, iliac cancellous bone graft is packed into the palatal, alveolar, and floor-of-the-nose skeletal defects. **G,** Left and right labial and palatal flaps are advanced for watertight closure. By advancing the left labial mucogingival flap, the keratinized tissue has been placed over the alveolar ridge, where the canine will eventually erupt. This is accomplished by splitting the attached gingiva at the last molar.

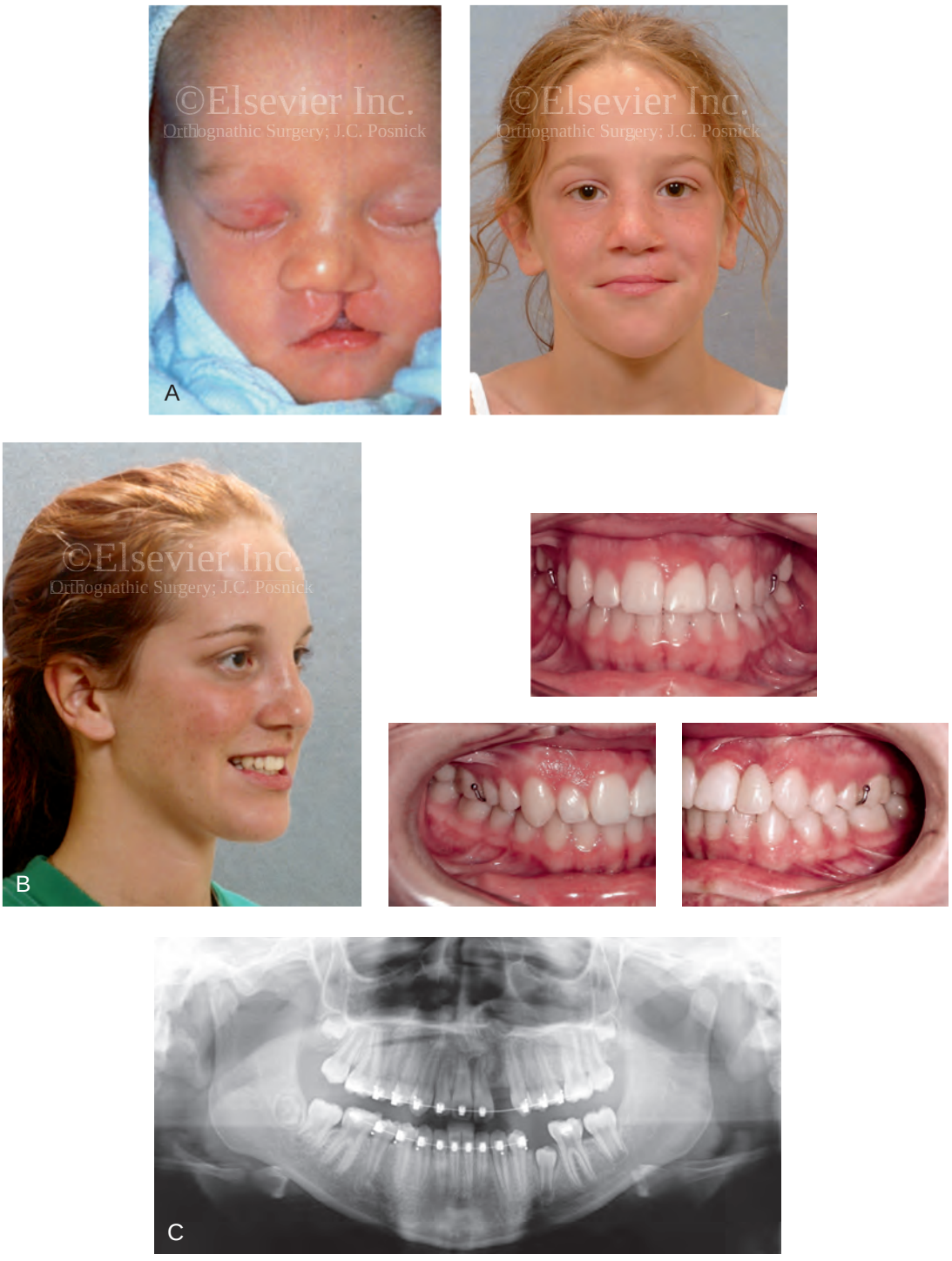
patient and his or her family must first agree about the dental, occlusal, speech, upper airway, and aesthetic objectives; only then can effective treatment go forward.

The advantage of coordinated care was confirmed by the Eurocleft Study, which found a lack of association between high-intensity disjointed treatment and favorable results. In other words, the greater the number of operations and the greater the number of years of orthodontic appliances worn (i.e., heavy burden of care), the worse the outcome.[25,108]

Treatment Protocol

Patients who present with a UCLP jaw deformity after being referred for possible orthognathic surgery are seen at a minimum by an orthodontist, an orthognathic surgeon, an otolaryngologist, and a speech pathologist. Additional consultations with other dental (ex. prosthodontist, pediatric or general dentist, periodontist) and medical (i.e., sleep specialist, geneticist) specialists are carried out as indicated. For the evaluation of a cleft dentofacial deformity, records and tests should include medical-quality photographs, cephalometric and dental radiographs, dental models, direct facial measurements, speech and velopharyngeal (VP) assessment (e.g., nasoendoscopic instrumentation), and a thorough evaluation of the upper airway.

The primary surgeon not only performs repair of the cleft lip and palate and corrects VP insufficiency but also plays a role in directing the patient's overall care. If the primary



• **Figure 32-6** A 16-year-old girl was born with van der Woude syndrome, including UCLP and lower lip pits. She has been managed by this surgeon since her birth. She underwent lip and palate repair during her early childhood, which was followed by successful bone grafting and fistula closure during the mixed dentition. She had normal maxillary growth and underwent standard orthodontic treatment with the maintenance of the cleft–dental gap. She underwent an open rhinoplasty that included the use of a septal cartilage (caudal strut) graft when she was 15 years old. There is sufficient bone volume and attached gingiva for a dental implant, which is planned for when she is 18 years old. **A,** Facial views at the time of birth and during the mixed dentition, just before alveolar/palatal bone grafting. **B,** Facial and occlusal views taken when the patient was a teenager after rhinoplasty and before dental implant placement. A removable retainer that includes the missing lateral incisor is in place. **C,** Panorex image taken when the patient was a teenager during orthodontic treatment indicates successful bone grafting of the alveolar cleft.

cleft surgeon is not trained in skeletal procedures, then a timely and seamless transition to the orthognathic maxillofacial surgeon should occur. The surgeon who is caring for the patient with clefting and a skeletal deformity should have a fundamental understanding of the patient's dental, speech, upper airway, and aesthetic needs. He or she should request consultation with appropriate specialists, evaluate the clinical information, and be prepared to perform orthognathic and intranasal procedures.

The orthodontist provides interceptive treatment during the mixed dentition in association with bone grafting and carries out definitive orthodontic treatment in conjunction with orthognathic surgery, when indicated. From the mixed dentition phase, the cleft orthodontist should recognize the patient with UCLP who may require orthognathic surgery.* The institution of extensive camouflage (dental compensatory) treatment is likely to jeopardize periodontal health and lead to late dental relapse. Proceeding with a compromised (camouflage) orthodontic approach should only be entered into with full disclosure to the family and other treating clinicians.

Before orthognathic surgery, a speech pathologist evaluates the patient to characterize VP function and to identify articulation errors that result from the cleft palate jaw deformity and the dental malocclusion (see Chapter 8). A baseline evaluation is important, because VP function may deteriorate after maxillary advancement. A nasoendoscopic guided speech assessment is useful to provide maximum objective data.[180] VP closure that is adequate before surgery may become borderline afterward, and VP closure that is borderline may become inadequate. Studies document that only a small percentage of patients require a primary pharyngeal flap or flap revision after maxillary advancement.[19,20,57,60,79,86,91,95,102,125,170,180] Articulatory distortions that result from malocclusion are also identified and cause-and-effect relationships are determined. The successful orthodontic and surgical correction of crossbites, open bite, cleft–dental gaps, negative overjet, and residual oronasal fistulas represents the most effective way to correct the identified articulation distortions (see Chapter 8). Unfortunately, the use of "oral–motor therapy" is still often applied to manage both jaw-deformity–related articulation errors and VP insufficiency; however, this type of treatment has not been proven beneficial for dynamic speech.

A thorough evaluation of the upper airway is conducted to assess for areas of obstruction (see Chapter 10). Studies suggest an increased prevalence of sleep-disordered breathing and obstructive sleep apnea in patients with cleft palate, especially in the presence of Robin sequence. A formal sleep study (i.e., an attended polysomnogram) is performed if there is a suggestion of obstructive sleep apnea (see Chapter 26). If indicated, simultaneous intranasal procedures (e.g., septoplasty; reduction of the inferior turbinates; recontouring of the nasal apertures, the floor of the nose, and the anterior nasal spine) should be carried out at the time of orthognathic surgery (see Chapters 10 and 15).[150]

Discussions among the treating medical and dental consultants, the patient, and the family clarify the need for and the extent of orthognathic and intranasal procedures. The overall plan for speech, jaw, upper airway, and dental rehabilitation and for the enhancement of facial aesthetics is agreed upon before the initiation of treatment.

Timing of Orthognathic Surgery

Definitive correction of the jaw deformity is best carried out when the skeleton is mature and before the patient finishes high school.[82,90] Maxillofacial growth is generally complete between the ages of 14 and 16 years in girls and 16 and 18 years in boys. However, skeletal growth is variable and may be further gauged by an analysis of sequential lateral cephalometric radiographs taken at 6-month intervals (see Chapter 17). The patient's and the family's preferences for the timing of the operation on the basis of psychosocial and functional needs are also taken into account.

As early as 1986, investigators showed that, if maxillary advancement is performed during the mixed dentition in a patient with a cleft palate, then another orthognathic procedure will be necessary for definitive correction after skeletal maturity is reached.[181-184] All research to date indicates that a Le Fort I osteotomy carried out during the mixed dentition in a patient with clefting—whether with standard or distraction osteogenesis (DO) techniques—results in no further horizontal maxillary growth (see the controversies section later in this chapter).[21,41,40,75]

Residual Deformities in the Adolescent with Unilateral Cleft Lip and Palate

Correction of the residual skeletal, soft-tissue, and dental deformities in the adolescent patient with UCLP challenges the ingenuity and skill of the orthognathic surgeon and the cleft team. The central deformity is maxillary hypoplasia (Figs. 32-7, 32-8, and 32-9), and it is frequently combined with residual oronasal fistula, bone defects, intranasal obstruction, soft-tissue scarring, and, occasionally, VP dysfunction (Figs. 32-10 through 32-21).[84,167] In addition, the maxillary lateral incisor at the cleft site is usually congenitally absent or deficient, thereby resulting in a cleft–dental gap. Secondary deformities of the nose, the mandible, and the chin region are also common.

The prevalence of these residual clefting deformities in mature patients with UCLP varies widely depending on the primary cleft surgeon's philosophy, available expertise, the individual's intrinsic biologic growth potential, and the patient's and family's interests. Published clinical surveys of individuals who were born with complete UCLP and treated at established cleft centers provide insight into alveolar cleft management. The studies indicate that, despite a

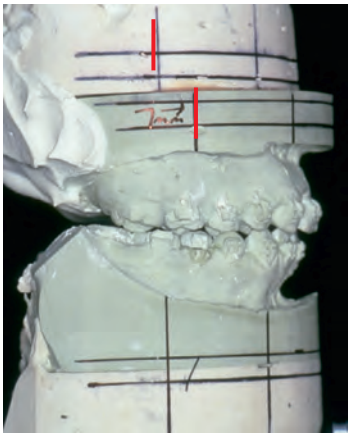
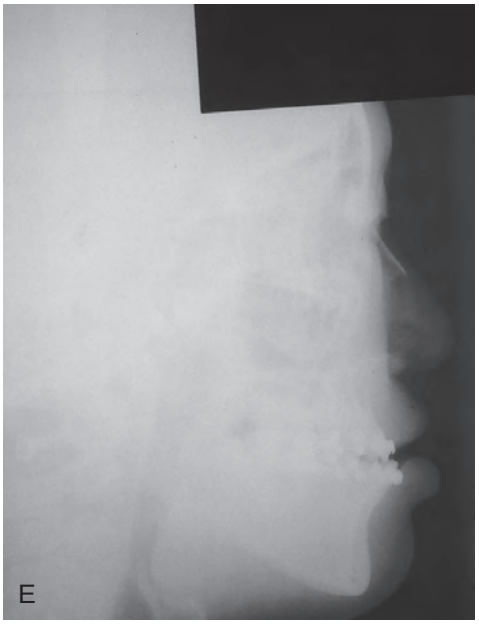
Text continued on p. 1355

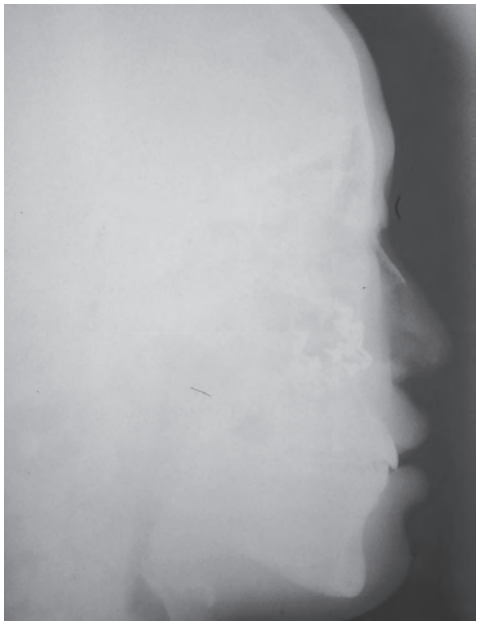
*References 14, 15, 28, 29, 42, 47-49, 54, 80, 98, 96, 97, 109-111, 121, 144, 147, 152-154, 157, 161

©Elsevier Inc.
Orthognathic Surgery; J.C. Posnick

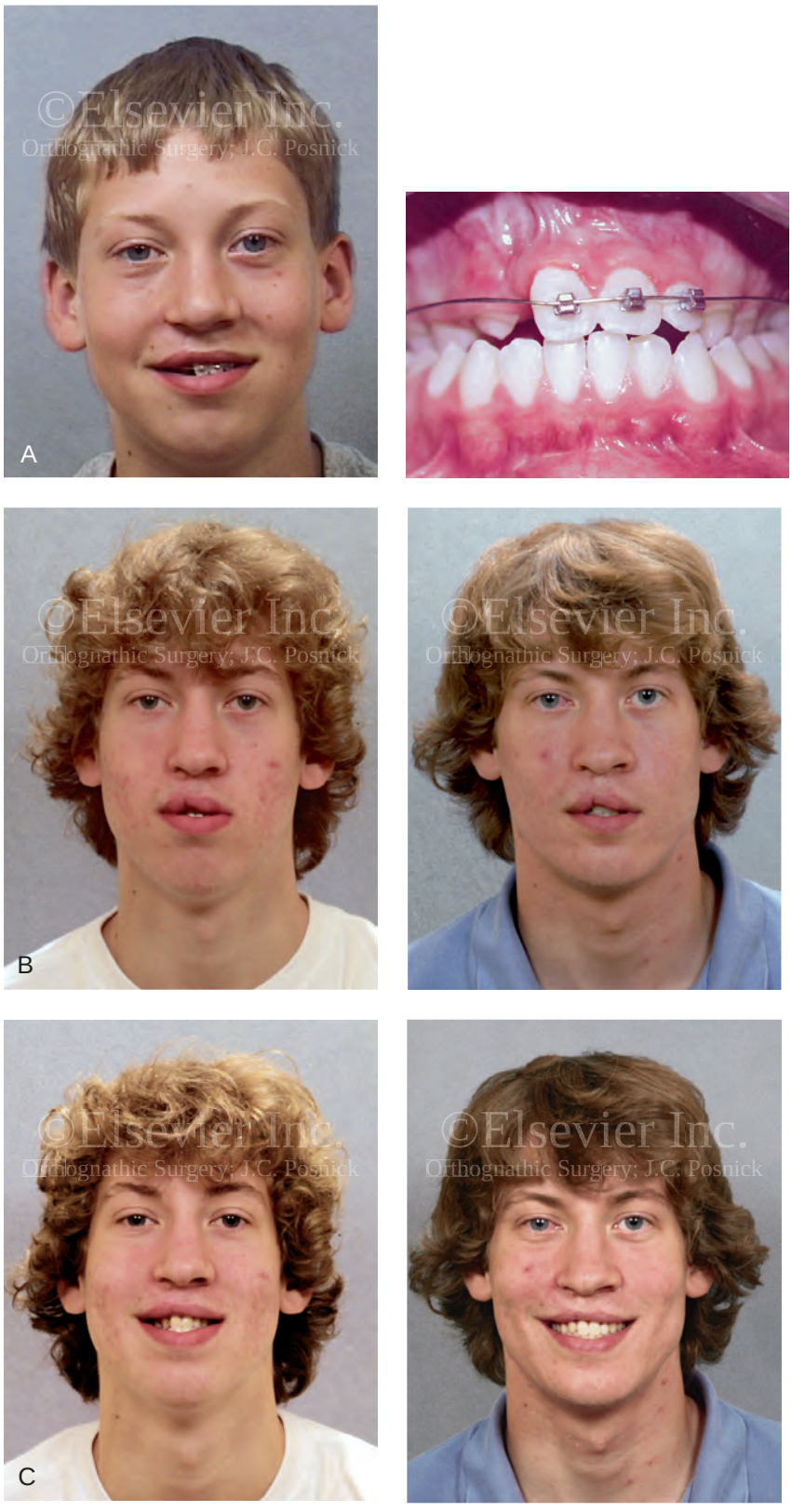
• **Figure 32-7** A 19-year-old woman was born with UCLP on the left side and underwent lip and palate repair followed by effective bone grafting and fistula closure during the mixed dentition. She has a useful lateral incisor at the cleft site but is missing the lateral incisor on the non-clefted side. A bicuspid has also been removed on the cleft side. As a teenager, she was referred to this surgeon and underwent a combined orthodontic and orthognathic surgical approach. The procedure included a standard Le Fort I osteotomy (horizontal advancement, vertical lengthening) and interpositional grafting. **A,** Frontal views with smile before and after reconstruction. **B,** Profile views before and after reconstruction.

Continued



• **Figure 32-7, cont'd C,** Occlusal and palatal views before and after reconstruction. **D,** Articulated dental casts that indicate analytic model planning. **E,** Lateral cephalometric radiographs before and after reconstruction. Note that, by limiting surgery to the maxilla, the cant (which also involved the mandible) cannot be corrected.



©Elsevier Inc.
Orthognathic Surgery; J.C. Posnick

• **Figure 32-8** A 20-year-old man was born with UCLP on the right side. Primary lip and palate repair were carried out at another institution. He was referred to this surgeon and underwent successful bone grafting and fistula closure during the mixed dentition. He underwent orthodontic closure of the cleft–dental gap and limited alignment by the time he was 14 years old. Final orthodontic decompensation was later carried out in combination with orthognathic surgery when he was 19 years old. The patient's procedures included maxillary Le Fort I osteotomy (horizontal advancement, vertical shortening, midline correction, cant correction, and clockwise rotation) with interpositional grafting; sagittal split ramus osteotomies (counterclockwise rotation and asymmetry correction); osseous genioplasty (horizontal advancement); and septoplasty, inferior turbinate reduction, and recontouring of the floor of the nose. **A,** The patient is shown during the mixed dentition after successful bone grafting and fistula closure. **B,** Frontal views in repose before and after reconstruction. **C,** Frontal views with smile before and after reconstruction.

Continued

• **Figure 32-8, cont'd** **D,** Oblique facial views before and after reconstruction. **E,** Profile views before and after reconstruction.

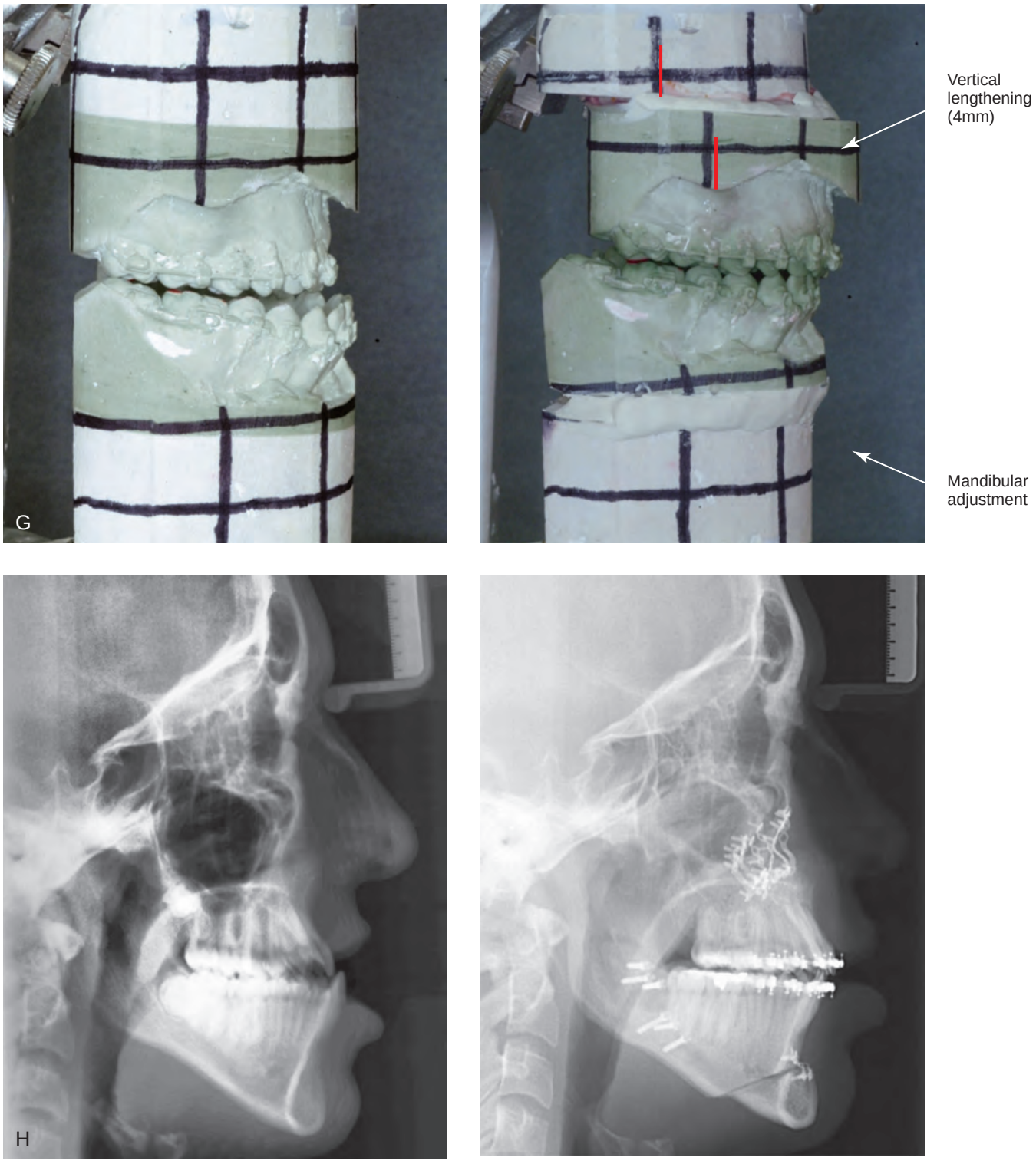
Prior to treatment with successful mixed dentition graft

Pre surgery

After treatment

• **Figure 32-8, cont'd F,** Occlusal views before definitive orthodontics, with decompensation in progress before surgery, and after reconstruction.

Continued



• **Figure 32-8, cont'd** **G,** Articulated dental casts that indicate analytic model planning. **H,** Lateral cephalometric radiographs before and after reconstruction.

• **Figure 32-9** A high school senior was born with a complete UCLP on the left side. He underwent lip and palate repair at another institution. He was then referred to this surgeon and underwent successful bone grafting and fistula closure during the mixed dentition. He developed a jaw deformity that was characterized by maxillary deficiency and secondary deformities of the mandible and the intranasal cavity. He underwent a combined orthodontic and surgical approach. Orthodontic decompensation included cleft–dental gap closure (absent lateral incisor). He then underwent jaw reconstruction. The patient's procedures included maxillary Le Fort I osteotomy (horizontal advancement, vertical shortening, midline correction, cant correction, and clockwise rotation) with interpositional grafting; sagittal split ramus osteotomies (clockwise rotation and asymmetry correction); osseous genioplasty (vertical shortening); and septoplasty, inferior turbinate reduction, and recontouring of the floor of the nose. Six months after successful orthognathic surgery, the patient underwent cleft rhinoplasty including rib cartilage (caudal strut) grafting. **A,** The patient is shown during the mixed dentition before bone grafting and fistula closure. **B,** Frontal views in repose before and after reconstruction. **C,** Frontal views with smile before and after reconstruction.

Continued

©Elsevier Inc.
Orthognathic Surgery; J.C. Posnick

• **Figure 32-9, cont'd** **D,** Oblique facial views before and after reconstruction. **E,** Profile views before and after reconstruction.

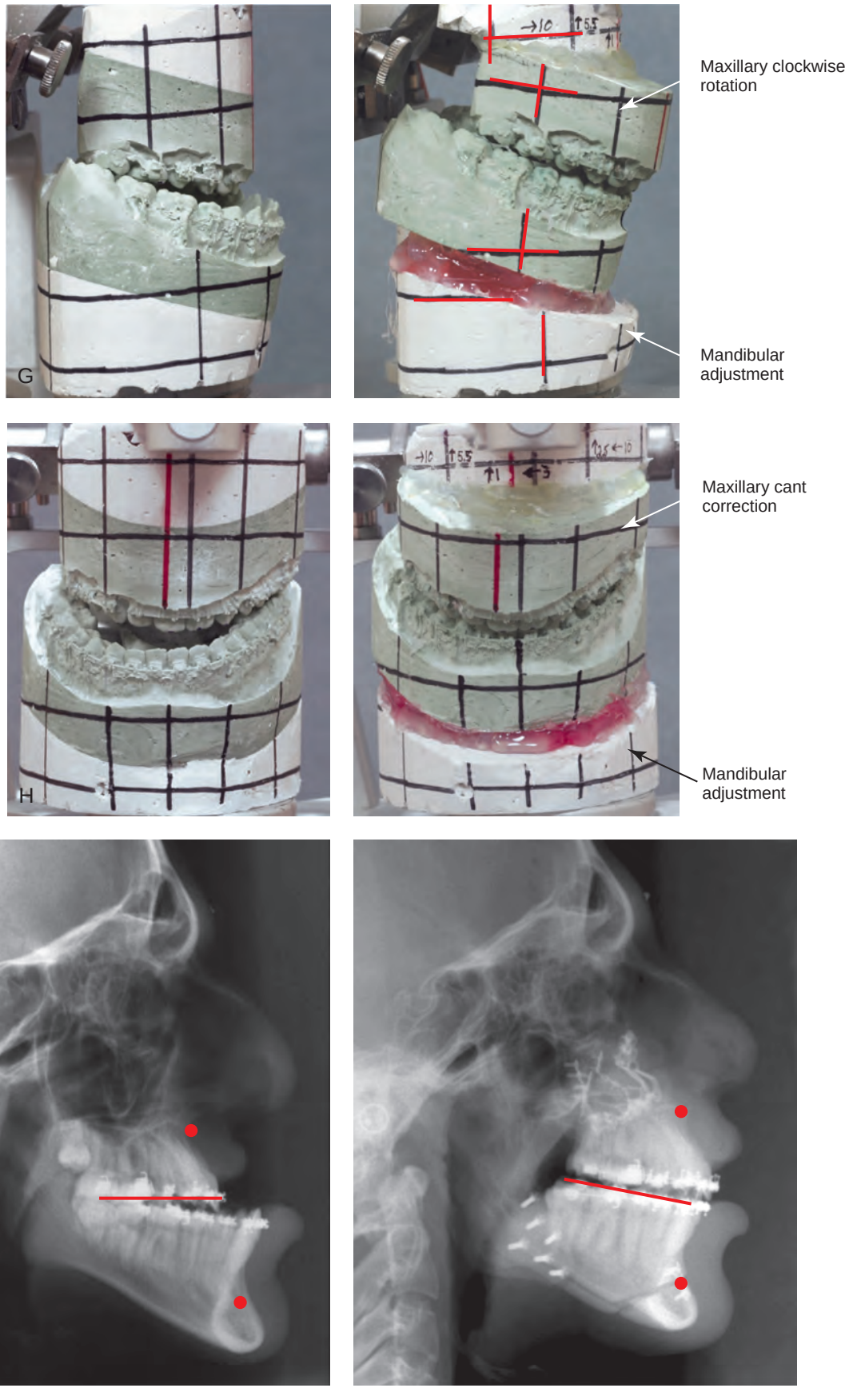
Prior to treatment with successful mixed dentition graft

Pre surgery

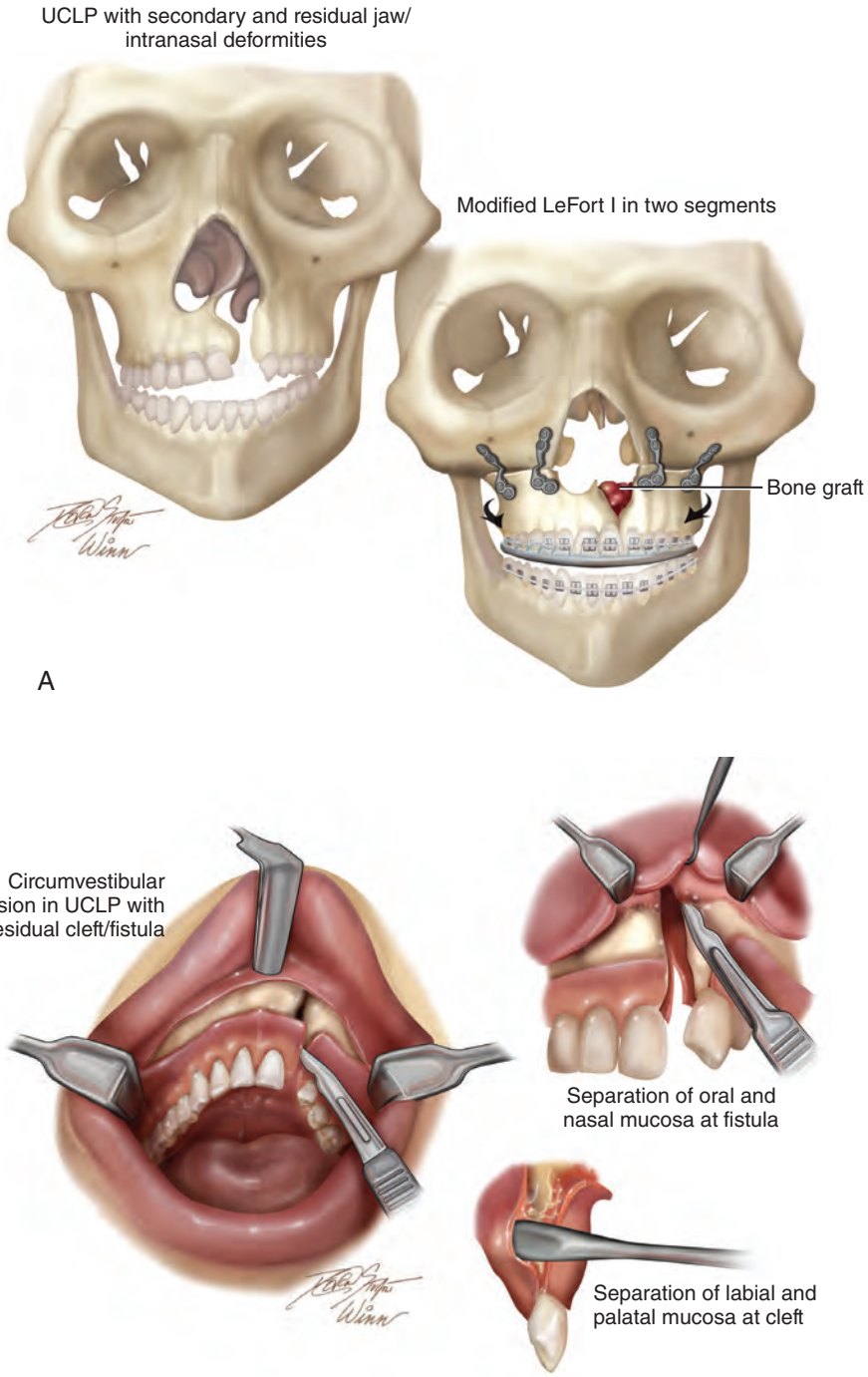
After treatment

• **Figure 32-9, cont'd F,** Occlusal views before definitive orthodontics, with orthodontic dental decompensation in progress, and after reconstruction.

Continued

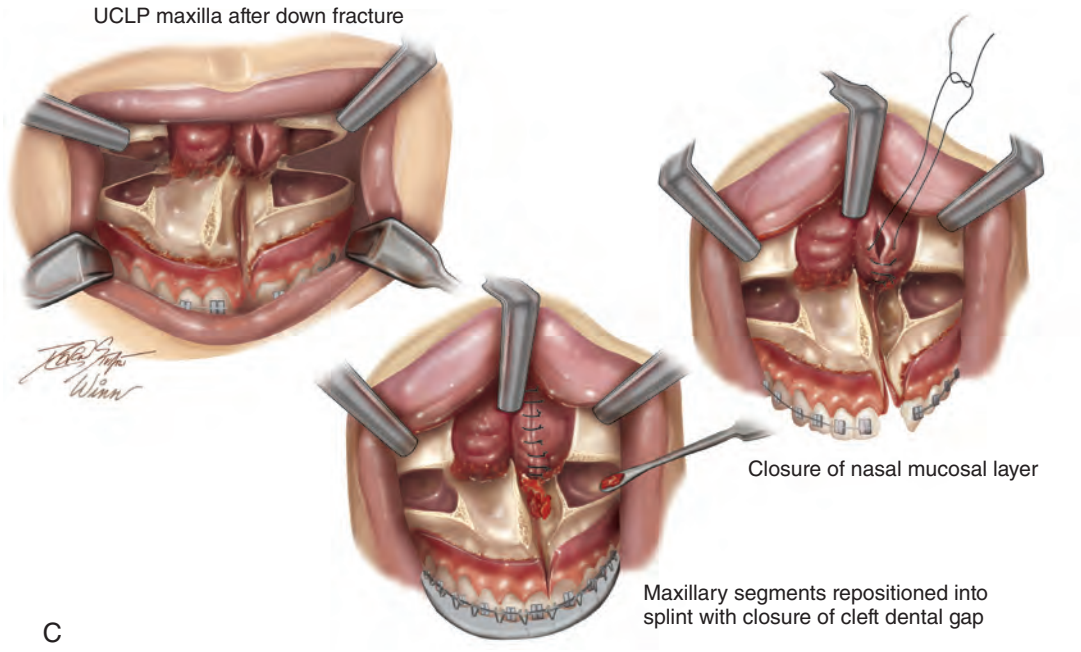


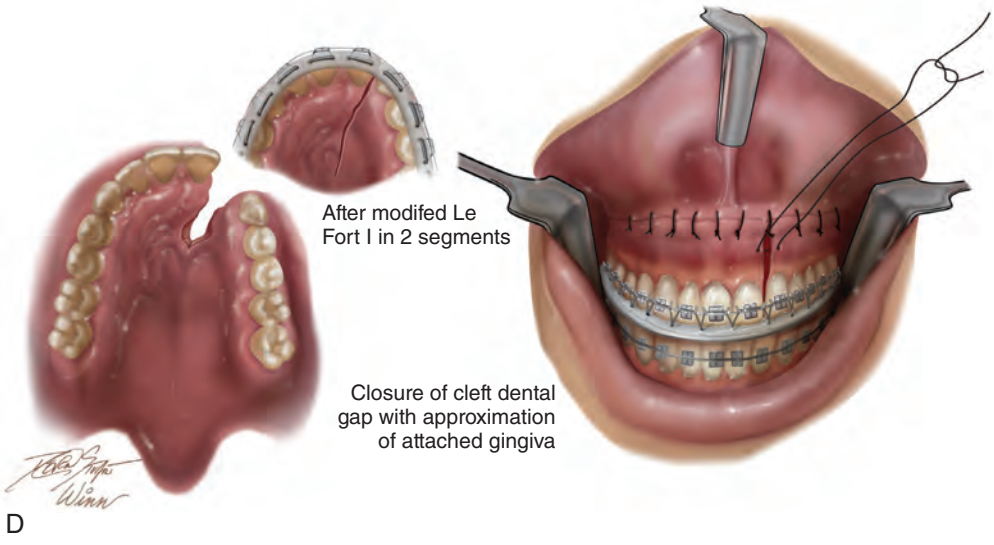
• **Figure 32-9, cont'd** **G** & **H,** Articulated dental casts that indicate analytic model planning. Note that, with the clockwise rotation of the mandible, the pogonion moves posterior while the incisors do not. **I,** Lateral cephalometric radiographs before and after reconstruction.

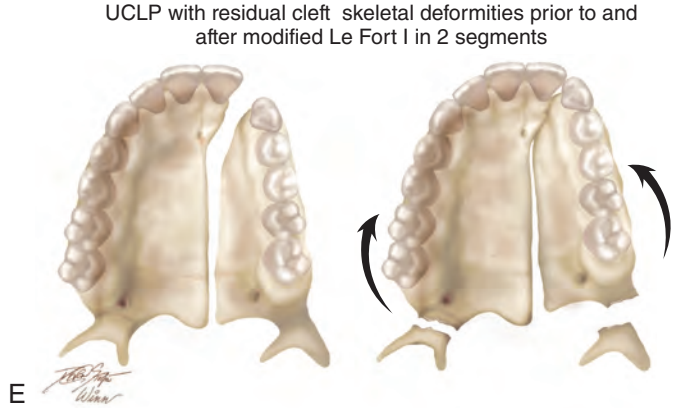


• **Figure 32-10** Illustrations of modified Le Fort I osteotomy in two segments as carried out in a patient with UCLP who did not undergo successful grafting during the mixed dentition and who presents as an adult with a jaw deformity. **A,** Frontal view of maxillofacial skeleton before and after Le Fort I osteotomy in two segments. The inferior turbinates have been reduced, and a submucous resection of the deviated septum has been performed. The nasal floor and the nasal spine have been recontoured with a rotary drill. Cancellous iliac bone graft has also been placed along the cleft nasal floor. Corticocancellous iliac graft will also be placed in gaps along the anterior maxilla on each side. **B,** Circumvestibular and perifistular incisions for exposure to complete osteotomies for down-fracture and later fistula closure.

Continued





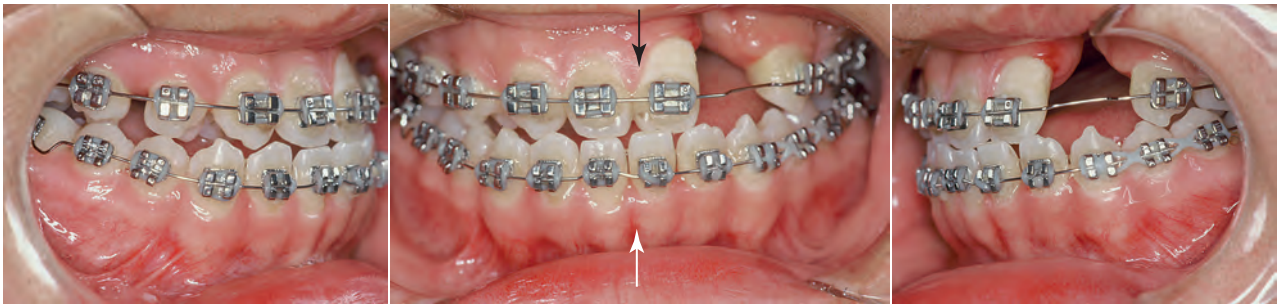


• **Figure 32-10, cont'd C,** Down-fractured Le Fort I osteotomy in two segments after the submucosal resection of the septum, the reduction of the inferior turbinates through the nasal mucosa opening, and then watertight nasal-side closure. **D,** Wound closure of both the labial and palatal aspects after differential segmental repositioning. **E,** Palatal view of bony segments before and after repositioning. *Parts A-E modified from an original illustration by Bill Winn.*

©Elsevier Inc. Orthognathic Surgery; J.C. Posnick

• **Figure 32-11** A 17-year-old girl who was born with UCLP. She underwent lip and palate repair during childhood, but she did not undergo effective bone grafting during the mixed dentition. A useful lateral incisor is not present at the cleft site. She was referred to this surgeon and underwent a comprehensive orthodontic and orthognathic surgical approach. The patient's procedures included a modified Le Fort I osteotomy in two segments (differential repositioning of the segments) with interpositional grafting as well as closure of the oronasal fistula, the cleft–dental gap, and the alveolar defect; an osseous genioplasty (vertical reduction and horizontal advancement); and septoplasty, inferior turbinate reduction, nasal floor recontouring. **A,** Frontal views with smile before and after reconstruction. **B,** Oblique views before and after reconstruction. *Continued*

• **Figure 32-11, cont'd C,** Profile views before and after reconstruction. **D,** Worm's-eye views before and after reconstruction.

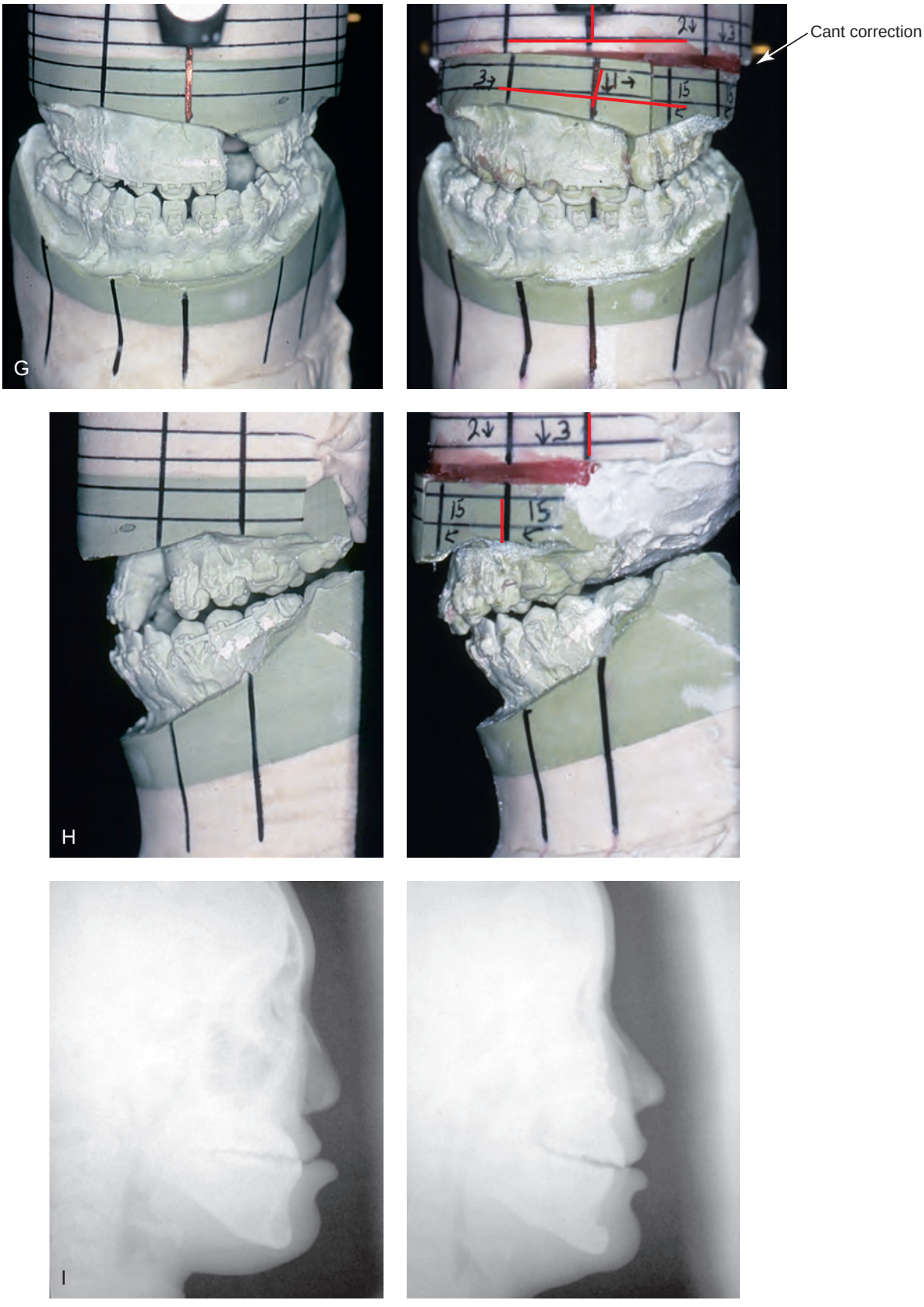


Pre surgery with residual cleft defects

After treatment

• **Figure 32-11, cont'd E,** Occlusal views with orthodontics in progress and after reconstruction. **F,** Palatal views with orthodontics in progress and after reconstruction. Note: The maxillary dentition is reconstructed with only eleven teeth. This provides a low maintenance dentition for the patient going forward.

Continued

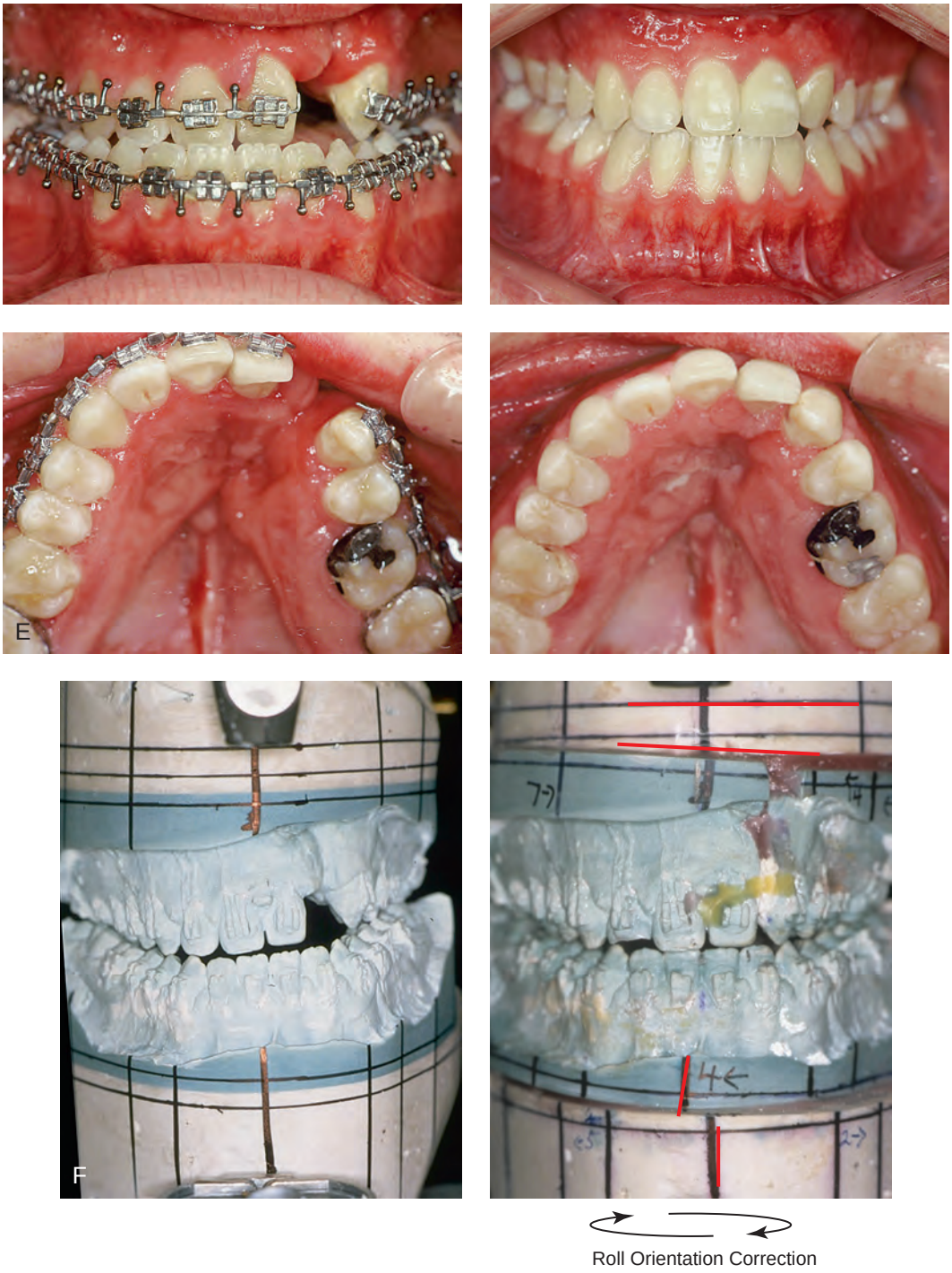


• **Figure 32-11, cont'd** **G** and **H,** Views of articulated dental casts that indicate analytic model planning. **I,** Lateral cephalometric radiographs before and after reconstruction. *A, E (top Center), E (bottom center), F, From Posnick JC, Tompson B: Modification of the maxillary Le Fort I osteotomy in cleft–orthognathic surgery: the unilateral cleft lip and palate deformity,* J Oral Maxillofac Surg *50:666-675, 1992.*

• **Figure 32-12** A 19-year-old man who was born with UCLP. He underwent lip and palate repair during childhood, but he did not undergo effective bone grafting during the mixed dentition. A useful lateral incisor is not present at the cleft site. He was referred to this surgeon and underwent a combined orthodontic and orthognathic surgical approach. The patient's procedures included a modified Le Fort I osteotomy in two segments (differential repositioning of the segments) with interpositional grafting, the correction of occlusal canting, and the closure of the oronasal fistula, the alveolar defect, and the cleft–dental gap; bilateral sagittal ramus osteotomies (correction of asymmetry); osseous genioplasty (vertical reduction and horizontal advancement); and septoplasty, inferior turbinate reduction, and nasal floor recontouring. **A,** Frontal views in repose before and after reconstruction. **B,** Frontal views with smile before and after reconstruction.

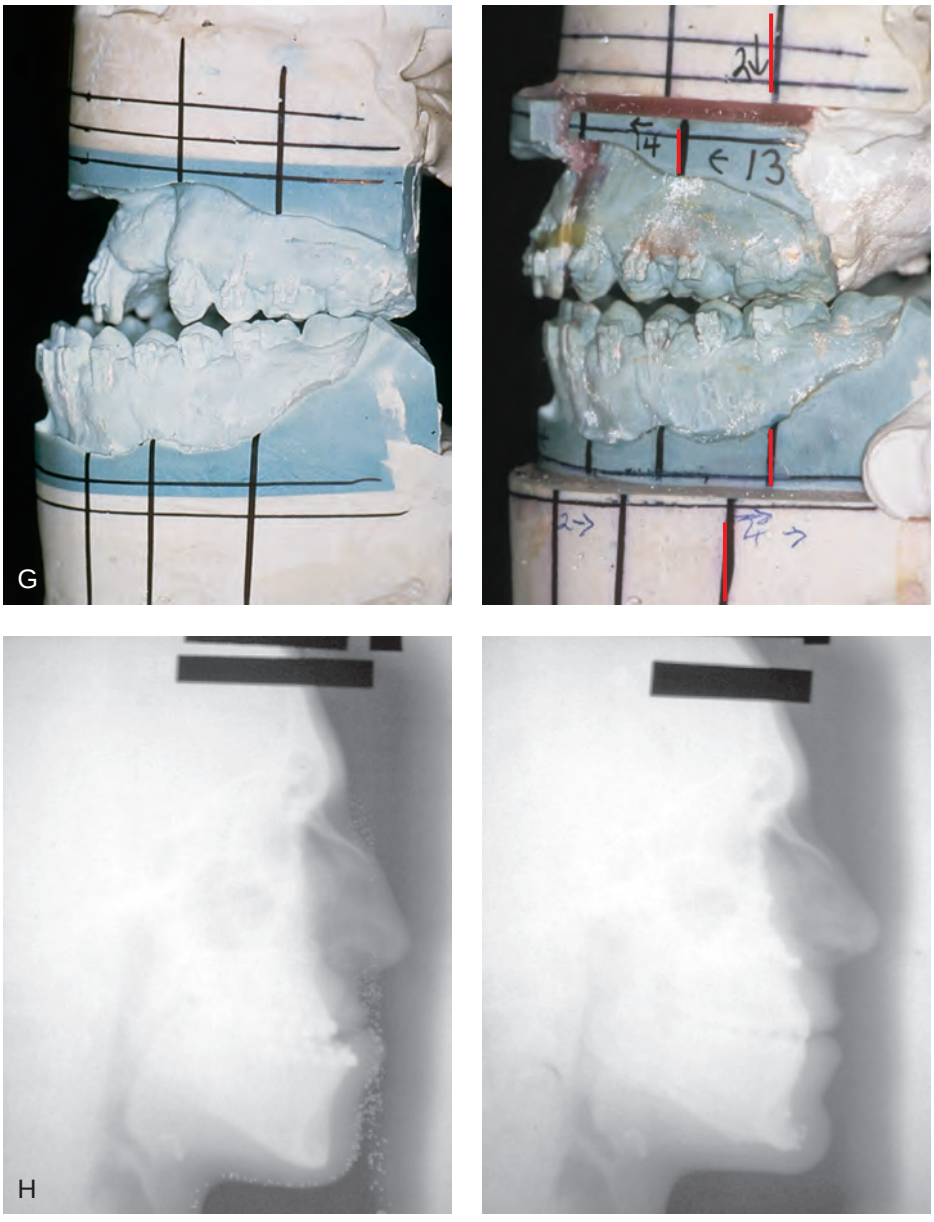
Continued

• **Figure 32-12, cont'd C,** Oblique views before and after reconstruction. **D,** Profile views before and after reconstruction.

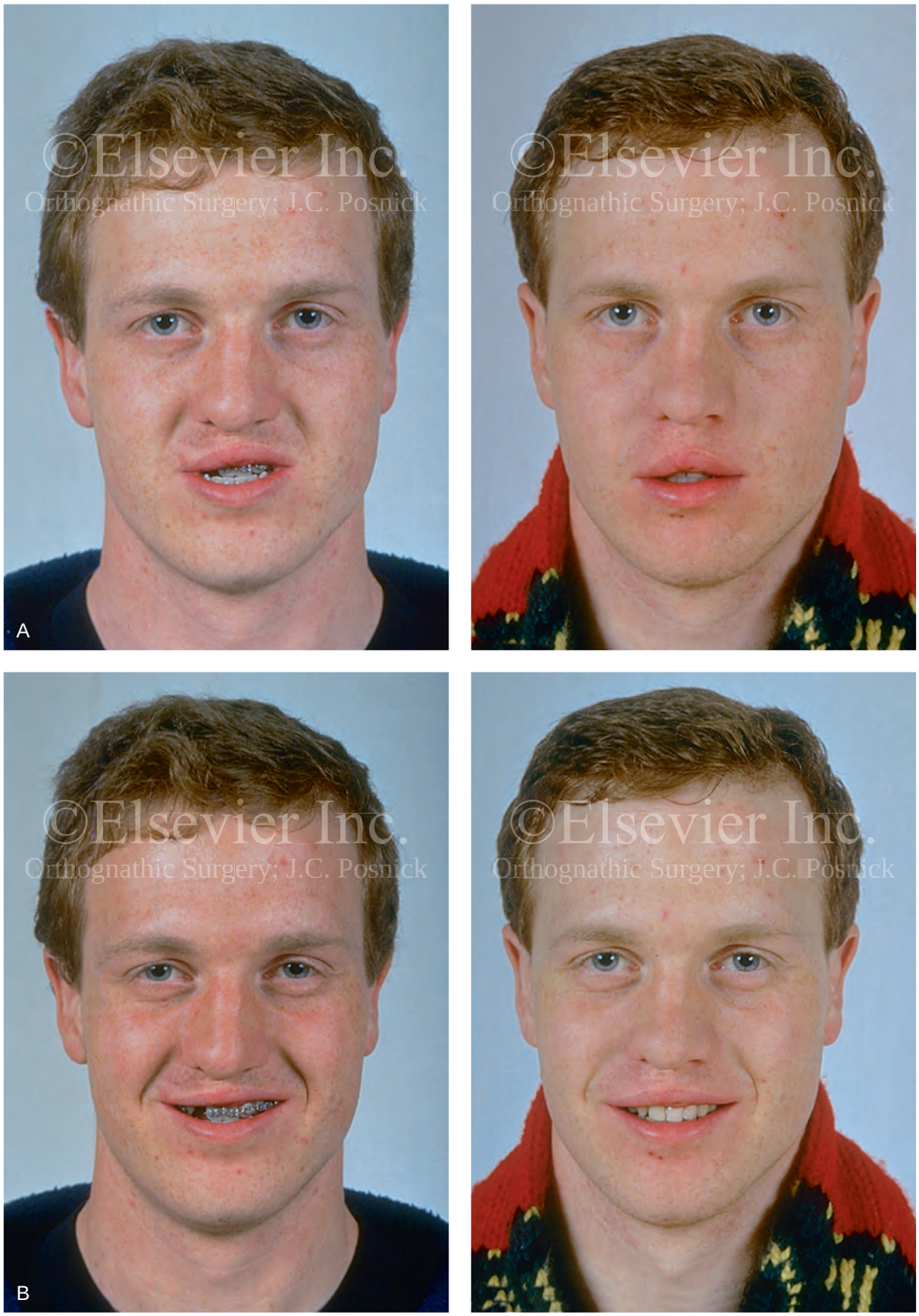


• **Figure 32-12, cont'd E,** Occlusal and palatal views with orthodontics in progress and after reconstruction. **F** and **G,** Articulated dental casts that indicate analytic model planning.

Continued



• **Figure 32-12, cont'd H,** Lateral cephalometric radiographs before and after reconstruction. *B, D, E, From Posnick JC, Tompson B: Modification of the maxillary Le Fort I osteotomy in cleft–orthognathic surgery: the unilateral cleft lip and palate deformity,* J Oral Maxillofac Surg *50:666-675, 1992.*



©Elsevier Inc.
Orthognathic Surgery; J.C. Posnick

• **Figure 32-13** A 17-year-old boy who was born with UCLP. He underwent lip and palate repair during childhood, but he did not undergo effective bone grafting during the mixed dentition. The lateral incisor is not present at the cleft site. He was referred to this surgeon as a teenager and underwent a combined orthodontic and orthognathic surgical approach. The patient's procedures included a modified Le Fort I osteotomy in two segments (differential repositioning of the segments) with interpositional grafting and closure of the oronasal fistula, the alveolar defect, and the cleft–dental gap; and septoplasty, inferior turbinate reduction, and nasal floor recontouring. **A,** Frontal views in repose before and after reconstruction. **B,** Frontal views with smile before and after reconstruction.

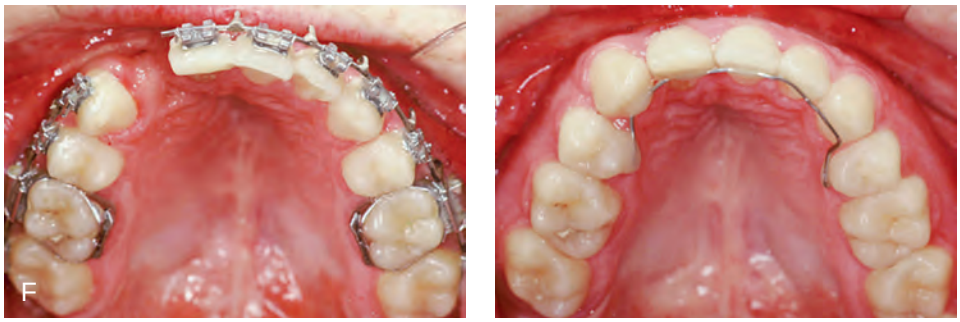
Continued

©Elsevier Inc.
Orthognathic Surgery; J.C. Posnick

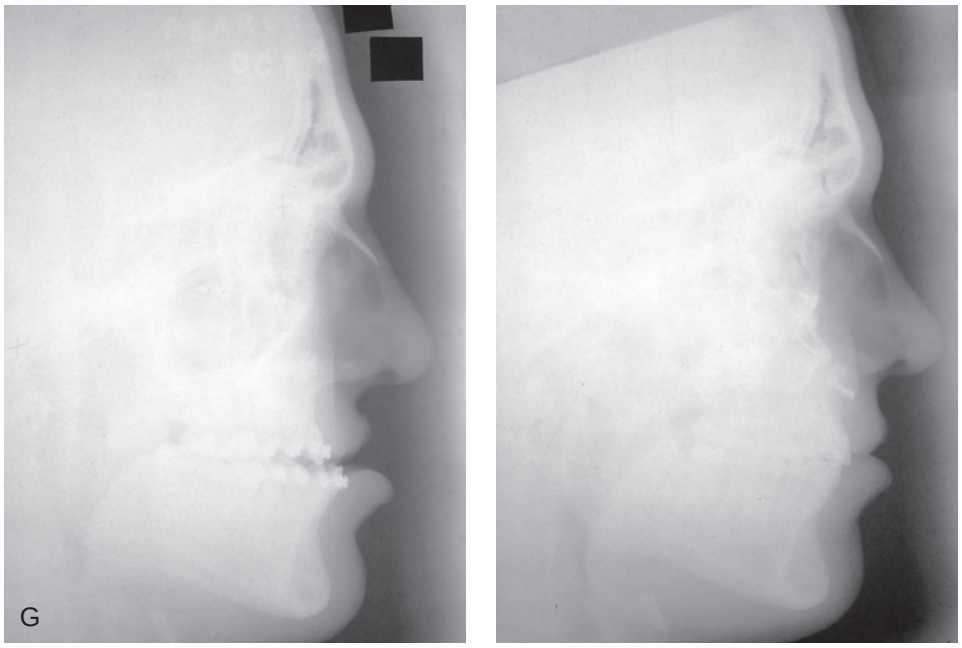
• **Figure 32-13, cont'd C,** Oblique views before and after reconstruction. **D,** Profile views before and after reconstruction.

Pre surgery with residual cleft defects

After treatment



Before and After Treatment



• **Figure 32-13, cont'd** **E,** Occlusal views with orthodontics in progress and after reconstruction. **F,** Palatal views with orthodontics in progress and after reconstruction. **G,** Lateral cephalometric radiographs before and after reconstruction. *B, D, E, F, From Posnick JC, Dagys AP: Skeletal stability and relapse patterns after Le Fort I maxillary osteotomy fixed with miniplates: the unilateral cleft lip and palate deformity,* Plast Reconstr Surg *94:924-932, 1994.*

• **Figure 32-14** A 20-year-old man who was born with UCLP on the left side. He underwent lip and palate repair during childhood, but he did not undergo effective bone grafting during the mixed dentition. A useful lateral incisor is not present at the cleft site. He has a retained primary molar and an absent lateral incisor on the non-cleft side. He was referred to this surgeon as a young adult and underwent a combined orthodontic and orthognathic surgical approach. The patient's procedures included a modified Le Fort I osteotomy in two segments (differential repositioning of the segments) with interpositional grafting and closure of the oronasal fistula, the alveolar defect, and the cleft–dental gap; and septoplasty, inferior turbinate reduction, and nasal floor recontouring. **A,** Frontal views with smile before and after reconstruction. **B,** Oblique views before and after reconstruction. **C,** Profile views before and after reconstruction.

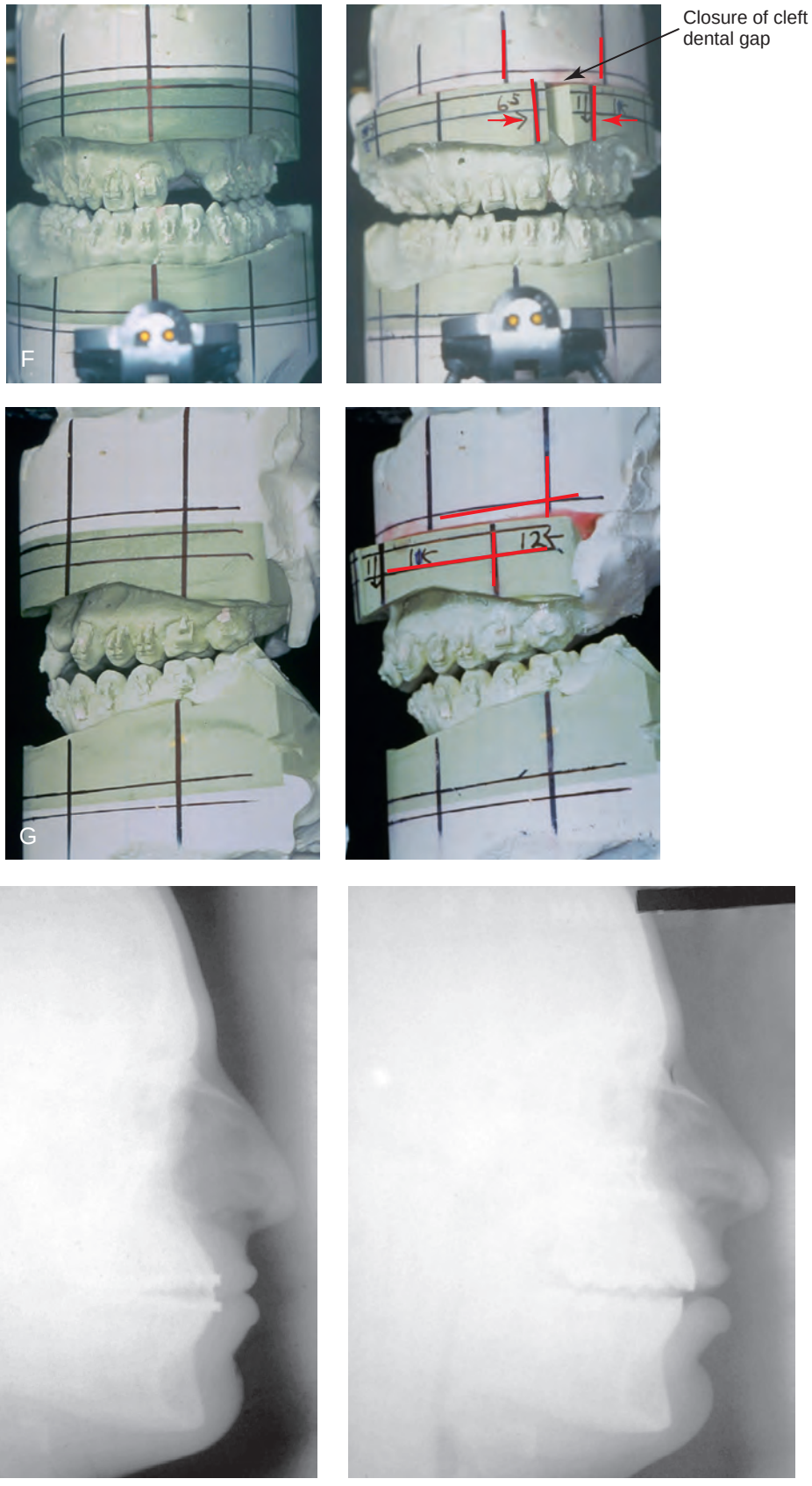
Pre surgery with residual cleft defects

After treatment

Before and After Treatment

• **Figure 32-14, cont'd D,** Occlusal views with orthodontics in progress and after reconstruction. **E,** Palatal views with orthodontics in progress and after reconstruction.

Continued



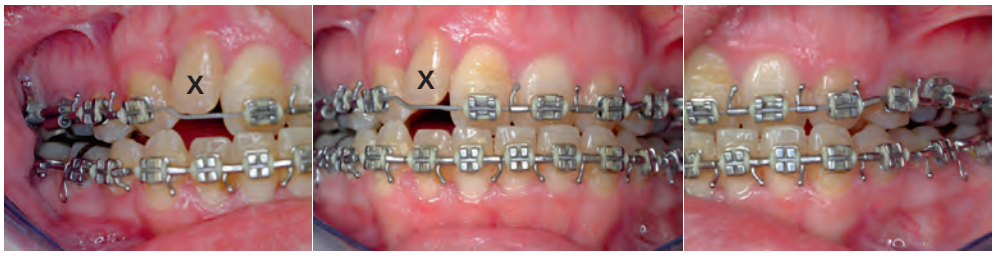
• **Figure 32-14, cont'd** **F** and **G,** Articulated dental casts that indicate analytic model planning. **H,** Lateral cephalometric radiographs before and after reconstruction. *A, C, D, F, From Posnick JC, Dagys AP: Skeletal stability and relapse patterns after Le Fort I maxillary osteotomy fixed with miniplates: the unilateral cleft lip and palate deformity,* Plast Reconstr Surg *94:924-932, 1994.*

©Elsevier Inc.
Orthognathic Surgery; J.C. Posnick

• **Figure 32-15** A 24-year-old schoolteacher who was born with UCLP. She underwent 4 bicuspid extractions as part of earlier orthodontic treatment. The retained lateral incisor at the cleft does not have an adequate root and required extraction. She was referred to this surgeon as an adult and underwent a combined orthodontic and orthognathic surgical approach. The patient's procedures included a modified Le Fort I osteotomy in two segments (differential repositioning of the segments) and closure of the oronasal fistula, the alveolar cleft, and the cleft–dental gap; bilateral sagittal split ramus osteotomies; osseous genioplasty; and septoplasty, inferior turbinate reduction, and nasal floor recontouring. **A,** Frontal views in repose before and after reconstruction. **B,** Frontal views with smile before and after reconstruction.

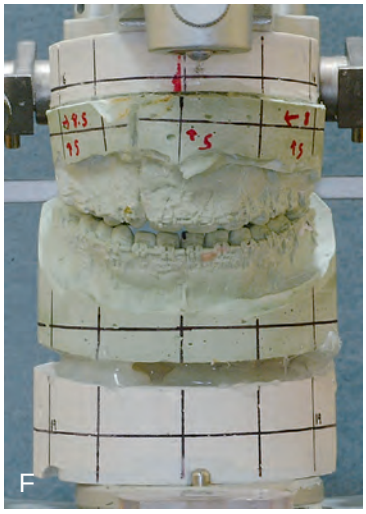
Continued

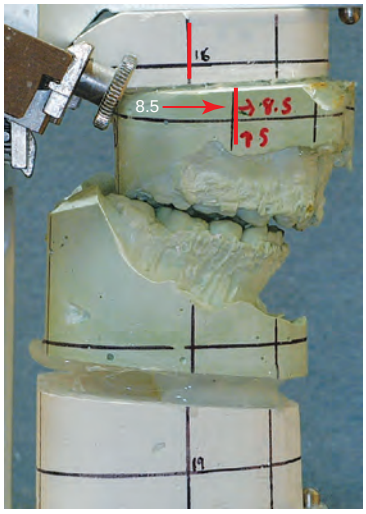
• **Figure 32-15, cont'd C,** Oblique views before and after reconstruction. **D,** Profile views before and after reconstruction.



Pre surgery with residual cleft defects

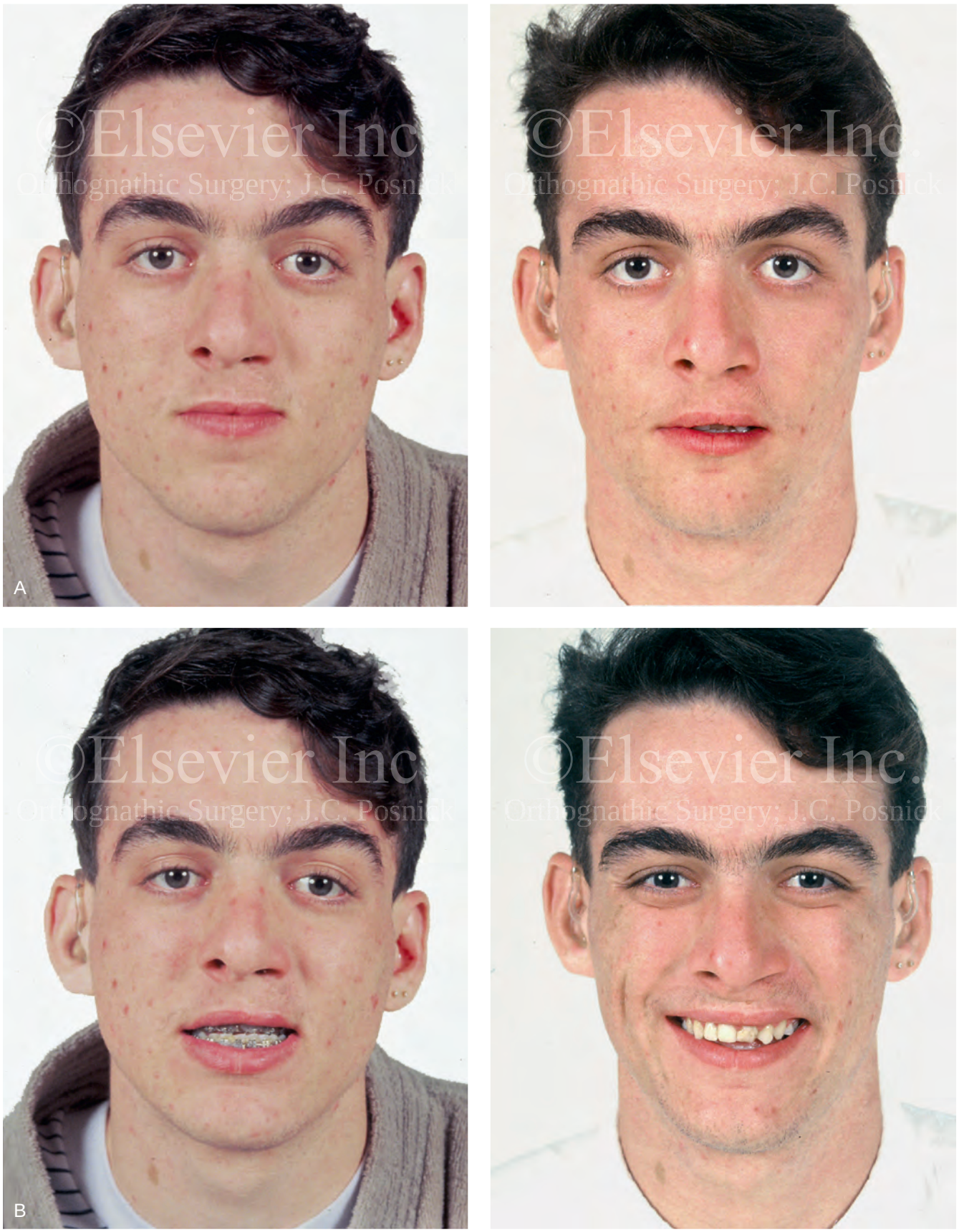
After treatment





Closure of cleft dental gap

• **Figure 32-15, cont'd E,** Occlusal views with orthodontics in progress and after reconstruction and dental rehabilitation. **F,** Articulated dental casts that indicate analytic model planning. **G,** Lateral cephalometric radiographs before and after reconstruction.



©Elsevier Inc. Orthognathic Surgery; J.C. Posnick

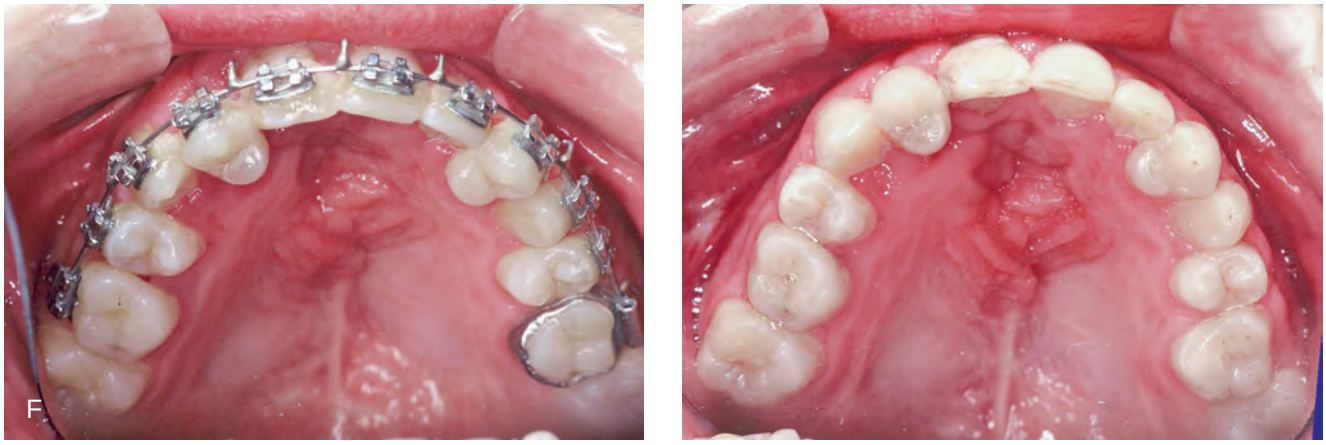
• **Figure 32-16** A 17-year-old boy who was born with UCLP. He was referred to this surgeon during the late mixed dentition and underwent effective bone grafting. He was congenitally missing his lateral incisor at the cleft site. There is transposition of the canine and the first bicuspids on each side of the upper jaw. He underwent a combined orthodontic and orthognathic surgical approach. The patient's procedures included a standard Le Fort I osteotomy (horizontal advancement) with interpositional grafting; osseous genioplasty (vertical reduction and horizontal advancement); and septoplasty, inferior turbinate reduction, and nasal floor recontouring. **A,** Frontal views in repose before and after reconstruction. **B,** Frontal views with smile before and after reconstruction.

• **Figure 32-16, cont'd C,** Oblique views before and after reconstruction. **D,** Profile views before and after reconstruction.

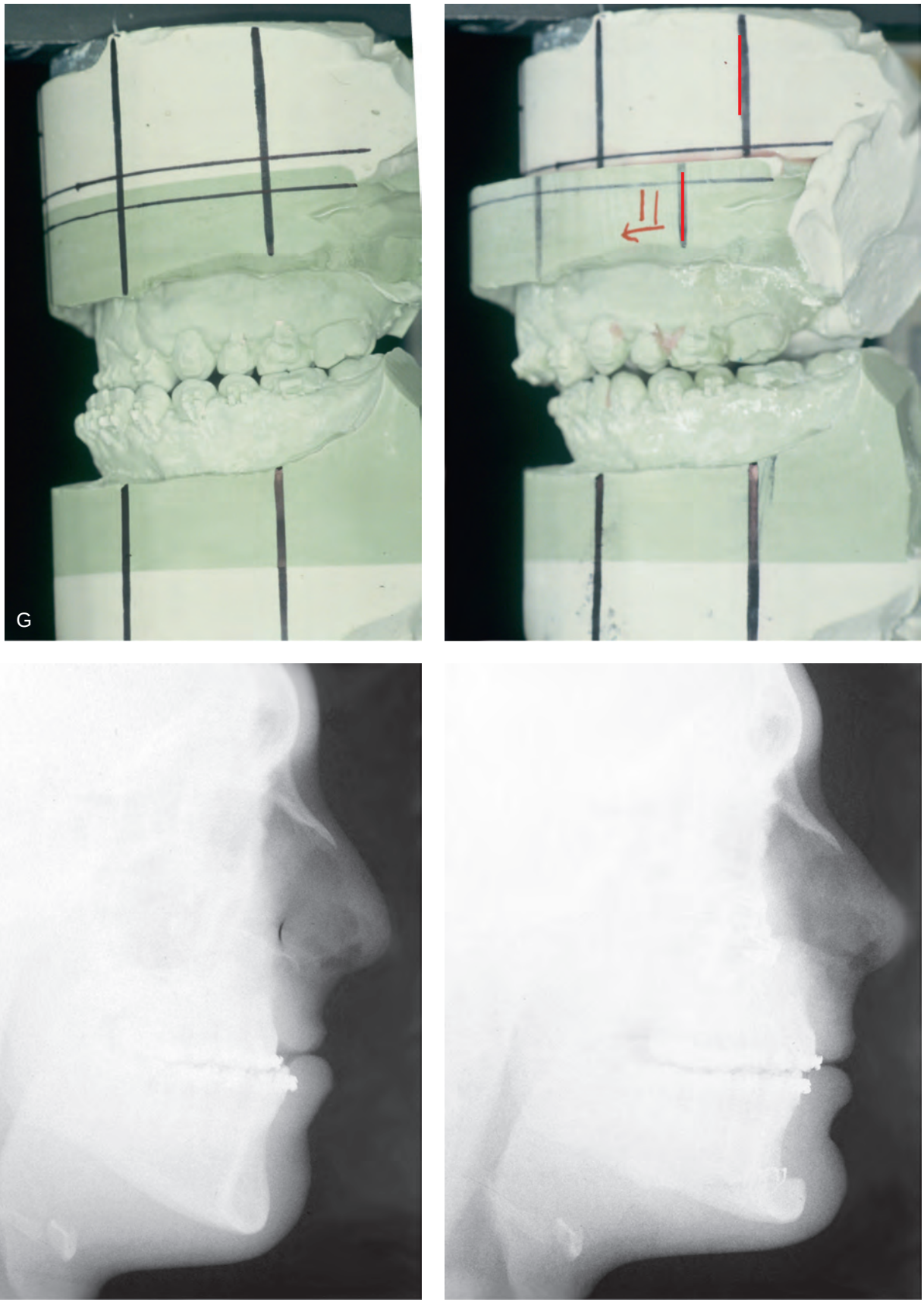
Continued

Pre surgery

After treatment



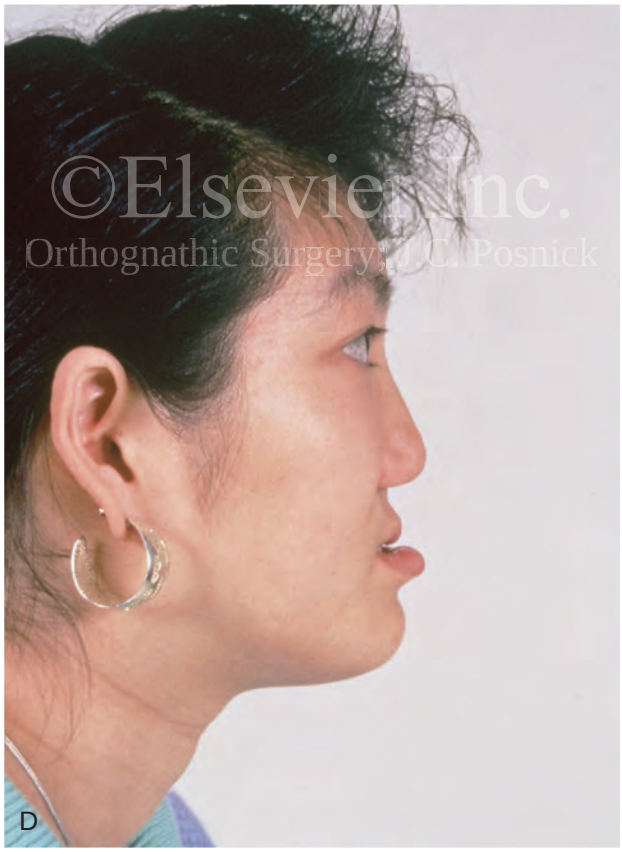
• **Figure 32-16, cont'd E,** Occlusal views with orthodontics in progress and after reconstruction. **F,** Palatal views with orthodontics in progress and after reconstruction.



• **Figure 32-16, cont'd G,** Articulated dental casts that indicate analytic model planning. **H,** Lateral cephalometric radiographs before and after reconstruction. Note that cosmetic recontouring and modification of the maxillary dental crowns would be beneficial.

©Elsevier Inc.
Orthognathic Surgery; J.C. Posnick

• **Figure 32-17** A 16-year-old girl who was born with UCLP. She was referred to this surgeon and underwent a combined orthodontic and orthognathic surgical approach. The patient's procedures included a modified Le Fort I osteotomy in two segments (differential repositioning of the segments) and the closure of the oronasal fistula, the alveolar defect, and the cleft–dental gap; and septoplasty, inferior turbinate reduction, and nasal floor recontouring. **A** and **B,** Frontal views in repose before and after reconstruction. **B,** Frontal views with smile before and after reconstruction.

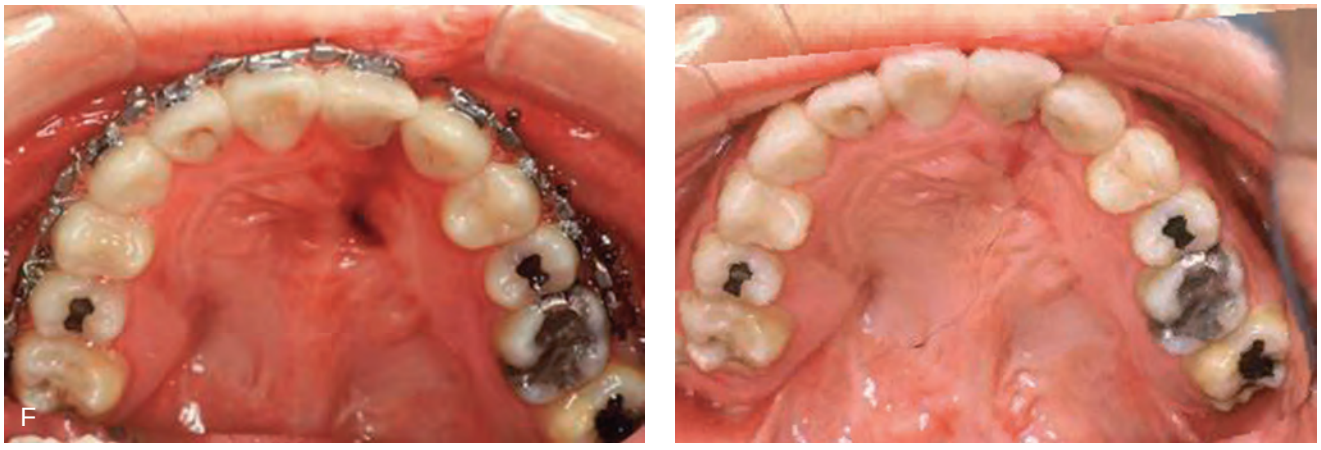


• **Figure 32-17, cont'd** **C** and **D,** Oblique facial views before and after reconstruction. **D,** Profile views before and after reconstruction.

Continued

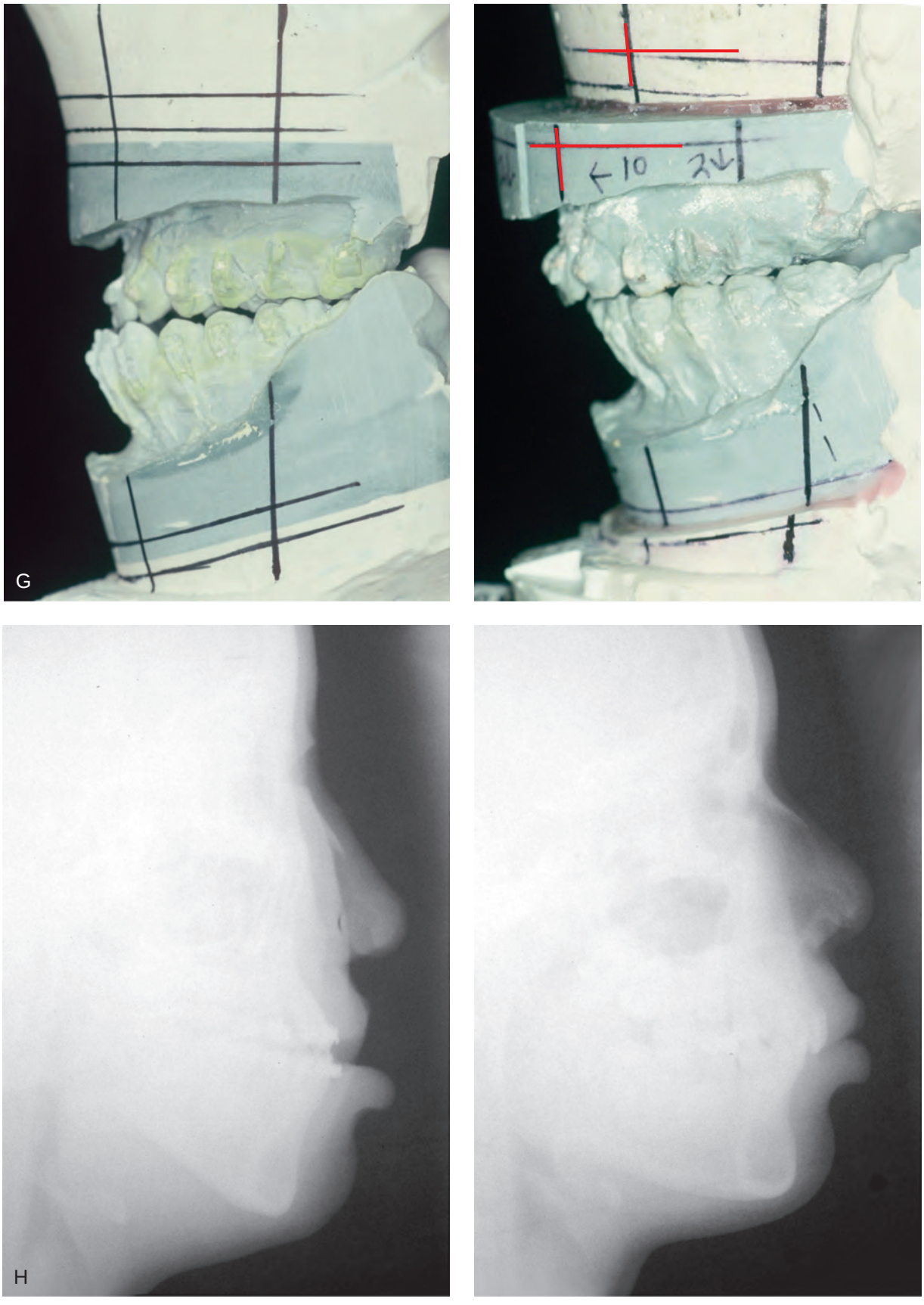
Prior to surgery with residual cleft defects

After treatment



Prior to and after treatment

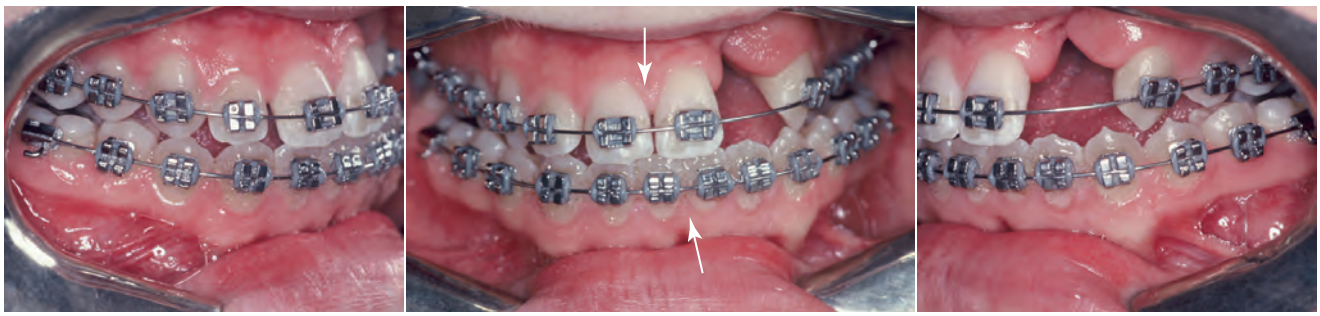
• **Figure 32-17, cont'd E,** Occlusal views with orthodontics in progress and after reconstruction. **F,** Palatal views with orthodontics in progress and after reconstruction.



• **Figure 32-17, cont'd G,** Articulated dental casts that indicate analytic model planning. **H,** Lateral cephalometric radiographs before and after reconstruction. *From Posnick JC: Orthognathic surgery in the cleft patient. In Russell RC, ed:* Instructional courses, Plastic Surgery Educational Foundation, *vol 4, St. Louis, Mo, 1991, Mosby–Year Book, p 129.*

©Elsevier Inc.
Orthognathic Surgery; J.C. Posnick

• **Figure 32-18** A 24-year-old woman who was born with UCLP. She did not undergo bone grafts in the mixed dentition. She is missing the lateral incisor on the cleft side. She was referred to this surgeon as an adult and underwent a combined orthodontic and orthognathic surgical approach. The patient's procedures included a modified Le Fort I osteotomy in two segments (differential repositioning of the segments) and closure of oronasal fistula, the alveolar cleft, and the cleft–dental gap; bilateral sagittal split ramus osteotomies; osseous genioplasty; and septoplasty, inferior turbinate reduction, and nasal floor recontouring. **A,** Frontal views with smile before and after reconstruction. **B,** Profile views before and after reconstruction.



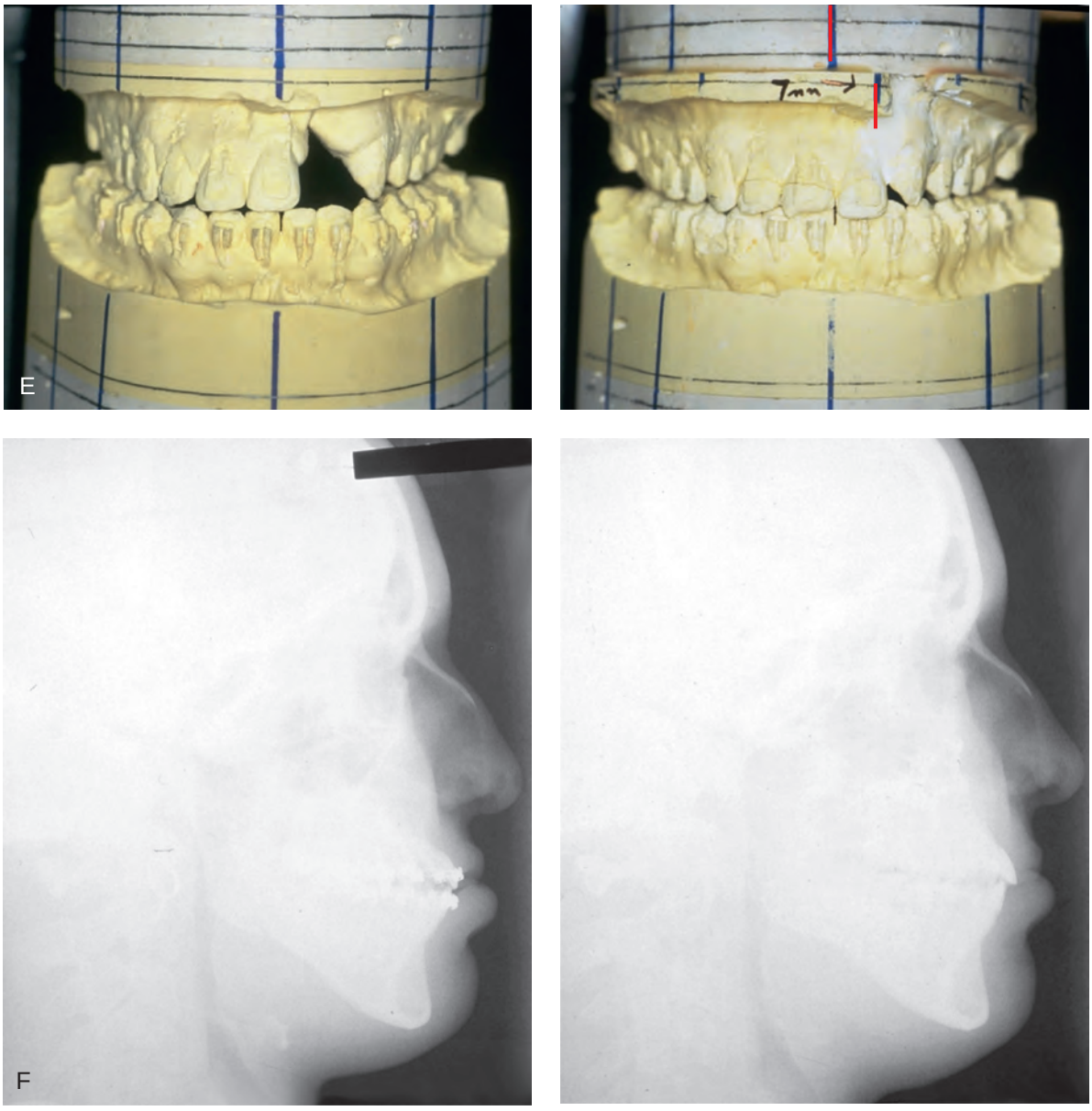
Prior to surgery with residual cleft defects

After treatment

Before and After Treatment

• **Figure 32-18, cont'd C,** Occlusal views with orthodontics in progress and after reconstruction and dental rehabilitation. **D,** Palatal views before and after reconstruction.

Continued

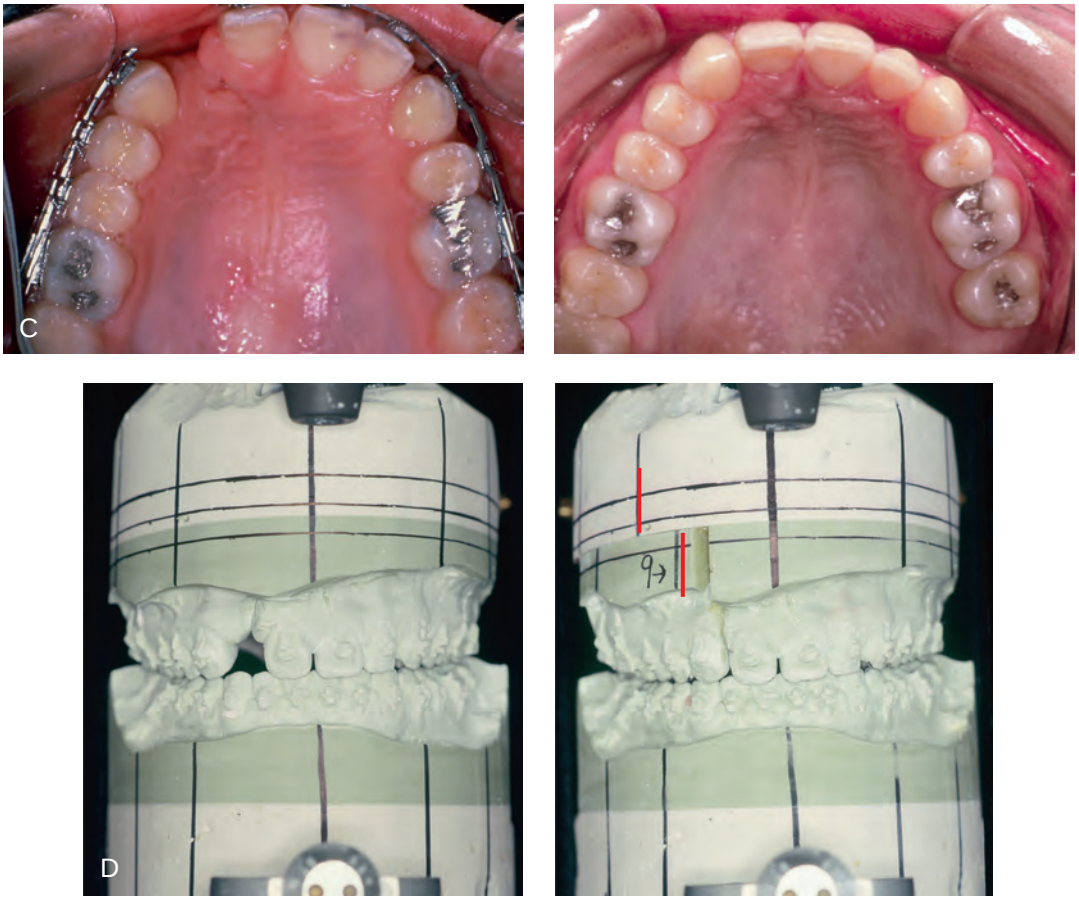


• **Figure 32-18, cont'd E,** Articulated dental casts that indicate analytic model planning. **F,** Lateral cephalometric radiographs before and after reconstruction. Note that the palatal fistula was successfully closed. Cosmetic modification of the maxillary anterior teeth was recommended.

Prior to surgery with residual cleft defects

After treatment

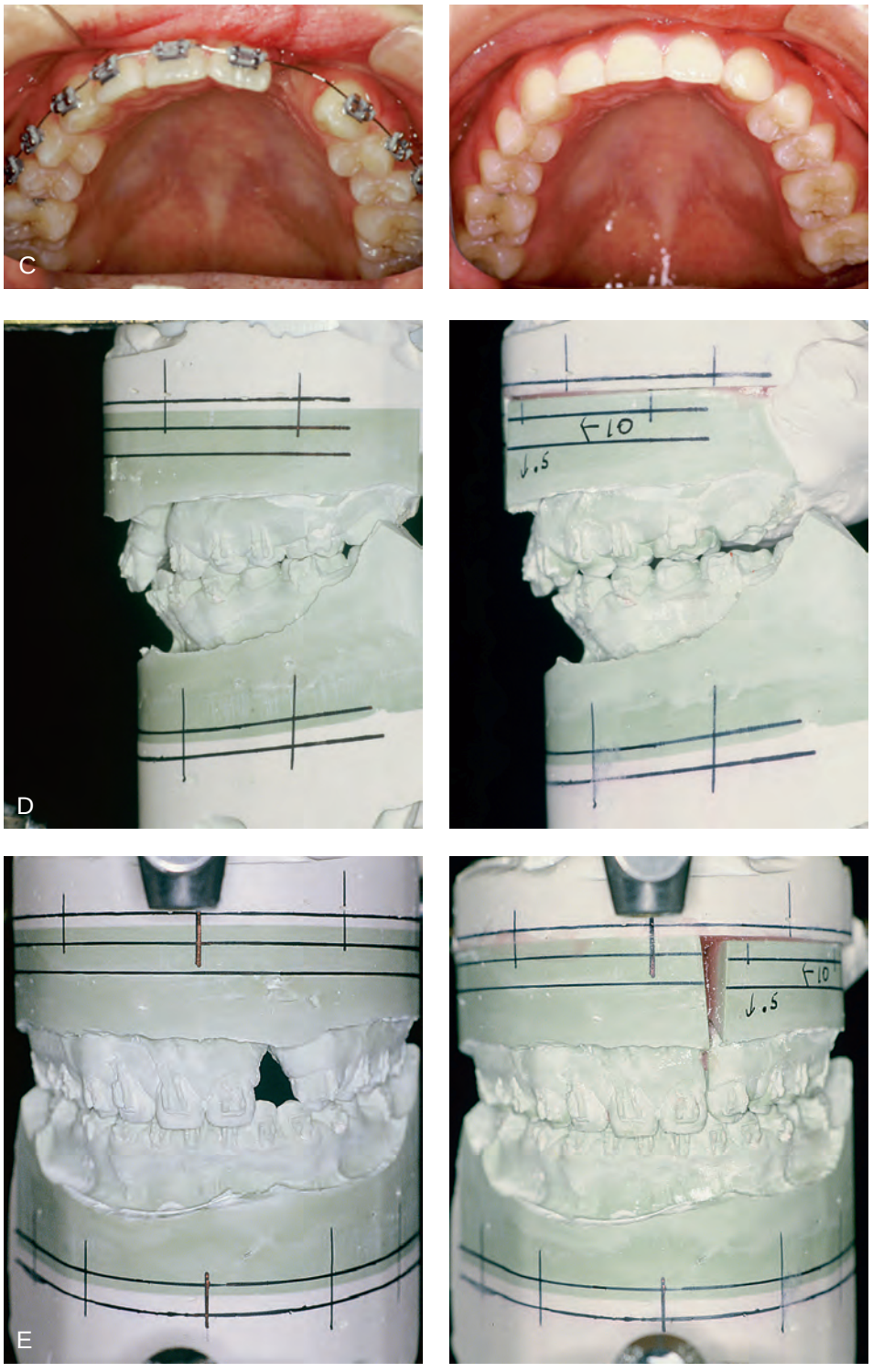
• **Figure 32-19** A 17-year-old girl who was born with UCLP. She did not undergo successful bone grafting in the mixed dentition. She is missing the lateral incisor on the cleft side. The first bicuspid was extracted on the non-cleft side. She was referred to this surgeon as a teenager and underwent a combined orthodontic and orthognathic surgical approach. The maxilla had good horizontal projection and vertical height. Of the options available to manage the residual upper dentoalveolar needs, a right posterior segmental osteotomy was completed to close the oronasal fistula, the alveolar defect, and the cleft–dental gap. **A,** Frontal views with smile before and after reconstruction. **B,** Occlusal views with orthodontics in progress and after reconstruction. *Continued*



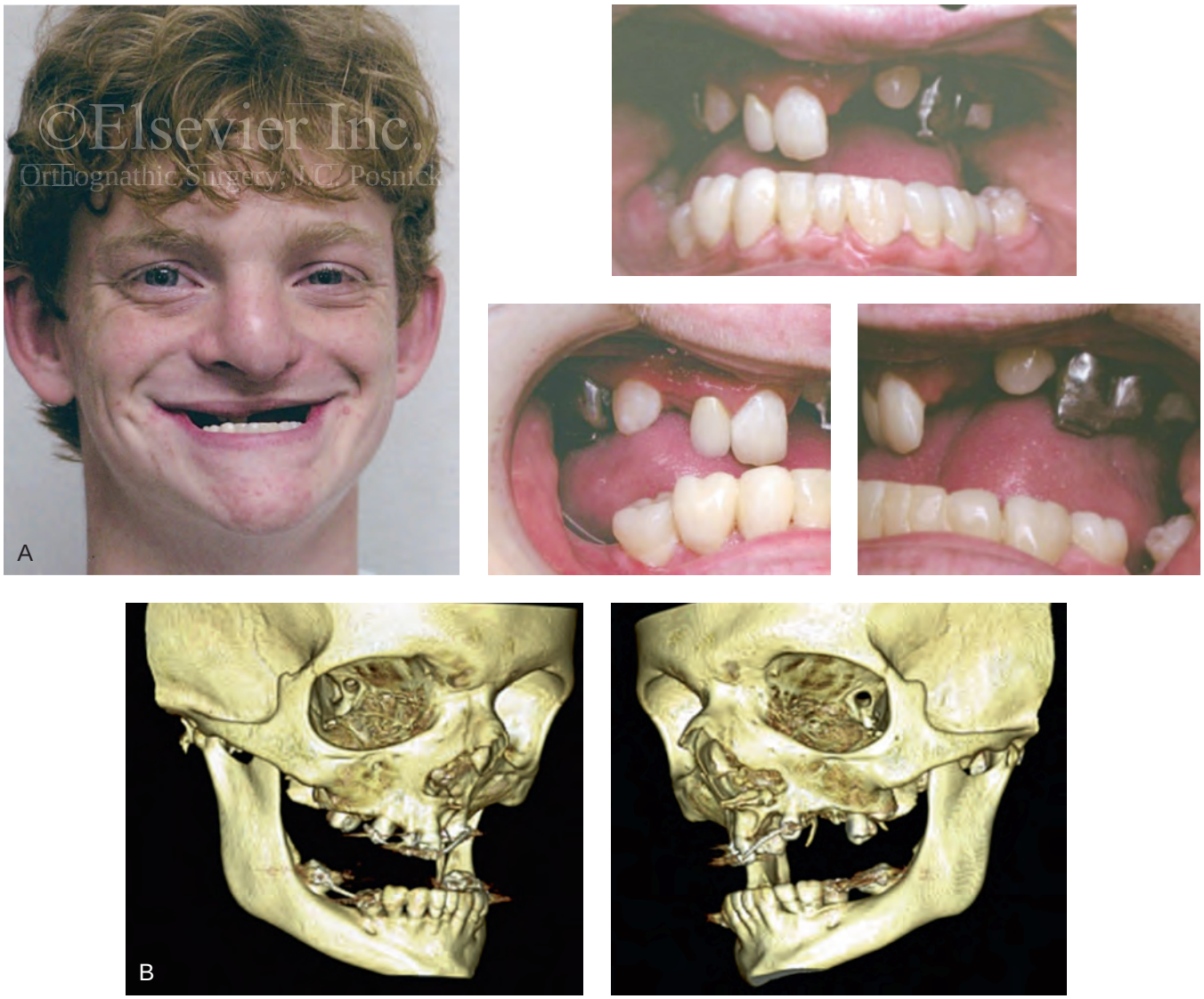
• **Figure 32-19, cont'd C,** Palatal views with orthodontics in progress and after reconstruction. **D,** Articulated dental casts that indicate analytic model planning.

Prior to surgery with residual cleft defects

• **Figure 32-20** A 17-year-old girl who was born with UCLP. She underwent lip and palate repair during childhood, but she did not undergo effective bone grafting during the mixed dentition. The lateral incisor is not present at the cleft site. She was referred to this surgeon and underwent a combined orthodontic and orthognathic surgical approach. The maxilla had good horizontal projection and vertical height. Of the options available to manage the residual upper dentoalveolar needs, a right posterior segmental osteotomy was completed to close the oronasal fistula, the alveolar defect, and the cleft–dental gap. **A,** Frontal views with smile before and after reconstruction. **B,** Occlusal views with orthodontics in progress and after reconstruction. *Continued*



• **Figure 32-20, cont'd C,** Palatal views with orthodontics in progress and after reconstruction. **D** and **E,** Articulated dental casts that indicate analytic model planning.



• **Figure 32-21** A teenage boy who was born with ectodermal dysplasia and complete UCLP on the left side. He underwent lip and palate closure during infancy. There are multiple congenital missing teeth in each arch. He was referred to this surgeon for evaluation when he was 14 years old. He retains only six long-term useful teeth in the maxilla (first and second molars) and displaced left and right canines. The maxilla is vertically and horizontally deficient. The mandible has satisfactory symmetry and horizontal projection. The patient underwent evaluations by specialists, including an orthodontist, a prosthodontist, a periodontist, a surgeon, a speech pathologist, an otolaryngologist, and a geneticist. Reconstruction and dental rehabilitation were felt to require the surgical repositioning of the maxilla followed by an overdenture. In the mandible, crown and bridge rehabilitation would be carried out. The prosthodontist requested 18 mm of horizontal advancement and 14 mm of vertical lengthening of the maxilla. A two-stage approach to maxillary reconstruction was undertaken.

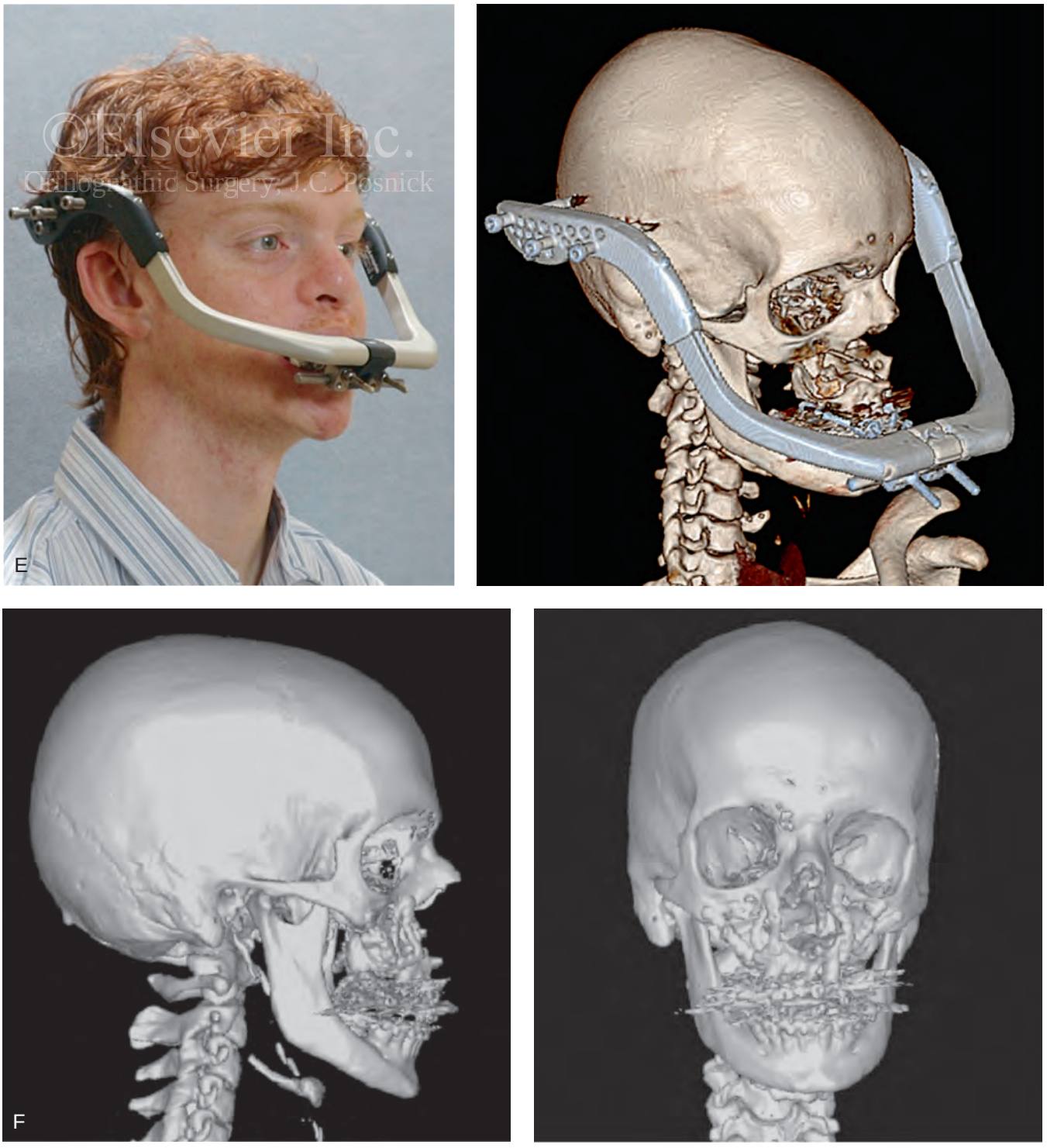
Stage I surgery included the following: 1) nasotracheal intubation; 2) Le Fort I osteotomy with down fracture and disimpaction; 3) septoplasty and inferior turbinate reduction; 4) application of a MED I external distraction device; and 5) securing of the MED I device to a prefabricated in-place chrome cobalt appliance fixed to the maxillary dentition. Successful **outpatient distraction** of the maxilla to the preferred position was accomplished over a 10-day time frame.

Stage II surgery involved the patient's return to the operating room for the following: 1) awake fiber-optic nasotracheal intubation; 2) removal of the MED I device; 3) harvesting of the anterior iliac corticocancellous graft; 4) reopening of the circumvestibular incision; 5) securing a prefabricated splint to the maxilla and then applying intermaxillary fixation (18 mm of advancement); 6) rotation of the maxillomandibular complex to achieve the desired vertical dimension (14 mm of lengthening); 7) application of plate and screw fixation to the maxilla; 8) crafting and inset of corticocancellous grafts to the left and right anterior maxilla; and 9) plate and screw fixation of each graft to the native maxilla. **After 6 weeks**, the maxilla achieved initial bone healing. The patient returned to a more regular diet and sports activities at that time. **Six months postoperatively**, the patient underwent a pharyngeal flap procedure to achieve velopharyngeal competence and an open rhinoplasty procedure that included a rib cartilage (caudal strut) graft. **Dental rehabilitation** included fixed bridgework in the mandibular arch and overdenture construction for the maxilla. **A,** Frontal and occlusal views when the patient was 14 years old. **B,** Computed tomography scan views that indicate the extent of maxillary hypoplasia.

Continued

©Elsevier Inc.
Orthognathic Surgery; J.C. Posnick

• **Figure 32-21, cont'd C,** Articulated dental casts that indicate analytic model planning. **D,** Facial and occlusal views before surgery with a prefabricated chrome–cobalt appliance fixed to the maxillary dentition.



©Elsevier Inc.
Orthognathic Surgery, J.C. Posnick

• **Figure 32-21, cont'd E,** The patient is shown after Le Fort I osteotomy followed by 10 days of MED I distraction to achieve the preferred maxillary position. A computed tomography scan is also shown 10 days after the Le Fort I procedure to demonstrate the advanced maxilla just before the removal of the MED I device. **F,** Computed tomography scan views are shown after the stage II procedures with the maxilla in its new location and now secured with bone graft and plate and screw fixation.

Continued

• **Figure 32-21, cont'd G,** Frontal facial views in repose before and after reconstruction and rehabilitation. **H,** Frontal views with smile before and after reconstruction and rehabilitation. **I,** Profile views before and after reconstruction and rehabilitation.

cleft team's best efforts, a number of children will not make themselves available for mixed-dentition grafting (i.e., before the eruption of the cleft side canine). In addition, a percentage of those who do undergo such procedures will have problems with grafting and require other means of reconstruction and dental rehabilitation. Williams and colleagues documented that, in the United Kingdom, only 84% of children with complete UCLP arrived for mixed-dentition grafting.[177] McIntyre and colleagues documented that only 75.4% of Scottish children with complete UCLP were grafted during the mixed dentition.[103] Daskalogiannakis and colleagues documented that, at The Hospital for Sick Children in Toronto, children with complete UCLP underwent mixed-dentition grafting only 56.1% of the time.[27] On a more encouraging note, Felstead and colleagues documented a 94% rate of radiographically successful mixed-dentition bone grafting in a consecutive series of children with UCLP or bilateral cleft lip and palate (BCLP) who were treated by a single surgeon (n = 53 alveolar cleft sites).[39] Despite a clinician's preferred approach to cleft management during infancy, childhood, and early adolescence and despite that clinician's best efforts, a subgroup of patients with UCLP patients will present during or after adolescence with multiple cleft-related problems that may include the following:

1. *Maxillary hypoplasia.* The maxilla is often vertically short and canted (upward on the cleft side), and the maxillary dental midline is usually shifted off of the facial midline toward the clefted side. Arch-width deficiency that results in a dental crossbite is usually present. The hypoplastic maxilla is retruded in the horizontal plane, thereby resulting in a concave midface profile. There will be an Angle Class III malocclusion and negative overjet. In general, the greater and lesser maxillary segments vary with regard to the degree of dysplasia, thereby making it difficult to achieve a satisfactory occlusion and facial appearance by repositioning the maxilla in one unit. Separation in two segments (through the cleft) with differential repositioning is often required for the management of the arch shape, even if successful mixed-dentition grafting was accomplished.
2. *Residual oronasal fistula.* Despite a preference for oronasal fistula closure during the mixed dentition, the UCLP candidate for orthognathic surgery will often have residual labial and palatal fistulas. Previous attempts at closure may have failed. Furthermore, buccal (non-keratinized) mucosa may have been placed over the cleft site, thereby resulting in a lack of attached gingiva (keratinized mucosa) in the tooth-bearing region and a loss of vestibular depth.
3. *Residual bony defects.* In the patient with UCLP who has not been successfully or adequately grafted during the mixed dentition, a bony defect not just at the alveolus but throughout the hard palate and the floor of the nose may exist. This results in an inferiorly displaced or deficient floor of the nose and nasal sill with rotation of the anterior nasal spine and the septum toward the cleft. There may also be inadequate alveolar bony, gingival, and periodontal support for the teeth adjacent to the cleft.
4. *Cleft–dental gap.* Studies confirm that, approximately 93% of the time in patients with UCLP, the lateral incisor is either congenitally absent or inadequate at the cleft site.[163,164] A hypoplastic lateral incisor or a supernumerary tooth may be present adjacent to the cleft, but it will have inadequate root development. Orthodontic closure of this dental gap by bodily moving the canine tooth into the bone-grafted lateral incisor location is generally considered the preferred approach. For a variety of reasons, this may not have occurred at the time of referral for orthognathic surgery. At times, there is mesial angulation of the canine (i.e., crown tipped) into a marginally grafted or non-grafted cleft site, while the apex of the root remains distal. The result may be a full or partial dental crown gap at the cleft site between the central incisor and the canine but with an even greater separation of the roots.

NOTE: There are a variety of reasons why the teenaged or adult patient may present with a cleft (lateral incisor) dental gap, including the following: 1) the patient may have "fallen through the cracks" without undergoing mixed-dentition grafting; 2) a grafting procedure may have been carried out but with only limited "graft take"; or 3) the cleft site may have been successfully grafted and followed by a "classic occlusion" approach with the intentional maintenance of the lateral incisor space for later prosthetic replacement. Clinicians that advocate this approach believe that "smile aesthetics" will be more favorable and that maintaining "cuspid protection" during lateral excursions is necessary over "group function."

I have most often found that, for the maxillary deficient individual with UCLP/BCLP with a congenitally absent lateral incisor, either orthodontic gap closure or eventual surgical cleft dental gap closure is preferred for the achievement of an efficient, long-term, low-maintenance dentition. In these cases, favorable smile aesthetics may also benefit from cosmetic enhancements such as crown lengthening or crown modification via dental recontouring or augmentation (i.e., composite resin or veneer buildups).

5. *Chin dysplasia.* The patient with UCLP will frequently suffer with a lifelong history of obstructed nasal breathing and an open-mouth posture. This is often the result of anatomic deformities, including septal deviations, inferior turbinate hypertrophy, an irregular nasal floor, and nasal vestibular stenosis. The presence of a pharyngeal flap since childhood may further

increase an open-mouth breathing tendency. The resulting chin deformities that occur during growth are often characterized by excess vertical length and horizontal retrusion.

6. *Mandibular dysplasia.* True mandibular prognathism or retrognathism is uncommon in the patient with UCLP. However, the need for mandibular osteotomies is frequent. This should be limited to the correction of secondary deformities that result in facial asymmetries and skeletal distortions and the occasional true anteroposterior discrepancy.[88,89,171]
7. *Nasal obstruction and sinus blockage.* Obstructed breathing through the nose and frequent bouts of sinusitis are typical among patients with UCLP. This results from a combination of septal deviation; enlarged inferior turbinates; deformities of the nasal aperture, the floor of the nose, and the anterior nasal spine region; and stenosis of the nasal vestibule.
8. *VP dysfunction.* It has been shown that approximately 20% of patients with a repaired cleft palate will have VP insufficiency by the time they are 5 years old. The adolescent with UCLP who arrives for the evaluation of a cleft jaw deformity may have already undergone a pharyngoplasty with a pharyngeal flap. The planned Le Fort I osteotomy with advancement will alter the upper airway and may also negatively affect VP function, as described later in this chapter.

Orthodontic Considerations in the Patient with Unilateral Cleft Lip and Palate with a Jaw Deformity

The adolescent or adult patient with UCLP who presents with maxillary hypoplasia and ineffective bone grafting performed earlier during his or her childhood will have two maxillary segments separated by a cleft (see Figs. 32-11 through 32-21). Each segment will have a degree of skeletal dysplasia in all three planes of space. From an orthodontic perspective, each segment should be evaluated and treated individually in anticipation of Le Fort I segmental repositioning.

Radiographic assessment is carried out before the orthodontic movement of the teeth adjacent to the bone-deficient cleft site. The Panorex radiograph is useful for assessing the overall morphology of the jaws and teeth as well as tooth angulation. Maxillary occlusal and periapical radiographs through the cleft site or preferably a cone beam computed tomography scan will help with the assessment of the amount of alveolar bone and crestal height surrounding the teeth.

There is variability with regard to the number of permanent incisors and the amount of alveolar bone in the anterior aspect of the UCLP maxilla. A lateral incisor-like tooth is frequently found along the edge of the cleft in the lateral segment. When a poorly formed lateral incisor is present, it should be extracted in the interest of long-term function and dental rehabilitation. Cassolato and colleagues have documented that, in patients with complete UCLP, the lateral incisor on the cleft side is normal and maintained in only 7% of cases.[17] Unerupted supernumerary teeth are also extracted either at the time of bone grafting during the mixed dentition or in conjunction with orthognathic surgery. The central incisor on the cleft side also has a high probability of being malformed.

The decision to extract a fully erupted, normally formed tooth (i.e., the first bicuspid) within the lesser or greater segment depends on the volume and height of the available alveolar bone to house the dental roots adjacent to the cleft and the degree of overall dental crowding within each segment of the jaw. The completion of the first bicuspid extraction in the greater segment, the lesser segment, or both is often required to ensure adequate alveolar bone for leveling and aligning retained teeth without weakening the periodontal support of the teeth adjacent to the cleft and throughout the alveolus (e.g., loss of crestal height, fenestration or dehiscence of the labial plate).

The orthodontist must be aware that the placement of preangulated brackets or artistic positioning bends can displace the roots of the teeth adjacent to the cleft from their alveolar housing with a loss of crestal bone height. A loss of vertical crest height along the cuspid is likely to occur with excessive mesial crown tipping. For these reasons, the orthodontic movement of the cleft-adjacent teeth is accomplished cautiously until the cleft defects are surgically closed, effectively bone grafted, or both.

A potential disadvantage of the modified Le Fort I approach in a patient with UCLP is that the differential advancement of the lesser dentoalveolar segment shifts the preoperative anterior dental gap (i.e., the lateral incisor region) into the posterior region (i.e., the second molar region) (see Fig. 32-5 and discussion later in this chapter). This is no different than the occlusal changes that will occur when the mixed-dentition bone-grafted cleft–dental gap (i.e., with absent lateral incisor) is orthodontically closed by canine substitution. Unfortunately, the mandibular second molar on the cleft side may no longer have an opposing maxillary molar. For this reason, the maxillary second molar should be orthodontically included in the arch form. Occasionally there will be a non-impacted maxillary wisdom tooth that can oppose the mandibular second molar. Articulated dental models with the maxillary segments in their proposed postoperative positions are analyzed to confirm that the posterior maxillomandibular occlusion will be acceptable.

The incorporation of all erupted teeth, including the second molars, within the orthodontic mechanics (especially in the lesser maxillary segment) will facilitate the development of the desired arch form. A lingual attachment on the maxillary first-molar bands allows for the use of a transpalatal arch. This may be useful to support molar anchorage during treatment and to stabilize the surgical result after splint removal. The development of an ideal mandibular arch is usually straightforward. A decompensated

mandibular arch is critical for the effective repositioning of the maxillary segments into the most favorable position at the time of orthognathic surgery.

At the time of the operation, the maxillary segments are secured into a prefabricated acrylic occlusal splint that is then typically left in place for 5 weeks. The patient is educated about the application of elastics and encouraged to remove the elastics intermittently to establish an early range of mandibular opening after surgery. The orthodontist sees the patient within 24 hours of splint removal to replace the maxillary sectional arch wires with a rigid continuous arch wire. Occlusal maintenance and active orthodontic treatment are reinitiated. Close monitoring is essential to maintain the arch form and dental positioning achieved (i.e., horizontal, transverse, and vertical). The placement of a transpalatal appliance (i.e., a wire or a removable palatal plate) may also be useful to maintain the new arch form that is created at the time of operation.

Immediate Presurgical Assessment

Approximately 4 to 6 weeks before the operation, the orthodontist will confirm that the preoperative orthodontic objectives have been met and then place the passive surgical wires. The surgeon takes the final records, which include alginate impressions of the maxillary and mandibular arches, a centric relation bite registration, a face-bow registration, and direct facial measurements. The patient's medical and dental records (e.g., radiographs, consultation reports, facial and occlusal photographs, dental models, special studies) are reviewed. Decisions are finalized with regard to the preferred vector changes (repositioning) of the jaws and the precise linear (millimeter) distances and angles to be accomplished in each jaw for the desired result (see Chapter 12).[2,38,44,45,68,76,94,100,101] Analytic model planning is carried out on the articulated dental casts, and splints are fabricated. The splints assist with the achievement of the precise occlusion and the preferred facial aesthetics that have been decided on preoperatively (see Chapter 13).

Orthognathic Approach for Unilateral Cleft Lip and Palate Deformities

Evolution of Surgical Technique

Historically, the literature warned of possible complications with maxillary osteotomy among patients with UCLP but provided only limited and often confusing descriptions of techniques to guide the orthognathic surgeon in the performance of safe, reliable osteotomies to solve these complex problems.[158]

As with other aspects of orthognathic surgery, Hugo Obwegeser's milestone contributions to cleft skeletal reconstruction are important (see Chapter 2). By the late 1960s, Obwegeser succeeded in advancing a cleft maxilla to the preferred location without the need for a compromised mandibular setback approach. He eventually felt comfortable with cleft maxillary advancements of up to 20 mm. He also realized that the adequate mobilization of the Le Fort I down-fracture was the key step in advancing the maxilla, whether the patient had clefting or not. With experience, the value of simultaneously closing the cleft–dental gap by moving the lesser segment further forward to position the canine in the lateral incisor location was appreciated. This differential segmental repositioning would essentially replace an anterior dental gap for an edentulous space in the posterior maxilla.

Early on, other surgeons without Obwegeser's level of expertise reported complications after advancing the maxilla in patients with UCLP.[113-117,119,120] In 1974, Willmar described the problems that occurred in 17 patients with UCLP who underwent Le Fort I osteotomy. One patient had aseptic necrosis and a partial loss of the lesser segment of the maxilla.[178] In 1974, Georgiade suggested that a camouflage approach with mandibular osteotomies and set-back was preferred to maxillary advancement to avoid complications.[51] Kiehn and others and Des Prez and Kiehn warned of blood-supply problems that might occur with maxillary osteotomies in patients with cleft lip and palate.[30,83] In 1975, Henderson and Jackson reported combining lip-scar revision, anterior fistula closure, and maxillary osteotomy in a one-stage procedure.[69] Their concept was innovative, but they did not specify details of their technique. In 1978, Jackson described the technique of Le Fort I osteotomy in patients with cleft lip and palate and noted that, if a large fistula was present, extensive flap mobilization for closure was required, and the blood supply might be compromised.[77]

Historically, surgeons were leery of completing a Le Fort I down-fracture and full mobilization in patients with UCLP because they feared flap necrosis with a subsequent loss of bone and teeth.[6,7,31-33,52,53,87] In 1980, Tideman and colleagues proposed the segmental palatal osteotomy for patients with cleft lips and palates.[168] This procedure required significant subperiosteal degloving with the potential for compromised flap circulation but without providing the needed direct exposure for full disimpaction. In 1980, Sinn also reported on the simultaneous Le Fort I advancement, oronasal fistula repair, and bone grafting of the alveolar cleft.[155] He stressed the importance of preserving a vertical soft-tissue pedicle, which was similar to that described by Tideman and colleagues. He completed osteotomies through tunnels rather than under direct vision and used a cheek rotation flap for oral-side closure of the labial and palatal oronasal fistulas. With this technique, nonkeratinized buccal mucosa (rather than attached gingiva) was brought into the cleft tooth-bearing region. In 1984, Ward-Booth and colleagues described the results of Le Fort II osteotomy in 13 patients with clefts for the management of midface hypoplasia in an attempt to improve blood supply to the alveolar segments.[175] The residual fistulas were not simultaneously closed, and fixation occurred with direct wires, intermaxillary fixation, and external appliances. In 1985, James and Brook described another variation for the

correction of maxillary hypoplasia in patients with cleft lips and palates via the transection of the hard palate.[78] They raised an extensive palatal flap and used three vertical stab incisions for exposure on the labial aspects of the maxilla. Access was then achieved through subperiosteal tunneling and without direct exposure. Fixation was with intermaxillary fixation and a halo head frame, which was maintained for 10 weeks. A second procedure was required for bone grafting and palatal fistula closure. The authors expressed concern that a more direct down-fracture of the maxilla would result in vascular compromise to the segments. In 1985, Poole and others proposed an additional modification of the Le Fort I osteotomy to be used in patients with cleft palates.[128] With their technique, a partial-thickness palatal flap was elevated, which left the greater palatine vessels in situ and allowed the maxilla to be repositioned anteriorly, without displacement of the soft palate. Poole and colleagues believed that this approach would limit interference with VP function; they also used small vertical incisions on the labial aspect that required tunneling and lacked direct exposure for osteotomies, disimpaction, fistula closure, bone-graft placement, and plate and screw fixation. Fixation in this series was with intermaxillary fixation, direct wires, and a halo craniomaxillofacial head frame.[128]

During the 1980s and the 1990s, Posnick used and refined Obwegeser's original techniques involving Le Fort I osteotomy for the treatment of UCLP deformities.[129-142] A key aspect was the circumvestibular incision, which allowed for direct exposure for dissection, osteotomies, disimpaction, fistula closure, septoplasty, inferior turbinate reduction, pyriform aperture recontouring, bone grafting, and the application of plate and screw fixation (see Fig. 32-5). This was found to be a reliable approach that did not involve the risk of circulation injury to the greater and lesser dento–osseous–musculo–mucosal segments. The visibility provided by the circumvestibular incision made possible the incorporation of routine closure of the cleft–dental gap through differential maxillary segmental repositioning without necrosis of the bone or any loss of teeth. This method also closes the cleft dead space and brings together the labial and palatal flaps without the need for subperiosteal undermining, which allows for the closure of recalcitrant oronasal fistulas without tension and the establishment of the periodontal health of the cleft adjacent teeth. The down-fracture provides ideal exposure for septoplasty; for the reduction of the hypertrophic inferior turbinates; and for the recontouring of the pyriform rims, the floor of the nose, and the anterior nasal spine. The success of this approach, as initially carried out by Obwegeser,[114-117,119,120] was confirmed by Bell's demonstration of the blood supply to these maxillary segments in animal studies.[6-7] The clinical advantages were then further documented by Posnick and colleagues in a consecutive series of UCLP patients.[129-142]

Since the mid 1990s, the literature concerning cleft orthognathic surgery has mostly focused on use of DO techniques to avoid the need for complete intraoperative mobilization of the maxilla in the hopes of improved long-term stability.[166] Although the issue of skeletal stability and dental relapse for precise long-term occlusion is important, it should not overshadow the value of achieving the other planned improvements of the airway and enhanced facial aesthetics.[129-142] Unfortunately, the use of DO techniques has not altered the challenges of cleft jaw surgery, which include the following: 1) a lack of postoperative growth when maxillary advancement is carried out in children; 2) a worsening of VP function in some patients; 3) injury to the teeth and bones if circulation to the segments is not maintained; 4) facial and intraoral sensory loss; 5) suboptimal facial aesthetics when basic principles are not followed; and 6) concern for postoperative skeletal or dental relapse (i.e., residual malocclusion). In addition, DO techniques require a prolonged and labor-intensive postoperative convalescence for the patient and family as compared with the standard approach. Despite these disadvantages, DO does offer hope to the occasional patient with UCLP who also has severe maxillary hypoplasia and anodontia (see Fig. 32-21 and the section about controversies later in this chapter).

Standard Le Fort I Osteotomy

The adolescent or adult CLP patient with a jaw deformity but without a residual fistula and with an intact alveolar ridge of adequate height and volume in the area of the cleft may have been born without alveolar clefting or had a successful graft.[34] For those with adequate alveolar ridge height and volume, a closed palate, and sufficient periodontal support, a standard Le Fort I osteotomy can be performed (see Figs. 32-8, 32-9, and 32-10). A segmental maxillary osteotomy to adjust the arch width, to correct the vertical dimension, or to close the cleft dental gap to avoid the need for a prosthetic lateral incisor may also be necessary in some of these patients (see Chapter 15). Unfortunately, even in the 21st century, a number of adults and adolescents with UCLP with maxillary hypoplasia also present with alveolar defects and oronasal fistulae. For these patients, a modified (two-segment) Le Fort I osteotomy should be considered, as discussed in the next section of this chapter (see Figs. 32-11 through 32-21).[129-142]

Modified Le Fort I Osteotomy (Two Segments)

> *In unilateral cleft cases, the dental gap of the [absent] lateral incisor can be eliminated by advancing the lateral alveolar process so that the canine is positioned next to the central incisor. The canine then subsequently is contoured to match the appearance of the lateral incisor.*[118,119]
>
> **HUGO OBWEGESER**

A maxillary circumvestibular incision is made from one zygomatic buttress to the other without the need to maintain a labial pedicle. Parallel vertical incisions are then made labially in the region of the residual oronasal fistula to

separate the oral and nasal mucosa on each side of the cleft. These incisions are perpendicular to the horizontal vestibular incision and follow the line angles of the teeth adjacent to the cleft (i.e., the canine and the central incisor). The nasal and oral mucosa are carefully incised along the palatal aspect of the residual oronasal fistula. Further separation of the tissue layers is accomplished when dissecting the nasal mucosa free of the bony floor during down-fracture. The complete separation of the oral and nasal layers is necessary for the initial down-fracture and then later for the fistula closure. This is accomplished without degloving the palatal mucosa from the underlying hard palate, because this would compromise the circulation to the maxillary segments.

Before osteotomy, subperiosteal dissection on the anterior surface of the maxilla is completed up to the infraorbital foramina. The anterior nasal spine, the pyriform apertures, and the floor of the nose are also exposed. Dissection continues posteriorly past the maxillary tuberosities to the pterygoid–maxillary suture on each side. After retractors have been put in place, horizontal osteotomies are carried out above the roots of the teeth and into the maxillary sinus through the lateral, anterior, and medial maxillary walls with a reciprocating saw and a long straight blade. The nasal septum is separated from the base of the maxilla with the use of a protected chisel. The pterygoid–maxillary sutures are separated with a curved chisel, and the maxilla is down-fractured. Direct trauma of the palatal mucosa via compressive forces during mobilization should be minimized, because such forces could compromise the vascular supply.

Subperichondrial and subperiosteal dissection of the deviated vomer, the perpendicular plate of the ethmoid, and the quadrangular cartilage is accomplished after the maxilla has been down-fractured. Resection of the deviated and buckled aspects of the septum (i.e., the bone and cartilage) is done with a rongeurs while preserving the structural components of the cartilaginous septum (i.e., the dorsal strut and the caudal strut) that are necessary to prevent a late saddle-nose deformity (see Chapter 15). If the inferior turbinates are enlarged, they are also reduced to improve the airway. The nasal mucosa flaps are sutured for a watertight nasal-side closure. If indicated, the impacted maxillary third molars are removed from above through the maxillary sinus (see Chapter 15).

The down-fractured maxilla may already be in two segments if the bony cleft was unrepaired. If the maxilla is intact but needs correction of the arch form, then it is separated using a reciprocating saw with short straight blade. The approximation of the segments to close the cleft–dental gap can occur after bony spurs are shaved from the alveolus along the distal aspect of the central incisor and the mesial aspect of the canine using a rotary drill with a watermelon bur. Care is taken to avoid penetrating the lamina dura, which would expose the dental root and may result in external root resorption. The maxillary segments are then ligated into a prefabricated acrylic occlusal splint. The segmental repositioning closes the cleft–dental gap, brings the alveolar ridges together, and approximates the labial and palatal mucosal soft tissues for oral-side fistula closure. The differential segmental repositioning approximates the mucosal edges along the palate, so they do not need to be sutured. In fact, to limit invaginations of the oral mucosa, the excision of redundant palatal mucosa is generally required just before the segments are placed in the splint. The placement of the splinted maxilla onto the mandibular dentition should be passive. The extent of maxillary advancement is based on the preferred occlusion and the facial aesthetics determined preoperatively. The ideal vertical dimension is achieved intraoperatively on the basis of the preoperative plan (see Chapter 12). The maxillary osteotomy sites are fixed in place with titanium miniplates and screws at each zygomatic buttress and pyriform rim in accordance with the principles originally described by Luhr.[92] An additional microplate is frequently applied horizontally across the cleft site to stabilize the closure of the cleft–dental gap. The intermaxillary fixation is released, and the occlusion is checked.

The lateral nasal rims, the floor of the nose, and the anterior nasal spine region are recontoured using a rotary drill with a watermelon bur to improve the nasal airway and to enhance nasal aesthetics (see Chapter 15). The dead space associated with the alveolar cleft has already been closed by the differential segmental repositioning. Autogenous iliac cancellous bone graft may be packed along the floor of the nose to raise the ipsilateral sill and across the cleft palate to stabilize the segments. Generally, a crafted iliac corticocancellous bloc graft is placed in between the zygomatic and pyriform fixation plates on each side. Each graft is tightly wedged into the space created by the horizontal advancement and the vertical lengthening (see Chapters 15 and 18). An additional microplate is contoured and secured across the osteotomy, and it also incorporates the graft. With the cleft–dental gap surgically closed, the redundant labial mucosa is excised. Attached gingiva and mucosal edges are approximated without tension and then directly sutured without the need for rotation flaps.

Mandibular and chin osteotomies to correct secondary deformities and facial asymmetries are frequently planned. If so, the osteotomies are completed and secured with plate and screw fixation in standard sequence (see Chapter 15). After the completion of all of the osteotomies and the placement of fixation and grafts, the wounds are closed. The upper and lower teeth are approximated with orthodontic elastics. When maxillary segmental osteotomies are completed, the splint remains wired to the upper teeth postoperatively (see the orthodontic considerations section earlier in this chapter).

Avoiding Pitfalls

Mobilization of the Down-Fractured Cleft Maxilla

Full mobilization of the Le Fort I down-fracture is a critical step in the achievement of accurate intraoperative maxillary

repositioning and then with long-term maintenance. Both pterygoid and nasomaxillary disimpaction forceps are used as needed to achieve full mobilization. Taking the necessary time to slowly complete the disimpaction (i.e., 10 to 15 minutes) is often crucial to achieving the desired results. Gaining confidence—first through observing and assisting an experienced cleft jaw surgeon and then by using one's own hands—is essential to achieving success with the mobilization process.

Horizontal Advancement and Vertical Lengthening of the Cleft Maxilla

The surgeon must often carry out significant horizontal advancement, vertical lengthening, and midline, cant, and occlusal plane correction to enhance facial aesthetics, achieve dental health, and open the airway in a patient with UCLP. The scarring of the palatal soft tissues from previous repairs may be of concern when planning an extensive advancement. A scarred and hypotonic repaired unilateral cleft lip may also caution the surgeon against the vertical lengthening that the midface demands.

In general, to achieve full skeletal correction, all or most of the negative overjet should be managed in the maxilla. The sagitally deficient maxilla in a patient with UCLP will also typically need vertical lengthening. When the maxilla is fully mobilized and repositioned as described previously, stabilization with titanium plates and screws (i.e., a plate at each zygomatic buttress and each pyriform aperture) is performed. The placement of a bloc corticocancellous graft wedged into the dead space between the zygomatic buttress and the pyriform aperture plate on each side is generally necessary (see Chapter 15). Further stabilization across each graft with a titanium plate and screws is also required (see Chapter 15).

Closure of Residual Oronasal Fistula and Alveolar Ridge Management

Ideally, full closure of a residual oronasal fistula and adequate alveolar bone grafting will have been carried out during the mixed dentition. If not, the modified Le Fort I osteotomy in two segments (as described previously) provides the opportunity for complete oronasal fistula closure of the palatal and labial aspects and for the management of skeletal deficiencies without the need for direct alveolar grafting. Many times, grafting and fistula closure will have been carried out during the mixed dentition but without complete correction. A residual fistula may remain on the palatal side; if the dental gap was not closed with the use of orthodontic mechanics (cuspid advancement) after grafting, the volume of bone at the cleft site is likely inadequate for dental implant placement. Unfortunately, when completing a Le Fort I down-fracture without segmental repositioning, any residual palatal fistula cannot be reliably closed, because this would require elevating the palatal flaps and a risk of aseptic necrosis. Direct augmentation of an intact but hypoplastic alveolar ridge at the time of down-fracture is also not practical, because excessive soft-tissue flap elevation around the ridge would be required.

Managing the Nasal Cavity and the Nose

In the patient with UCLP, the inferior turbinates will often be enlarged, asymmetric, and partially blocking the nasal cavity. The reduction of enlarged inferior turbinates will be beneficial for breathing and sinus drainage. The procedure is accomplished through the Le Fort down-fracture (see Chapter 15 and Fig. 32-10).

In the patient with UCLP, the septal bone and cartilage generally show significant deviation and thickening, thus further obstructing the airway. The completion of a submucous resection of the deviated portions of the septum will be helpful to open the airway. The preservation of caudal and dorsal cartilaginous struts will prevent a saddle deformity; this is accomplished through the Le Fort down-fracture (see Chapter 15 and Fig. 32-10).

In the patient with UCLP, the pyriform rims are generally constricted and asymmetric. The nasal floor is uneven, and the anterior nasal spine is deviated. After Le Fort down-fracture, the recontouring of the pyriform rims and the nasal floor and spine using a rotary drill with a watermelon bur is helpful to open the airway and improve nasal aesthetics (see Chapter 15 and Fig. 32-10).

Attempts to "control" the nasal soft-tissue envelope with suturing techniques (e.g., the alar cinch stitch) after Le Fort I osteotomy are often suggested. In my experience, manipulating the nasal soft tissues with suturing techniques after Le Fort I may be counterproductive to both the airway and the facial aesthetics. We anticipate that the nasal soft tissues will be redraped over the repositioned maxilla and the recontoured pyriform rims, nasal floor, and nasal spine for an overall improvement in aesthetics. A definitive rhinoplasty to fully correct the UCL nasal deformities is often beneficial; this is best postponed until 6 to 12 months after jaw reconstruction (see Chapter 38).

Immediately after surgery, the nasal cavity is not packed, and the septum is not stented. Coagulated blood will partially block nasal breathing. The clots and scab are expected to dislodge and be self-eliminating after the underlying mucosa is initially healed (i.e., 5 to 7 days after surgery). The use of saline nasal sprays and the institution of sinus precautions are helpful during this time (see Chapter 11).

Management of the Mandibular Deformity

In the individual with UCLP, the mandible is often secondarily deformed. In a previously published study, 70% of the patients with UCLP had secondarily deformed mandibles to the extent that sagittal split ramus osteotomies were beneficial to improve facial symmetry and proportions.[141] The mandibular repositioning is not carried out in an attempt to avoid maxillary advancement but rather to improve overall facial morphology (see Chapter 15).

Management of the Chin Deformity

In the patient with UCLP with maxillary hypoplasia, the chin is often secondarily deformed, with increased vertical length and a flat pogonion. An intraoral oblique inferior border osteotomy is carried out with repositioning and reshaping of the distal chin to accomplish the preferred morphology (see Chapters 15 and 37).

Clinical Management after Initial Surgical Healing

Managing the details of the in-hospital and at-home convalescence during the initial healing of the orthognathic patient are essential for a successful outcome (see Chapter 11). Cephalometric and dental radiographs and facial and occlusal photographs are obtained at standard postoperative intervals to document patient healing. The orthodontist sees the patient within 24 hours of splint removal (approximately 5 weeks after surgery) and replaces the maxillary sectional arch wires with a rigid continuous arch wire. The maxillary teeth are ligated together to maintain the surgical dental-gap closure, the horizontal advancement, and the transverse expansion. Active orthodontic maintenance and finishing are started. The use of a transpalatal appliance (i.e., a wire or a palatal plate) may also be used to stabilize the new arch form. Close monitoring by an orthodontist for skeletal and dental shifts during the first 6 months after surgery is essential.

Speech can be objectively reassessed 3 to 6 months after surgery. We prefer to do so with the use of nasoendoscopic instrumentation. Definitive cleft soft-tissue procedures (e.g., cleft rhinoplasty, lip scar revision, pharyngeal flap or flap revision) can be carried out as early as 6 months after orthognathic surgery. After the orthodontic appliances are removed, any planned definitive dental restorative work can be finalized.

Orthognathic Surgery for Unilateral Cleft Lip and Palate: Review of Study

Patients and Methods

Posnick and Tompson prospectively assessed the cleft deformity and clinical results of 66 consecutive adolescents and young adults (age range, 15 to 25 years; mean, 18 years) with UCLP who underwent orthognathic surgery by one surgeon (Posnick) with the use of a single surgical protocol during a 6-year time period.[141] All patients underwent perioperative orthodontic treatment and were judged to be skeletally mature at the time of jaw surgery. The clinical follow-up period after maxillary advancement ranged from 1 to 7 years (mean, 40 months). The countries of origin of the patients varied (e.g., Canada, United States, Eastern Europe, Southeast Asia), as did their races (e.g., Caucasian, Asian, African). These patients had their cleft lips and palates repaired by many different surgeons who were using a variety of protocols. The number and extent of previous revisions of the lip, nose, and palate varied greatly (range, 1 to 9 procedures). Many patients had undergone multiple attempts at closure of the residual oronasal fistula and at the reconstruction of the alveolar clefts with bone graft. Seven of the 66 patients with UCLP had previously undergone orthognathic surgery by another surgeon; all seven had residual maxillary hypoplasia, a recalcitrant oronasal fistula, and a cleft–dental gap.

The basic orthognathic procedure carried out by Posnick included a modified Le Fort I osteotomy in two segments (n = 66). Twenty-three of these patients also require simultaneous sagittal split ramus osteotomies to correct facial asymmetry and disproportion. Thirty-five of the 66 patients (53%) underwent osseous genioplasty (vertical reduction and horizontal advancement) to improve their profile aesthetics and lip position.

The 66 adolescents with UCLP had multiple residual deformities, and all but one had residual oronasal fistulas of varying sizes. Sixty-four of the 66 patients (97%) presented with negative overjet at the central incisors. Sixty of the 66 patients (91%) had a lateral (posterior) crossbite of the lesser segment and a maxillary dental midline that was not coincident with the facial midline. An Angle Class III malocclusion was present on the cleft side in 64 of 66 patients. In 60 of the 66 patients (91%), a congenitally absent or structurally inadequate lateral incisor was present at the cleft site. Simultaneous differential repositioning of the maxillary segments was planned for all 66 patients. In 57 of the 60 patients who presented with a cleft–dental gap, surgical closure through segmental repositioning also was planned. Despite the successful preoperative orthodontic cleft–dental gap closure in six patients, surgical arch width and coordination problems remained and required segmental repositioning.

Results

Sixty-one of the 66 patients (92%) underwent successful simultaneous oronasal fistula closure. In the five patients with residual fistulas, definitive closure with standard mucogingival and palatal flaps as a secondary procedure was possible and did not require bone grafting. Surgical cleft–dental gap closure was achieved and maintained in all but 3 of the 57 patients in whom it was attempted. In these patients, the dental gap closure was achieved initially; however, over the subsequent 12 months, the teeth in the lesser segment shifted position. The dental gap opened even though the fistula remained closed, and skeletal continuity across the cleft remained intact.

Nine patients required fixed (prosthetic) bridgework, either to complete the closure of the cleft–dental gap or to achieve other aspects of dental rehabilitation. A bridge was required in three patients in whom a dental gap was intentionally left open and in three others when the space reopened. The techniques of porcelain-veneer finish,

composite resin buildups, and tooth sculpting were used to resurface incisor teeth in three other patients as a result of chronic tooth decay and enamel dysplasia.

In all patients, keratinized mucosa was successfully maintained along the labial surface of the cleft-adjacent teeth (n = 132 teeth). Preoperative gingival recession along the distal aspect of the central incisor as a result of longstanding limited alveolar support could not be improved at the time of the orthognathic surgery. Five of the 132 cleft-adjacent teeth required root canal therapy, and five teeth were noted to have further gingival recession after the completion of orthodontic treatment.

The long-term maintenance of overjet was measured directly from the late (>1 year) postoperative lateral cephalometric radiograph; all but two patients (i.e., 64 out of 66) maintained a positive overjet. The long-term maintenance of overbite was also measured directly from the late postoperative lateral cephalometric radiograph; 60 of 66 patients (91%) maintained a positive overbite; 4 patient shifted to a neutral overbite; and 2 patients relapsed into a negative overbite.

Complications were few and generally not serious. One patient who had also undergone septoplasty and inferior turbinate reduction was returned to the operating room for nasal packing to manage epistaxis on postoperative day 10. In another patient, the maxilla was repositioned a second time to reduce the vertical height (gingival show) for improved facial aesthetics on postoperative day 3. No loss of segmental bone or teeth occurred as a result of aseptic necrosis, infection, or for any other reason.

Skeletal Stability after Modified Le Fort I for Unilateral Cleft Lip and Palate Deformity: Review of Study

In general, published studies of long-term skeletal stability after cleft–orthognathic surgery tend to group populations that should be considered separately (e.g., BCLP, UCLP, isolate cleft palate); they have an inadequate follow-up period (e.g., <1 year) or too small of a sample for statistically significant results; or they only provide information about cephalometric measured length and angles rather than evaluate clinically relevant parameters (e.g., maintenance of overjet).

Patients and Methods

Posnick and colleagues reviewed medical records and cephalometric radiographs and completed current surgical and orthodontic clinical examination of all patients (n = 45) with UCLP who had undergone Le Fort I osteotomy by a single surgeon (Posnick) during a 3-year period.[139] The following information was noted: all previous maxillofacial procedures, details of the orthognathic procedures, osteotomy stabilization techniques used, the presence of a pharyngoplasty, the extent of bone grafting, the segmentalization of the Le Fort I osteotomy, perioperative orthodontics, the age at surgery, the age at final follow up, the amount of overjet and overbite at the 1 year or more postoperative visit, and perioperative morbidity.

For all patients, the prospective protocol required a lateral cephalogram preoperatively, immediately (i.e., 3 to 7 days after surgery), at 6 to 8 weeks, and at 1 year after surgery. At the time of the clinical review, patients were excluded from the study if one or more interval cephalograms were not available (n = 7) or if less than 3 mm of horizontal advancement was required to correct their skeletal deficiency (n = 3).

The serial radiographs for each patient were analyzed with a modified version of the method described by Bachmayer and colleagues.[5,6] On each preoperative tracing, the horizontal and vertical coordinates that represented the patient's natural head position were constructed so that they passed through the sella. These reference lines were transferred and became the x-y grid template onto which all of the postoperative maxillary positional changes could be traced and measured. The end result was a Cartesian coordinate system that illustrated the horizontal and vertical directional changes of the maxilla at intervals after surgery. The measurements of the differences in the position of the maxilla at different postoperative points were taken from the grids and were calculated to within 0.5 mm. In addition, the incisor overjet and overbite measurements from the 1-year (or more) postoperative cephalogram were documented and compared with the measurements taken at the final (1 year or more postoperative) clinical assessment. Differences between groups (e.g., pharyngeal flap in place) were assessed via Student's *t*-test. Any association between the magnitude of surgical change and the degree of relapse was examined with the Pearson correlation coefficient (*r*) and the least-squares linear regression analysis. Correlations were considered significant when the *P* value was less than .05.

Results

During this study period, 45 skeletally mature patients with UCLP patients underwent Le Fort I maxillary advancement. None were lost to follow up and the final clinical follow-up examination ranged from 1.5 to 4.5 years (mean, 2.5 years) from the time of the surgical procedure and the close of the study. Seven patients missed one of their longitudinal cephalometric records, and three required less than 3 mm of horizontal advancement to correct their maxillary deformity. The 10 excluded patients were reviewed and were not clinically different from the other 35, varying only for the reasons outlined. All patients in the study were judged to be skeletally mature at the time of operation on the basis of the assessment of either a carpal (wrist) radiograph or at least two static serial cephalometric radiographs taken at 6-month intervals. For the 35 patients who were included in the study, the age at operation ranged from 14 to 25 years (mean, 18 years).

All 35 study patients underwent modified Le Fort I osteotomies (two segments) with varying degrees of horizontal advancement, transverse arch widening, and vertical change. Eleven of the 35 patients also underwent sagittal split ramus osteotomies of the mandible to correct asymmetries and secondary deformities; 22 had vertical reduction genioplasty with or without chin advancement to improve their profile aesthetics. In 13 of 35 patients, a pharyngoplasty was in place at the time of the maxillary osteotomy. None required takedown or modification of the flap to achieve passive intraoperative maxillary placement. All 35 patients underwent perioperative orthodontic treatment that was coordinated with their surgical procedure.

No statistically significant differences were seen in the initial vertical or horizontal surgical changes and the postoperative maintenance (relapse) between patients who had maxillary surgery alone or those who had surgery in both jaws and those who had a pharyngoplasty in place at the time of their Le Fort I osteotomy ($P < .05$). Therefore, these patients were considered a homogeneous group for further analysis. The mean horizontal advancement at the incisors achieved for the group was 6.9 mm, with 5.3 mm maintained 1 year later. In 11 of the 35 patients, the relapse was less than 1.0 mm. The amount of horizontal relapse did not correlate significantly with the amount of maxillary advancement ($r = .31$).

In the 13 patients with a pharyngoplasty in place at the time of the Le Fort I osteotomy, the mean effective horizontal advancement was 8.2 mm immediately after the operation and 6.5 mm 1 year later. Interestingly, the 22 patients without a pharyngoplasty in place required less horizontal advancement, with a range of 6.4 mm and 4.9 mm maintained at 1 year. The mean vertical change of the maxilla was 2.1 mm initially after the operation and 1.7 mm 1 year later. In 12 of the 35 patients, no vertical change was necessary; in the remaining patients, vertical directional change was necessary and achieved. Analysis indicated that vertical relapse did not correlate significantly with the magnitude of change that occurred at the time of surgery ($r = .41$). The authors also measured the overjet and overbite directly from the cephalometric radiographs at the 1-year postoperative interval in each patient. A positive overjet was maintained in all patients, whereas a positive overbite was maintained in 86% (30 of 35 patients). This data confirms that, with the combination of orthodontics and orthognathic surgery, a functional long-term occlusion was achieved in most of the patients with UCLP.

Controversies and Unresolved Issues

Staging of Maxillary Reconstruction

The described modified Le Fort I segmental osteotomies as a method of managing end-stage oronasal fistulas, alveolar defects, and cleft–dental gaps in patients with UCLP who have maxillary hypoplasia is not intended to replace standard techniques and the accepted sequencing of treatment. We always prefer secondary bone grafting during the mixed dentition typically with orthodontic closure of the cleft dental gap. However, the method described does offer an alternative approach when the opportunity for grafting during the mixed dentition (i.e., before the eruption of the permanent canine) is lost and a jaw deformity also exists. Published studies from major cleft centers in the United Kingdom, Scotland, and Canada confirm that, despite best efforts, a number of patients in the mixed dentition will not arrive for treatment, and a percentage of those who do will undergo unsuccessful grafting.[39,103,108,177] A two-stage approach to the adolescent or adult with maxillary hypoplasia, residual alveolar clefts, oronasal fistulas, a cleft–dental gap, and nasal obstruction is not cost- or time-effective and, in my experience, the potential overall morbidity is increased. It is in these patients with UCLP that the modified Le Fort I osteotomy offers a reasonable opportunity for the resolution of residual end-stage problems (e.g., maxillary hypoplasia, alveolar defect, residual fistulas, cleft–dental gap, nasal obstruction) in a safe and effective way.

In the patient with UCLP, there are several options for the management of a cleft–dental gap.* From both facial aesthetic and dental health perspectives, the long-term use of a removable partial denture is always a second choice. Fixed bridgework is a viable alternative, but this requires the partial destruction of adjacent normal teeth, it may look artificial, it requires replacement at intervals throughout the patient's life, and it demands ongoing meticulous oral hygiene (i.e., high-maintenance dentition). Placement of a single-tooth osseous integrated implant is an attractive alternative, but the implant's aesthetic success is dependent on adequate bone height, width, and volume and sufficient attached gingiva.[62,93,122,124,146,165,166,172,174,185] This is not routinely established at the cleft site, even after bone and gingival grafting. In addition, it requires staged surgical procedures, and it also produces a high-maintenance dentition. Coordinated comprehensive periodontal and prosthetic rehabilitation is required, and even then failure to provide a natural-looking smile may occur. In a published study conducted at The Hospital for Sick Children in Toronto, only 10% of complete UCLP lateral incisor dental gaps were managed with a dental implant and crown.[17] Other dental refinements for a patient's dysmorphic and often hypoplastic and decayed anterior maxillary teeth include porcelain-veneer buildups, composite bonding, sculpting of the teeth, and bleaching techniques. Each of these refinements may improve the patient's function, smile aesthetics, and self-esteem.

A major advantage of the described modified Le Fort I (segmental) osteotomies in the patient with UCLP is its ability to simultaneously close the cleft dead space, the residual oronasal fistulas, and the alveolar defect and to stabilize the dentoalveolar segments. The study by Posnick

*References 1, 10, 17, 58, 62, 64, 74, 93, 112, 122, 124, 145, 146, 159, 161, 163, 165, 166, 172, 174, 176, 185

and colleagues documents the improved long-term periodontal health achieved in the cleft dentoalveolar regions when this technique is used.[141] The long-term benefits to the patient of the resulting low-maintenance dentition cannot be over stated. Another advantage of this approach occurs when the lesser segment is advanced to attain this goal. By doing so, a relative arch-width expansion is gained at the mandibular molar and the premolar region without the actual need for a lateral shift of the whole segment. This limits any arch-width relapse tendencies.

Velopharyngeal Function after Le Fort I Advancement

Uncertainties about VP function and the management of an in-place pharyngeal flap should no longer be limiting factors when orthognathic surgery is necessary in a patient with a cleft. A nasoendoscopic guided examination by a speech pathologist and a surgeon who are familiar with cleft anatomy can reasonably predict current and expected VP function in a patient who is scheduled for a Le Fort I osteotomy (see Chapter 8). When postoperative VP deterioration is anticipated, the patient and family are counseled about the sequencing of treatment. Clinical studies have now documented that VP function will deteriorate in a similar fashion when either DO or standard Le Fort I osteotomy techniques are used.[19,20,57,60,79,86] Despite the frequent need for significant maxillary advancement to normalize the skeleton and the facial aesthetics in patients with UCLP, I have not had to transect an in-place pharyngeal flap to achieve maxillary mobilization. Our research and that of others confirms that a pharyngeal flap in place at the time of Le Fort I osteotomy does not increase complications nor does it result in a higher incidence of relapse.[141] The definitive reassessment of VP function after cleft Le Fort I advancement can be carried out 3 months after surgery. A primary or revision pharyngeal flap can be safely carried out within 6 months after surgery in conjunction with cleft rhinoplasty or labial revision, if indicated.

Mixed Dentition Le Fort I Osteotomy

By the mid 1980s, research clarified that, if jaw surgery is undertaken in the growing patient with a cleft palate, another procedure to advance the maxilla will likely be required when skeletal maturity is reached.[181-184] More recently, several investigators again tested this theory by proceeding with mixed-dentition Le Fort I procedures using DO techniques. All research to date indicates that the Le Fort I advancement carried out during the mixed dentition in the patient with clefting—whether with standard or DO techniques—results in no significant further horizontal growth.[21,26,40,41,59,61,75,126] As the mandible continues to grow, an Angle Class III malocclusion will occur with the need for either additional Le Fort I advancement or mandibular set-back.

Skeletal Relapse after Le Fort I Osteotomy in Patients with Clefting

There are more than 100 published articles reviewing skeletal stability and relapse in patients with clefting who have undergone Le Fort I advancement with the use of either standard osteotomy or DO techniques.* Proponents of DO techniques frequently state that, when more than 10 mm of horizontal maxillary advancement are required, the use of standard osteotomies with plate and screw fixation and bone grafting may lead to a greater degree of relapse. However, these studies do not report convincing data that demonstrate significant differences in relapse patterns between the two techniques. To give this situation some perspective, it should be noted that only 5% of patients with clefting who are undergoing Le Fort I advancement will require more than 10 mm of horizontal advancement at the incisors. All clinicians agree that this subgroup of patients is the most challenging to treat, but the reasons for this go beyond the degree of horizontal maxillary deficiency. These patients are also likely to present with multiple residual end-stage deformities (see the section about residual skeletal deformities earlier in this chapter), multiple missing teeth, and previously failed surgical procedures.

Aksu and colleagues documented a horizontal maxillary (skeletal) relapse of 22% in patients with cleft lips and palates after Le Fort I osteotomy with the use of DO techniques.[4] He and colleagues reported their results for adolescent patients with repaired cleft lips and palates and maxillary hypoplasia who then underwent Le Fort I advancement with the use of an external DO device.[65] The patients (n = 17) were treated at one center between 2000 and 2006, and they had at least 1 year of follow up and a full set of records. DO treatment was started on day 5 (1 mm per day) and continued until a Class II occlusion (i.e., overcorrection) was achieved. Consolidation time ranged from 4 to 12 weeks, and this was followed by several more months of face mask therapy. The first four patients (two UCLP and two BCLP) who were treated developed fibrous non-union, and all four required reoperation and rigid (i.e., titanium plate and screw) fixation to achieve union. The authors then extended the consolidation period to a minimum of 12 weeks for the remaining patients (n = 13); all of these patients achieved bony union. In the group that achieved satisfactory bony union (13 out of 17 patients; 76%), the mean horizontal relapse was 11.9%, with 5 of 13 patients (38%) developing no better than end-to-end occlusion. Another orthognathic procedure was necessary in 5 of the 13 patients (38%) who initially achieved bony union to obtain a satisfactory occlusion. Overall, 9 out of 17 patients (53%) required two orthognathic procedures. In 2011, **Chen and colleagues** reported a 30.7% incidence of horizontal skeletal relapse 1 year after Le Fort I osteotomy in

*References 3, 11-13, 18, 21-24, 27, 35-37, 40, 41, 43, 46, 50, 55, 59, 61, 65-67, 71-73, 81, 107, 127, 129-142, 149, 160, 162, 168, 173, 179

patients with clefts who were treated with DO techniques (i.e., the RED device). This was in a consecutive series of patients who presented with Class III malocclusion as a result of cleft maxillary hypoplasia and who agreed to a combined orthodontic and surgical approach.[21] **Posnick and colleagues** documented the degree of horizontal relapse and overall occlusal success when using the modified Le Fort I technique as described in this chapter for patients with clefts. They measured horizontal change, stability, and information concerning the maintenance of overjet and overbite from 1-year or more postoperative cephalometric radiographs and clinical examinations.[141] The results were reported in accordance with cleft type: patients with UCLP had 6.9 mm mean advancement with 5.3 mm maintained; 94% of patients with BCLP maintained positive overjet for the long term; and patients with isolate cleft palate had 6.1 mm mean advancement with 5.1 mm maintained. Interestingly, the degree of relapse that was documented was less than that generally reported by clinicians who use DO approaches (see earlier).

Standard Approach versus Distraction Osteogenesis Approach to Le Fort I Advancement

As Obwegeser stated in the 1960s and as was confirmed by Precious and colleagues, "relapse in cleft patients [after Le Fort I advancement] may well be more related to failure to adequately mobilize the maxilla and free it of abnormal soft tissue attachments than anything inherent in the osteotomy or the specific [cleft] diagnosis."[113-117,119,120]

I agree with Obwegeser when he stated the following: "Today, many surgeons will resort to the use of distraction devices to gradually advance the cleft maxilla. Although in some circumstances this may be appropriate, it should be remembered that most cleft patients can be treated efficiently, even when requiring significant advancement with the classic Le Fort I type procedure."[119]

Standard thinking is for the surgeon to commit to an approach (DO versus standard technique) for Le Fort I advancement before he or she arrives in the operating room. The surgeon then sticks to the plan, no matter what happens intraoperatively. On the basis of a review of the literature and almost three decades of personal experience as a cleft jaw surgeon, I make the following observations and recommendations in this area.

Observations About the Distraction Osteogenesis Approach

- DO techniques are always the patient's second choice when compared with the standard approach of Le Fort I osteotomy with rigid (plate and screw) internal fixation. The length of convalescence after surgery involving DO is longer; it will include at least 3 months of limited diet and physical activities, and it is generally followed by several additional months of face mask therapy. The DO device is also awkward and socially embarrassing, as it typically blocks the patient's visual fields (e.g., the RED, BLUE, or GREEN external DO devices). Furthermore, there is no overall reduction in the potential for perioperative complications with DO techniques.
- If the cleft maxilla is adequately down-fractured, mobilized, and stabilized with interpositional bone grafts and rigid plate and screw fixation, it has a high probability of healing as planned with a predictable and less extensive convalescence than would be necessary with DO.
- An experienced orthognathic surgeon will be more confident with his or her ability to down-fracture and fully mobilize the cleft maxilla than a less experienced surgeon. Therefore, the less experienced surgeon will more likely select DO techniques to avoid the personal stress of the intraoperative maxillary mobilization process.
- Even for the experienced cleft orthognathic surgeon, there will be the occasional patient in whom the extent of maxillary hypoplasia and associated deformities (e.g., missing teeth, lack of alveolar bone) will lead him or her to choose a DO technique. Despite its protracted healing requirements and its limited ability to correct all of the cleft deformities, the DO device's gradual stretching ability can mobilize even the most recalcitrant maxilla (see Fig. 32-21).

Recommendations for the Distraction Osteogenesis Approach

For most cleft patients with maxillary hypoplasia, reserving DO techniques to serve as a "bail out" when the down-fractured maxilla cannot be adequately mobilized is the approach that I recommend. The rare patient and family at risk can be informed of this contingency plan in advance. This approach allows the surgeon to have flexibility in the operating room to make the right decision at the right time for the patient in need to achieve the most efficient convalescence and the most favorable long-term objectives.

Conclusions

There are now convincing clinical studies that document a high prevalence of jaw deformities in mature patients with repaired UCLP. The methods described to manage the adolescent and adult with UCLP who presents with jaw deformities, malocclusion, a residual oronasal fistula, bony defects, a cleft–dental gap, nasal obstruction, sleep apnea, and aesthetic needs are generally safe and reliable when performed by a dedicated orthognathic surgeon and team. Successful cleft orthognathic and intranasal procedures provide a stable foundation on which final soft-tissue lip and nose reconstruction may be carried out.

References

1. Abyholm F, Bergland O, Semb G: Secondary bone grafting of alveolar clefts. *Scand J Plast Reconstr Surg* 15:127, 1981.
2. Al-Waheidi EMH, Harradine NWT, Orth M: Soft tissue profile changes in patients with cleft lip and palate following maxillary osteotomies. *Cleft Palate Craniofac J* 35:535–543, 1998.
3. Araujo A, Schendel SA, Wolfort, LM, Epker BN: Total maxillary advancement with and without bone grafting. *J Oral Surg* 36:849–858, 1978.
4. Aksu M, Saglam-Aydinatay B, Akcan CA, et al: Skeletal and dental stability after maxillary distraction with a rigid external device in adult cleft lip and palate patients. *J Oral Maxillofac Surg* 68:254–259, 2010.
5. Bachmayer DI, Ross RB, Munro IR: Maxillary growth following Le Fort III advancement surgery in Crouzon, Apert, and Pfeiffer syndromes. *Am J Orthod Dentofacial Orthop* 90:420, 1986.
6. Bell WH, Levy BM: Revascularization and bone healing after posterior maxillary osteotomy. *J Oral Surg* 29:313, 1971.
7. Bell WH, You ZH, Finn RA, et al: Wound healing after multisegmental Le Fort I osteotomy and transection of the descending palatine vessels. *J Oral Maxillofac Surg* 53:1425, 1995.
8. Berkowitz S: State of the art in cleft palate orofacial growth and dentistry: a historical perspective. *Am J Orthod* 74:564–576, 1978.
9. Berkowitz S, Mejia M, Bystrik A: A comparison of the effects of the Latham-Millard procedure with those of a conservative treatment approach for dental occlusion and facial aesthetics in unilateral and bilateral complete cleft lip and palate: part 1. Dental occlusion. *Plast Reconstr Surg* 113:1–18, 2004.
10. Boyne PJ, Sands NR: Secondary bone grafting of residual alveolar and palatal clefts. *J Oral Surg* 30:87, 1972.
11. Braun TW: (Discussion of) Modification of the maxillary Le Fort I osteotomy in cleft-orthognathic surgery: The unilateral cleft lip and palate deformity. *J Oral Maxillofac Surg* 50:675, 1992.
12. Braun TW, Sotereanos GC: Orthognathic and secondary cleft reconstruction of adolescent patients with cleft palate. *J Oral Surg* 38:425, 1980.
13. Braun TW, Sotereanos GC: Orthognathic surgical reconstruction of cleft palate deformities in adolescents. *J Oral Surg* 39:255, 1981.
14. Canady JW, Thompson SA, Colburn A: Craniofacial growth after iatrogenic cleft palate repair in a fetal bovine model. *Cleft Palate Craniofac J* 34:69, 1997.
15. Capelozza Filho L, Normando AD, da Silva Filho OG: Isolated influences of lip and palate surgery on facial growth: Comparison of operated and unoperated male adults with UCLP. *Cleft Palate Craniofac J* 33:51–56, 1996.
16. Case CS: *A practical treatise on the techniques and principles of dental orthopedia and prosthetic correction of cleft palate*, ed 2, Chicago, 1922, The C.S. Case Co.
17. Cassolato SF, Ross B, Daskalogiannakis J, et al: Treatment of dental anomalies in children with complete unilateral cleft lip and palate at SickKids Hospital, Toronto. *Cleft Palate Craniofac J* 46 (2): 166–172, 2009.
18. Champy M: Surgical treatment of midface deformities. *Head Neck Surg* 2:451, 1980.
19. Chanchareonsook N, Samman N, Whitehill TL: The effect of cranio-maxillofacial osteotomies and distraction osteogenesis on speech and velopharyngeal status: A critical review. *Cleft Palate Craniofac J* 43:477–487, 2006.
20. Chanchareonsook N, Whitehill TL, Samman N: Speech outcome and velopharyngeal function in cleft palate: Comparison of Le Fort I maxillary osteotomy and distraction osteogenesis—early results. *Cleft Palate Craniofac J* 44:23–32, 2007.
21. Chen PK, Por YC, Liou EJ, Chang FC: Maxillary distraction osteogenesis in the adolescent cleft patient: Three-dimensional computed tomography analysis of linear and volumetric changes over five years. *Cleft Palate Craniofac J* 40:445–454, 2011.
22. Cheung LK, Chua HD: A meta-analysis of cleft maxillary osteotomy and distraction osteogenesis. *Int J Oral Maxillofac Surg* 35:14, 2006.
23. Cho BC, Kyung HM: Distraction osteogenesis of the hypoplastic midface using a rigid external distraction system: The results of a one- to six-year follow-up. *Plast Reconstr Surg* 118:1201, 2006.
24. Cohen SR, Burnstein FD, Stewart MB, Rathburn MA: Maxillary-midface distraction in children with cleft lip and palate: A preliminary report. *Plast Reconstr Surg* 99:1421–1428, 1997.
25. Cohen SR, Corrigan M, Wilmot J, Trotman CA: Cumulative operative procedures in patients aged 14 years and older with unilateral or bilateral cleft lip and palate. *Plast Reconstr Surg* 96:267–271, 1995.
26. Correa Normando AD, da Silva Filho OG, Capelozza Filho L: Influence of surgery on maxillary growth in cleft lip and/or palate patients. *J Craniomaxillofac Surg* 20:111, 1992.
27. Daskalogiannakis J, Mehta M: The need for orthognathic surgery in patients with repaired complete unilateral and complete bilateral cleft lip and palate. *Cleft Palate Craniofac J* 46:498–502, 2009.
28. Daskalogiannakis J, Ross RB: Effect of alveolar bone grafting in the mixed dentition on maxillary growth in complete unilateral cleft lip and palate patients. *Cleft Palate Craniofac J* 34:455, 1997.
29. DeLuke DM, Marchand A, Robles EC, Fox P: Facial growth and the need for orthognathic surgery after cleft palate repair: Literature review and report of 28 cases. *J Oral Maxillofac Surg* 55:694–698, 1997.
30. Des Prez JD, Kiehn CL: Surgical positioning of the maxilla: Symposium on management of cleft lip and palate and associated deformities. *Ann Plast Reconstr Surg* 8:222, 1974.
31. Dodson TB, Neuenschwander MC, Bays RA: Intraoperative assessment of maxillary perfusion during Le Fort I osteotomy. *J Oral Maxillofac Surg* 52:827, 1994.
32. Dodson TB, Neuenschwander MC: Maxillary perfusion during Le Fort I osteotomy after ligation of the descending palatine artery. *J Oral Maxillofac Surg* 55:51, 1997.
33. Drommer R: Selecting angiographic studies prior to Le Fort I osteotomy in patients with cleft lip and palate. *J Maxillofac Surg* 7:264, 1979.
34. Drommer R: The history of the "Le Fort I osteotomy." *J Maxillofac Surg* 14:119–122, 1986.
35. Drommer R, Luhr HG: The stabilization of osteotomized maxillary segments with Luhr miniplates in secondary cleft surgery. *J Maxillofac Surg* 9:166–169, 1981.
36. Erbe M, Stoelinga PJW, Leenen RJ: Long-term results of segmental repositioning of the maxilla in cleft palate patients without previously grafted alveolo-palatal clefts. *J Craniomaxillofac Surg* 24:109–117, 1996.
37. Escenazi LB, Schendel SA: An analysis of Le Fort I maxillary advancement in cleft lip and palate patients. *Plast Reconstr Surg* 90:779–786, 1992.
38. Ewing M, Ross RB: Soft tissue response to orthognathic surgery in persons with unilateral cleft lip and palate. *Cleft Palate Craniofac J* 30:320–327, 1993.
39. Felstead AM, Deacon S, Revington P: The outcome for secondary alveolar bone grafting in the southwest UK region post-CSAG. *Cleft Palate Craniofacial* 47:359–362, 2010.
40. Figueroa AA, Polley JW, Friede H, et al: Long-term skeletal stability after maxillary advancement with distraction osteogenesis using a rigid external distraction device in cleft maxillary deformities. *Plast Reconstr Surg* 114:1382, 2004.
41. Figueroa AA, Polley JW, Ko EW: Maxillary distraction for the management of cleft maxillary hypoplasia with a rigid external distraction system. *Semin Orthod* 5:46, 1999.
42. Filho LC: Isolated influences of lip and palate surgery on facial growth: Comparison of operated and unoperated male adults. *Cleft Palate Craniofac J* 33:51, 1996.

43. Fitzpatrick B: Midface osteotomy in the adolescent cleft patient. *Aust Dent J* 22:338, 1977.
44. Freihofer HPM, Jr: The lip profile after correction of retro-maxillism in cleft and non-cleft patients. *J Maxillofac Surg* 4:136–141, 1976.
45. Freihofer HPM, Jr: Changes in nasal profile after maxillary advancement in cleft and non-cleft patients. *J Maxillofac Surg* 5:20–27, 1977.
46. Freihofer HPM, Jr: Results of osteotomies of the facial skeleton in adolescence. *J Maxillofac Surg* 5:267, 1977.
47. Friede H, Lilja J: Dentofacial morphology in adolescent or early adult patients with cleft lip and palate after a treatment regime that included vomer flap surgery and pushback palate repair. *Scand J Plast Reconstr Hand Surg* 28:113–121, 1994.
48. Fudalej P, Hortis-Dzierzbicka M, Dudkiewicz Z, Semb G: Dental arch relationship in children with complete unilateral cleft lip and palate following Warsaw (one-stage repair) and Oslo protocols. *Cleft Palate Craniofac J* 46:648–653, 2009.
49. Fudalej P, Hortis-Dzierzbicka M, Obloj B, et al: Treatment outcome after one-stage repair in children with complete unilateral cleft lip and palate assessed with the Goslon Yardstick. *Cleft Palate Craniofac J* 46:374–380, 2009.
50. Garrison BT, Lapp TH, Bussard DA: The stability of the Le Fort I maxillary osteotomies in patients with simultaneous alveolar cleft bone grafts. *J Oral Maxillofac Surg* 45:761, 1987.
51. Georgiade NG: Mandibular osteotomy for the correction of facial disproportion in the cleft lip and palate patient. Symposium on management of cleft lip and palate and associated deformities. *Plast Reconstr Surg* 8:238, 1974.
52. Gillies HD, Millard DR, Jr: *The principles and art of plastic surgery*, Boston, 1957, Little, Brown.
53. Gillies HD, Rowe NL: L'ostéotomie du maxillaire supérieur enoisagée essentiellement dans le cas de bec-de-lièvre total. *Rev Stomatol* 55:545–552, 1954.
54. Gnoinski W: Early identification of candidates for corrective maxillary osteotomy in cleft lip and palate group. *Scand J Plast Reconstr Surg* 21:39, 1987.
55. Good PM, Mulliken JB, Padwa BL: Frequency of Le Fort I osteotomy after repaired cleft lip and palate or cleft palate. *Cleft Palate Craniofac J* 44:396–401, 2007.
56. Grayson BH, Cutting CB: Presurgical nasoalveolar orthopedic molding in primary correction of the nose, lip, and alveolus of infants bone with unilateral and bilateral clefts. *Cleft Palate Craniofac J* 38:193–198, 2011
57. Guyette TW, Polley JW, Figueroa A, Smith BE: Changes in speech following maxillary distraction osteogenesis. *Cleft Palate Craniofac J* 38:199–205, 2001.
58. Hall HD, Posnick JC: Early results of secondary bone grafts in 106 alveolar clefts. *J Oral Maxillofac Surg* 41:289, 1984.
59. Harada K, Baba Y, Ohyama K, et al: Maxillary distraction osteogenesis for cleft lip and palate children using an external, adjustable, rigid distraction device: A report of 2 cases. *J Oral Maxillofac Surg* 59:1492, 2001.
60. Harada K, Ishii Y, Ishii M, et al: Effect of maxillary distraction osteogenesis on velopharyngeal function: A pilot study. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 93:538, 2002.
61. Harada K, Sato M, Omura K: Long-term maxillomandibular skeletal and dental changes in children with cleft lip and palate after maxillary distraction. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 102:292, 2006.
62. Harrison JW: Dental implants to rehabilitate a patient with an unrepaired complete cleft. *Cleft Palate Craniofac J* 29:485, 1992.
63. Hathaway R, Daskalogiannakis J, Mercado A, et al: The Americleft study: An inter-center study of treatment outcomes for patients with unilateral cleft lip and palate part 2. Dental arch relationship. *Cleft Palate Craniofac J* 48:244–251, 2011.
64. Hathaway RR, Eppley BLE, Hennon DK, et al: Primary alveolar cleft bone grafting in UCLP: Arch dimensions at age 8. *J Craniofac Surg* 10:58–67, 1999.
65. He Dongmei, Genecov DG, Barcelo R: Nonunion of the external maxillary distraction in cleft lip and palate: Analysis of possible reasons. *J Oral Maxillofac Surg* 68:2402–2411, 2010.
66. Hedemark A, Freihofer HP, Jr: The behavior of the maxilla in vertical movements after Le Fort I osteotomy. *J Maxillofac Surg* 6:244, 1978.
67. Heliövaara A, Ranta R, Hukki J, Rintala A: Skeletal stability of Le Fort I osteotomy in patients with unilateral cleft lip and palate. *Scand J Plast Reconstr Surg Hand Surg* 35:43–49, 2001.
68. Heliövaara A, Hukki J, Ranta R, et al: Soft tissue profile changes after Le Fort I osteotomy in UCLP patients. *J Craniomaxillofac Surg* 28:25–30, 2000.
69. Henderson D, Jackson IT: Combined cleft lip revision, anterior fistula closure and maxillary osteotomy: A one-stage procedure. *Br J Oral Surg* 13:33, 1975.
70. Henkel KO, Gundlach KKH: Analysis of primary gingivoperiosteoplasty in alveolar cleft repair. Part I: facial growth. *J Craniomaxillofac Surg* 25:266–269, 1997.
71. Hirano A, Suzuki H: Factors related to relapse after Le Fort I maxillary advancement osteotomy in patients with cleft lip and palate. *Cleft Palate Craniofac J* 38:1–10, 2001.
72. Hochban W, Ganss C, Austermann KH: Long-term results after maxillary advancement in patients with clefts. *Cleft Palate Craniofac J* 30:237–243, 1993.
73. Houston WJB, James DR, Jones E, Kavvadia S: Le Fort I maxillary osteotomies in cleft palate cases. Surgical changes and stability. *J Craniomaxillofac Surg* 17:9–15, 1989.
74. Hsieh CH, Ko EW, Chen PK, Huang CS: The effects of gingivoperiosteoplasty on facial growth in patients with complete unilateral cleft lip and palate. *Cleft Palate Craniofac J* 47:439–446, 2010.
75. Huang CS, Harikrishnan P, Liao YF, et al: Long-term follow-up after maxillary distraction osteogenesis in growing children with cleft lip and palate. *Cleft Palate Craniofac J* 44:274, 2007.
76. Hui E, Hägg EU, Tideman H: Soft tissue changes following maxillary osteotomies in cleft lip and palate and non-cleft patients. *J Craniomaxillofac Surg* 22:182–186, 1994.
77. Jackson IT: Cleft and jaw deformities. In *Symposium on Reconstruction of Jaw Deformities*, 1978, C.V. Mosby Co., p 113.
78. James D, Brook K: Maxillary hypoplasia in patients with cleft lip and palate deformity—the alternative surgical approach. *Eur J Orthop* 7:231, 1985.
79. Janulewicz J, Costello BJ, Buckley MJ, et al: The effects of Le Fort I osteotomies on velopharyngeal and speech functions in cleft patients. *J Oral Maxillofac Surg* 62:308–314, 2004.
80. Jorgenson RJ, Shapiro SD, Odiner KL: Studies on facial growth and arch size in cleft lip and plate. *J Craniofac Genet Dev Biol* 4:33, 1984.
81. Kanno T, Mitsugi M, Hosoe M, et al: Long-term skeletal stability after maxillary advancement with distraction osteogenesis in nongrowing patients. *J Oral Maxillofac Surg* 66:1833–1846, 2008.
82. Kapp-Simon KA: Psychological interventions for the adolescent with cleft lip and palate. *Cleft Palate Craniofac J* 32:104, 1995.
83. Kazanjian VH: Remembrance of things past. *Plast Reconstr Surg* 35:(1)5–13, 1965.
84. Kiehn CL, DesPrez JD, Brown F: Maxillary osteotomy for late correction of occlusion and appearance in cleft lip and palate patients. *Plast Reconstr Surg* 42:203, 1968.
85. Kingsley NW: *A treatise on oral deformities (as a branch of mechanical surgery)*, New York, 1880, Appleton & Co.
86. Ko EW, Figueroa AA, Guyette TW, et al: Velopharyngeal changes after maxillary advancement in cleft patients with distraction osteogenesis using a rigid external distraction device: 1-year cephalometric follow-up. *J Craniofac Surg* 10:312–320, 1999.
87. Lanigan DT: Wound healing after multisegmental Le Fort I osteotomy and transection of the descending palatine vessels [discussion]. *J Oral Maxillofac Surg* 53:1433, 1995.
88. Laspos CP, Kyrkanides S, Moss ME, et al: Mandibular and maxillary asymmetry in

individuals with unilateral cleft lip and palate. *Cleft Palate Craniofac J* 34:232, 1997.
89. Laspos CP, Kyrkanides S, Tallents RH, et al: Mandibular asymmetry in noncleft and unilateral cleft lip and palate individuals. *Cleft Palate Craniofac J* 34:410, 1997.
90. Leonard BJ, Brust JD, Abrahams G, et al: Self-concept of children and adolescents with cleft lip and/or palate. *Cleft Palate Craniofac J* 28:347, 1991.
91. Linton JL: Comparative study of diagnostic measures in borderline surgical cases of unilateral cleft lip and palate and noncleft class III malocclusions. *Am J Orthod Dentofacial Orthop* 113:526–537, 1998.
92. Luhr HG: Zur stabilen osteosynthese bei unterkiefer-frakturen. *Dtsch Zahnarztl Z* 23:754, 1968.
93. Lund TW, Wade M: Use of osseointegrated implants to support a maxillary denture for a patient with repaired cleft lip and palate. *Cleft Palate Craniofac J* 30:418, 1993.
94. Mansour S, Burstone C, Legan H: An evaluation of soft-tissue changes resulting from Le Fort I maxillary surgery. *Am J Orthod* 84:37–47, 1983
95. Marrinan EM, LaBrie RA, Mulliken JB: Velopharyngeal function in nonsyndromic cleft palate: Relevance of surgical technique, age at repair, and cleft type. *Cleft Palate Craniofac J* 35:95–100, 1998.
96. Mars M, Asher-McDade C, Brattström V, et al: A six-center international study of treatment outcomes in patients with clefts of the lip and palate. Part 3: Dental arch relationships. *Cleft Palate Craniofac J* 29:405–408, 1992.
97. Mars M, Houston WJB: A preliminary study of facial growth and morphology in unoperated male unilateral cleft lip and palate subjects over 13 years of age. *Cleft Palate J* 27:7–10, 1990.
98. Mars M, Plint DA, Houston WJB, et al. The Goslon Yardstick: A new system of assessing dental arch relationships in children with unilateral clefts of the lip and palate. *Cleft Palate J* 24:314–322, 1987.
99. Matic DB, Power SM: The effects of gingivoperiosteoplasty following alveolar molding with a pun-retained Latham appliance versus secondary bone grafting on midfacial growth in patients with unilateral clefts. *Plast Reconstr Surg* 122:863–870, 2008
100. McCance AM, Moss JP, Fright WR, et al: Three-dimensional analysis techniques. Part 1: Three-dimensional soft-tissue analysis of 24 adult cleft palate patients following Le Fort I maxillary advancement: A preliminary report. *Cleft Palate Craniofac J* 34:36, 1997.
101. McCance AM, Orth M, Moss JP et al: Three-dimensional analysis techniques. Part 4: Three-dimensional analysis of bone, and soft tissue to bone ratio of movements in 24 cleft patients following Le Fort I osteotomy: A preliminary report. *Cleft Palate Craniofac J* 43:58–62, 1997.
102. McComb R, Marrinan E, Nuss RC, et al: Predictors of velopharyngeal insufficiency after Le Fort I maxillary advancement in patients with cleft palate. *J Oral Maxillofac Surg* 69:2226–2232, 2011.
103. McIntyre GT, Devlin MF: Secondary alveolar bone grafting (CLEFTSiS) 2000-2004. *Cleft Palate Craniofac J* 47:66–72, 2010.
104. McKinstry RE: *Cleft palate dental care: A historical perspective*, Arlington, Va, 2000, ABI Publications.
105. Millard DR, Jr, Latham RA: Improved primary surgical and dental treatment of clefts. *Plast Reconstr Surg* 86:856–871, 1990
106. Millard DR, Latham RA, Huifen X, et al: Cleft lip and palate treated by presurgical orthopedics, gingivoperiosteoplasty, and lip adhesion (POPLA) compared with previous lip adhesion method: a preliminary study of serial dental casts. *Plast Reconstr Surg* 103:1630–1644, 1999.
107. Molina F, Ortiz Monasterio F, de la Paz Aguilar M, Barrera J: Maxillary distraction: Aesthetic and functional benefits in cleft lip-palate and prognathic patients during mixed dentition. *Plast Reconstr Surg* 101:951, 1998.
108. Mølsted K, Brattström V, Prahl-Andersen B, et al: The Eurocleft study: Intercenter study of treatment outcomes in patients with complete cleft lip and palate. Part 3: Dental arch relationships. *Cleft Palate Craniofac J* 42:78–82, 2005.
109. Motohashi N, Kuroda T, Filho LC, et al: P-A cephalometric analysis of nonoperated adult cleft lip and palate. *Cleft Palate Craniofac J* 31:193, 1994.
110. Nanda SK: Patterns of vertical growth in the face. *Am J Orthod Dentofacial Orthop* 93:103–116, 1988.
111. Nollet PJPM, Katsaros C, van't Hof MA, et al: Treatment outcome after two-stage palatal closure in unilateral cleft lip and palate: A comparison with Eurocleft. *Cleft Palate Craniofac J* 42:512–516, 2005.
112. Nordquist GG, McNeill RW: Orthodontic vs restorative treatment of the congenitally absent lateral incisor—long-term periodontal and occlusal evaluation. *J Periodontol* 46:139–143, 1975.
113. Obwegeser HL: *Correction of the facial appearance of harelip and cleft palate patients by surgery on the jaws*. Exerpta Medica International Congress Series No 141. Reconstructive surgery. Thermal injury and other subjects, pp 110–117, 1966.
114. Obwegeser HL: Surgery as an adjunct to orthodontics in normal and cleft palate patients. *Trans Eur Orthod Soc* 42:343–353, 1967.
115. Obwegeser HL: *Surgical correction of deformities of the jaws in adult cleft cases.* Paper read at the First International Conference on Cleft Lip and Palate, Houston, Tex, April 14-17, 1969.
116. Obwegeser HL: Surgical correction of small or retrodisplaced maxillae: The "dish-face" deformity. *Plast Reconstr Surg* 43:351, 1969.
117. Obwegeser HL: Surgical correction of maxillary deformities. In Grabb WC, Rosenstein SW, Bzoch KR, editors: *Cleft lip and palate*, Boston, 1971, Little and Crown, pp 515–556.
118. Obwegeser HL: Chirurgische behandlungsmoglichkeiten von sekundardeformierungen bei spaltpatienten. *Fortschr. Kieferorthop* 49:272–296, 1988.
119. Obwegeser HL: Orthognathic surgery and a tale of how three procedures came to be: A letter to the next generations of surgeons. *Clin Plast Surg* 34:331–355, 2007.
120. Obwegeser HL, Lello GE, Farmand M: Correction of secondary cleft deformities. In Bell WH, editor: *Surgical correction of dentofacial deformities. New Concepts,* Vol III, Vol 13, Philadelphia, 1985, Saunders, pp 592–638.
121. Palmer CR, Hamlen M, Ross RB, Lindsay WK: Cleft palate repair: Comparison of the results of two surgical techniques. *Can J Surg* 12:32–67, 1969.
122. Parel SM, Branemark PI, Jansson T: Osseointegration in maxillofacial prosthetics: Part I: Intraoral applications. *J Prosthet Dent* 55:490, 1986.
123. Perko M: The history of treatment of cleft lip and palate. *Prog Pediatr Surg* 20:239–248, 1986.
124. Perrott D, Sharma AB, Vargevik K: Endosseous implants for pediatric patients: Unknown factors, indications, contraindications, and special considerations. *Oral Maxillofac Surg Clin North Am* 6:79, 1994.
125. Phillips JH, Klaiman P, Delorey R, MacDonald DB: Predictors of velopharyngeal insufficiency in cleft palate orthognathic surgery. *Plast Reconstr Surg* 115:681–686, 2005.
126. Polley JW, Figueroa AA: Management of severe maxillary deficiency in childhood and adolescence through distraction osteogenesis with an external, adjustable, rigid distraction device. *J Craniofac Surg* 8:181, 1997.
127. Polley JW, Figueroa AA: Rigid external distraction: Its application in cleft maxillary deformities. *Plast Reconstr Surg* 102:1360, 1998.
128. Poole MD, Robinson PP, Nunn ME: Maxillary advancement in cleft lip and palate patients: A modification of the Le Fort I osteotomy and preliminary results. *J Maxillofac Surg* 14:123, 1986.
129. Posnick JC: (Discussion of) Orthognathic surgery in cleft patients treated by early bone grafting. *Plast Reconstr Surg* 87:840, 1991.
130. Posnick JC: Orthognathic surgery in the cleft patient. In Russel RC, editor: *Instructional courses, plastic surgery education foundation,* Vol 4, St Louis, Mo, 1991, CV Mosby Co, pp 129–157,
131. Posnick JC: Orthognathic surgery for the cleft lip and palate patient. *Semin Orthod* 2(3):205–214, 1996.
132. Posnick JC: The treatment of secondary and residual dentofacial deformities in the cleft

patient. Surgical and orthodontic therapy. *Clin Plast Surg* 24(3):583–597, 1997.
133. Posnick JC: Cleft lip and palate: Bone grafting and management of residual oro-nasal fistula. In Posnick JC, editor: *Craniofacial and maxillofacial surgery in children and young adults,* Vol 33, Philadelphia, 2000, WB Saunders Co, pp 827–859.
134. Posnick JC: Cleft-orthognathic surgery: The unilateral cleft lip and palate deformity. In Posnick JC, editor: *Craniofacial and maxillofacial surgery in children and young adults,* Vol 34, Philadelphia, 2000, WB Saunders Co, pp 860–907.
135. Posnick JC: The staging of cleft lip and palate reconstruction: Infancy through adolescence. In Posnick JC, editor: *Craniofacial and maxillofacial surgery in children and young adults,* Vol 32, Philadelphia, 2000, WB Saunders Co, pp 785–826.
136. Posnick JC, Agnihotri N: Managing chronic nasal airway obstruction at the time of orthognathic surgery: A twofer. *J Oral Maxillofac Surg* 69:695–701, 2011.
137. Posnick JC, Al-Qattan MM, Pron G: Facial sensibility in cleft and non-cleft adolescents one year after undergoing Le Fort I osteotomy. *Plast Reconstr Surg* 194(3):431–435, 1994.
138. Posnick JC, Dagys AP: Skeletal stability and relapse patterns after Le Fort I maxillary osteotomy fixed with miniplates: The unilateral cleft lip and palate deformity. *Plast Reconstr Surg* 94(7):924–932, 1994.
139. Posnick JC, Ewing MP: Skeletal stability after Le Fort I maxillary advancement in patients with unilateral cleft lip and palate. *Plast Reconstr Surg* 85(5):706–710, 1990.
140. Posnick JC, Tompson B: Modification of the maxillary Le Fort I osteotomy in cleft-orthognathic surgery: The unilateral cleft lip and palate deformity. *J Oral Maxillofac Surg* 50(7):666–675, 1992.
141. Posnick JC, Tompson B: Cleft-orthognathic surgery: Complications and long-term results. *Plast Reconstr Surg* 96(2):255–266, 1995.
142. Posnick JC, Ricalde P: Cleft-orthognathic surgery. *Clin Plast Surg* 31(2):315–330, 2004.
143. Renkielska A, Wojtaszek-Slominska A, Dobke M: Early cleft lip repair in children with unilateral complete cleft lip and palate: a case against primary alveolar repair. *Ann Plast Surg* 54:595–597, 2005.
144. Roberts HG, Semb G, Hathorn I, Killingback N: Facial growth in patients with unilateral clefts of the lip and palate: A two-center study. *Cleft Palate Craniofac J* 31:372–375, 1996.
145. Robertsson S, Mohlin B: The congenitally missing upper lateral incisor: A retrospective study of orthodontic space closure versus restorative treatment. *Eur J Orthod* 22:697–710, 2000.
146. Ronchi P, Chiapasco M, Frattini D: Endosseous implants for prosthetic rehabilitation in bone grafted alveolar clefts. *J Craniomaxillofac Surg* 23:382, 1995.
147. Rosenstein SW: Facial growth and the need for orthognathic surgery after cleft palate repair: Literature review and report of 28 cases [discussion]. *J Oral Maxillofac Surg* 55:698, 1997.
148. Ross BR: Treatment variables affecting facial growth in complete unilateral cleft lip and palate. Part 7: An overview of treatment and facial growth. *Cleft Palate J* 24:71–77, 1987.
149. Samman N, Cheung LK, Tideman H: A comparison of alveolar bone grafting with and without simultaneous maxillary osteotomies in cleft palate patients. *Int J Oral Maxillofac Surg* 23:65–70, 1994.
150. Sandham A, Murray JAM: Nasal septal deformity in unilateral cleft lip and palate. *Cleft Palate Craniofac J* 30:222, 1993.
151. Saperstein EL, Kennedy DL, Mulliken JB, Padwa BL: Facial growth in children with complete cleft of the primary palate and intact secondary palate. *J Oral Maxillofac Surg* 70:e66–e71, 2012.
152. Schnitt DE, Agir H, David DJ: From birth to maturity: A group of patients who have completed their protocol management. Part I. Unilateral cleft lip and palate. *Plast Reconstr Surg* 113:805–817, 2004.
153. Shaw WC, Asher-McDade C, Brattstrom V, et al: A six-center international study of treatment outcomes in patients with clefts of the lip and palate. *Cleft Palate Craniofac J* 29:393, 1992.
154. Sinko K, Caacbay E, Eagsch R, et al: The Goslon Yardstick in patients with unilateral cleft lip and palate: Review of a Vienna sample. *Cleft Palate Craniofac J* 45:87–91, 2008.
155. Sinn DP: Simultaneous maxillary expansion and advancement, repair of oronasal fistula, and bone grafting of the alveolar cleft. In Bell WH, Proffit WR, White RP, editors: *Surgical correction of dentofacial deformities*, Philadelphia, 1980, WB Saunders.
156. Skoog T: The use of periosteal flaps in the repair of cleft of the primary palate. *Cleft Palate J* 2:332–339, 1965.
157. Smabel Z: Treatment effects on facial development in patients with unilateral cleft lip and palate. *Cleft Palate Craniofac J* 31:437, 1994.
158. Steinkamm W: *Die pseudo-progenie und ihre behandlung*. University Berlin, inaugural dissertation, 1938.
159. Stoelinga PJ, Haers PE, Lennen RJ, et al: Late management of secondarily grafted clefts. *Int J Oral Maxillofac Surg* 19:91, 1990.
160. Stoelinga PJ, vd Vijver HR, Leenen RJ, et al: The prevention of relapse after maxillary osteotomies in cleft palate patients. *J Craniomaxillofac Surg* 15:325, 1987.
161. Susami T, Ogihara Y, Matsuzaki M, et al: Assessment of dental arch relationships in Japanese patients with unilateral cleft lip and palate. *Cleft Palate Craniofac J* 43:96–102, 2006.
162. Suzuki EY, Motohashi N, Ohyama K: Longitudinal dento-skeletal changes in UCLP patients following maxillary distraction osteogenesis using RED system. *J Med Dent Sci* 51:27–33, 2004.
163. Suzuki A, Takahama Y: Maxillary lateral incisor of subjects with cleft lip and/or palate: Part 1. *Cleft Palate Craniofac J* 29:376, 1992.
164. Suzuki A, Takahama Y: Maxillary lateral incisor of subjects with cleft lip and/or palate: Part 2. *Cleft Palate Craniofac J* 29:380, 1992.
165. Takahashi T, Fukuda M, Yamaguchi T, et al: Use of an osseointegrated implant for dental rehabilitation after cleft repair by periosteoplasty: A case report. *Cleft Palate Craniofac J* 35:268, 1997.
166. Takahashi T, Fukuda M, Yamaguchi T, et al: Use of endosseous implants for dental reconstruction of patients with grafted alveolar clefts. *J Oral Maxillofac Surg* 55:576, 1997.
167. Tessier P, Tulasne JF: Secondary repair of cleft lip deformity. *Clin Plast Surg* 11:747, 1984.
168. Tideman H, Stoelinga P, Gallia L: Le Fort I advancement with segmental palatal osteotomies in patients with cleft palates. *J Oral Surg* 38:196, 1980.
169. Tomanova M, Mullerova Z: Growth of the dental arch in patients with complete unilateral cleft lip and palate after primary periosteoplasty. *Acta Chir Plast* 36:119–123, 1994.
170. Trindale IE, Yamashita RP, Suguimoto RM, et al: Effects of orthognathic surgery on speech and breathing of subjects with cleft lip and palate: Acoustic and aerodynamic assessment. *Cleft Palate Craniofac J* 40:54–64, 2003.
171. Tulloch JFC: A six-center international study of treatment outcome in patients with clefts of the lip and palate: Evaluation of maxillary asymmetry (commentary). *Cleft Palate Craniofac J* 30:22, 1993.
172. Turvey TA: Use of the Branemark implant in the cleft palate patient. *Cleft Palate Craniofac J* 28:304, 1991.
173. Turvey TA, Vig KWL, Fonseca RJ: Maxillary advancement and contouring in the presence of cleft lip and palate. In Turvey TA, Vig KWL, Fonseca RJ, editors: *Facial clefts and craniosynostosis: Principles and management*, Philadelphia, 1996, Saunders, pp 441–503.
174. Verdi FJ, Jr, Shanzi GL, Cohen SR, et al: Use of the Branemark implant in the cleft palate patient. *Cleft Palate Craniofac J* 28:301, 1991.
175. Ward-Booth RP, Bhatia SN, Moos KF: A cephalometric analysis of the Le Fort II osteotomy in the adult cleft patient. *J Maxillofac Surg* 12:208, 1984.
176. Westbrook MT, Jr, West RA, McNeil RW: Simultaneous maxillary advancement and closure of bilateral alveolar clefts and

oronasal fistulas. *J Oral Maxillofac Surg* 41:257, 1983.
177. Williams AC, Bearn D, Mildinhall S, et al: Cleft lip and palate care in the United Kingdom—the Clinical Standards Advisory Group (CSAG) study. Part 2: Dentofacial outcomes and patient satisfaction. *Cleft Palate Craniofac J* 38:24–29, 2001.
178. Willmar K: On Le Fort I osteotomy: A follow-up study of 106 operated patients with maxillo-facial deformity. *Scand J Plast Reconstr Surg* 12(Suppl 1):1–68, 1974.
179. Wiltfang J, Hirschfelder U, Neukam FW, et al: Long-term results of distraction osteogenesis of the maxilla and midface. *Br J Oral Maxillofac Surg* 40:473, 2002.
180. Witzel MA, Munro IR: Velopharyngeal insufficiency after maxillary advancement. *Cleft Palate J* 14:176, 1977.
181. Wolford LM: Effects of orthognathic surgery on nasal form and function in the cleft patient. *Cleft Palate Craniofac J* 29:546–555, 1992.
182. Wolford LM, Cassano DS, Cottrell DA, et al: Orthognathic surgery in the young cleft patient: Preliminary study on subsequent facial growth. *J Oral Maxillofac Surg* 66:2524–2536, 2008.
183. Wolford LM, Karras SC, Mehra P: Considerations for orthognathic surgery during growth: Part 1. Mandibular deformities. *Am J Orthod Dentofacial Orthop* 119:95, 2001.
184. Wolford LM, Karras SC, Mehra P: Considerations for orthognathic surgery during growth: Part 2. Maxillary deformities. *Am J Orthod Dentofacial Orthop* 119:102, 2001.
185. Zachrisson BU, Stenvik A: Single implants—optimal therapy for missing lateral incisors? *Am J Orthod Dentofacial Orthop* 126:13A–15A, 2004.

33

Cleft–Orthognathic Surgery: The Bilateral Cleft Lip and Palate Deformity

JEFFREY C. POSNICK, DMD, MD

- Facial Growth Implications of Cleft Palate Repair in the Infant with Bilateral Cleft Lip and Palate Deformity
- Coordinated Team Approach
- Treatment Protocol
- Timing of Orthognathic Surgery
- Residual Deformities in the Adolescent with Bilateral Cleft Lip and Palate
- Orthodontic Considerations in the Patient with Bilateral Cleft Lip and Palate with Jaw Deformity
- Immediate Presurgical Assessment
- Orthognathic Approach for Bilateral Cleft Lip and Palate Deformities
- Avoiding Pitfalls
- Clinical Management after Initial Surgical Healing
- Orthognathic Surgery for Bilateral Cleft Lip and Palate: Review of Study
- Controversies and Unresolved Issues
- Conclusions

The reconstruction of bilateral cleft lip and palate is inextricably connected with orthodontic care and skeletal surgery. The successful management of these individuals is likely to present a formidable clinical challenge for the orthognathic surgeon, the orthodontist, and the dental team.

Facial Growth Implications of Cleft Palate Repair in the Infant with Bilateral Cleft Lip and Palate Deformity

Ross confirmed that individuals who are born with cleft lip and palate have intrinsic deficiencies in the midfacial skeleton that are made significantly worse by operations.[118] Mulliken and colleagues from Boston Children's Hospital found that 76.5% of teenagers with repaired bilateral cleft lip palate (BCLP) required maxillary advancement.[43] They showed that the need for orthognathic surgery is dependent on the severity of the cleft type as well as the number and extent of previous operative procedures. Results from The Hospital for Sick Children in Toronto, Canada, showed that 65.1% of their patients with BCLP required or underwent orthognathic surgery, whereas 70% of children with BCLP who were referred to their team some time after cleft lip repair were found to require orthognathic surgery.[23] David and colleagues from the cleft craniofacial unit in Adelaide, Australia, followed a consecutive group of patients with BCLP from birth to maturity and determined the need for orthognathic surgery.[24] Each patient was followed according to treatment protocol and then at 18 years of age assessed for dentofacial deformity. A skeletal Class III malocclusion that required orthognathic correction was present in 17 of the 19 patients (89.5%), whereas the remaining 2 patients were found to have a malocclusion that was manageable with orthodontics alone. Fourteen underwent orthognathic surgery, and the remaining three declined the offer for correction.

Saperstein and colleagues discussed facial growth in children with complete clefting of the primary palate (i.e., the lip through the incisal foramen) but with an intact secondary palate (i.e., the incisal foramen through the uvula).[120] This was a retrospective, cross-sectional analysis of nonsyndromal patients with bilateral complete clefting of the

primary palate (n = 7) as compared with those with bilateral complete clefting of both the primary and secondary palates (n = 27). Angular and linear measurements of the midfacial region were made on lateral cephalograms. The research documented that individuals with a bilateral cleft of the primary palate who underwent lip repair during infancy most frequently have a normal or even slightly forward maxillary position as compared with age-matched controls. This was in contrast with children with a bilateral cleft of both the primary and secondary palates who underwent lip repair followed by palate repair before they were 1 year old, who demonstrated a high incidence of maxillary deficiency. The study clarified that cleft palate repair carried out before the patient is 1 year old—and not the cleft lip repair itself—is responsible for the high incidence of midface hypoplasia.

It is now clear that orthognathic surgery is a necessary component of the treatment of a majority of individuals who are born with BCLP. Unfortunately, the necessary jaw reconstruction is often considered "too risky" and therefore postponed, delayed, ignored, or refused. If so, the patient is likely to be treated with orthodontic and prosthetic camouflage techniques. Adding further difficulty to successful rehabilitation is controversy about the indications and confusion regarding the exact techniques, timing, and extent of surgery needed to achieve reliable and predictable results. The occlusal, speech, upper airway, and aesthetic objectives in the adolescent patient with BCLP must first be clarified. A surgical, orthodontic, and dental treatment plan can then be coordinated to achieve improved head and neck function, facial enhancement, and dental rehabilitation.

Coordinated Team Approach

The care of the patient with BCLP is best delivered by an integrated group of dental and medical specialists who evaluate and then provide coordinated definitive treatment.[8,77,92] It is no longer acceptable for individual surgeons, orthodontists, restorative dentists, speech pathologists, and otolaryngologists to carry out extended treatment without considering all aspects of the patient's care and communicating effectively with the patient, the family, and the collaborating clinicians. The Eurocleft study found no association between high-intensity treatments and favorable results. In other words, the more operations and years of orthodontic appliances (i.e., heavy burden of care), the worse the outcome.[79]

Treatment Protocol

Adolescents and adults with BCLP who also have jaw deformities should be seen at a minimum by an orthodontist, an orthognathic surgeon, an otolaryngologist, and a speech pathologist. Additional consultations with other dental (e.g. a prosthodontist, a pediatric or general dentist, a periodontist) and medical (e.g., sleep specialist, medical geneticist) specialists are obtained, if necessary. For the initial evaluation and eventual treatment of the cleft dentofacial deformity, records and tests should include the following: medical-quality photographs with views of the face and the occlusion; cephalometric and dental radiographs; dental models; direct facial measurements; speech assessment; and thorough evaluation of the upper airway.

In addition to performing lip, palate, and velopharyngeal (VP) repairs, the primary cleft surgeon generally plays a role in advising the patient and the family about the patient's further care. If the cleft surgeon is not trained in skeletal procedures, then a timely and seamless transition to the maxillofacial surgeon who will continue on with the patients' reconstruction should go forward. The cleft jaw surgeon should have a fundamental understanding of the patient's dental, speech, upper airway, and aesthetic needs. The surgeon should request consultation with appropriate specialists, evaluate the clinical information, and be prepared to go forward with coordinated and timely up-to-date orthognathic procedures.

The orthodontist provides interceptive treatment during the mixed dentition in coordination with bone grafting; and carries out definitive orthodontics in conjunction with orthognathic surgery, when indicated. From the mixed-dentition phase, the orthodontist should recognize the patient with BCLP who will likely be in need of orthognathic surgery.[20,21,25,36,52,53,64,80,90,114,117,121,132,145] Instituting extensive camouflage (dental compensatory) treatment is likely to jeopardize periodontal health and lead to late dental relapse of the occlusion. Proceeding with a compromised orthodontic (camouflage) approach should only be entered into with full disclosure to the family and other treating clinicians.

Before orthognathic surgery, a speech pathologist performs an evaluation to characterize VP function and to identify articulation errors that result from the cleft palate, the jaw deformity, and the dental malocclusion (see Chapter 8). Such evaluation is important, because VP function may deteriorate after maxillary advancement. A nasoendoscopic guided speech assessment is preferred to obtain maximum objective data. VP closure that is adequate before surgery may become borderline afterward, and VP closure that is borderline may become inadequate. The successful orthodontic, surgical, and dental correction of crossbites, open bite, cleft–dental gaps, negative overjet, and residual oronasal fistula represents the most effective way to correct the identified articulation distortions (see Chapter 8).

A thorough evaluation of the upper airway is conducted to assess for any areas of obstruction (see Chapter 10). A formal sleep study (polysomnogram) is completed if there is a suspicion of obstructive sleep apnea (see Chapter 26). If indicated, simultaneous intranasal procedures to open the airway should be carried out at the time of orthognathic surgery (see Chapters 10 and 15).

Discussions with the treating medical and dental consultants as well as the patient and his or her family clarify the need for and the extent of orthognathic procedures (e.g., Le Fort I osteotomy, sagittal ramus osteotomies, osseous

genioplasty) and intranasal procedures (e.g., septoplasty, inferior turbinate reduction, recontouring of the nasal floor and rims). The overall plan for speech, jaw, upper airway, and dental rehabilitation and the enhancement of facial aesthetics is agreed to before the initiation of treatment.

Timing of Orthognathic Surgery

Correction of the cleft jaw deformity is best carried out with a consideration of skeletal maturity and before the patient finishes high school.[66,69] Maxillofacial growth is generally complete between the ages of 14 and 16 years in girls and the ages of 16 and 18 years in boys. However, skeletal growth is variable, and it may be further clarified by an analysis of sequential cephalometric radiographs taken at intervals (see Chapter 17). Patient and family preferences for the timing of reconstruction on the basis of the patient's psychosocial and functional needs (i.e., speech, swallowing, chewing, and breathing) should also be taken into account.

As early as 1986, investigators showed that, if Le Fort I advancement is completed during the mixed dentition in a patient with a cleft palate, then another orthognathic procedure will be necessary after skeletal maturity is reached.[140-143] All research to date indicates that a Le Fort I osteotomy carried out at an early age, with the use of either standard or distraction osteogenesis (DO) techniques, results in no further horizontal maxillary growth.[16,34,35,46,48,59,95]

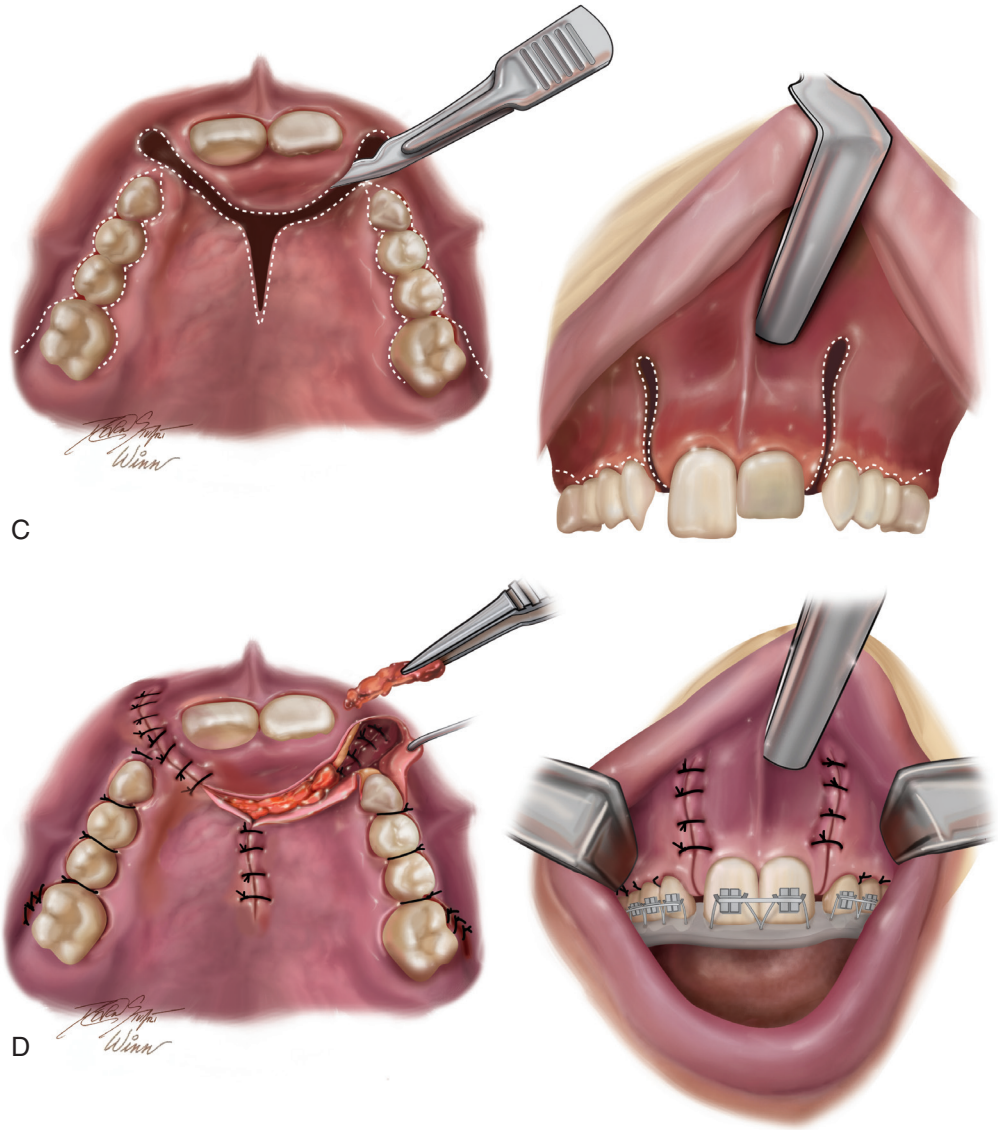
Residual Deformities in the Adolescent with Bilateral Cleft Lip and Palate

The adolescent patient with BCLP often has several residual deformities that can be challenging to manage. The central deformity is maxillary hypoplasia, which is often combined with residual oronasal fistula, bone defects, intranasal obstruction, soft-tissue scarring, and occasionally VP dysfunction (Figs. 33-1 through 33-17). In addition, the maxillary lateral incisors are usually congenitally absent or hypoplastic (93% of the time), and the patients often have cleft–dental gaps.[13] Secondary deformities of the nose, the mandible, and the chin region are also common. The prevalence of these residual deformities in mature patients with BCLP varies widely, depending on the treating clinician's philosophy, available expertise, the individual's intrinsic biologic growth potential, and patient and family interests. Published clinical surveys indicate that, despite a cleft team's best efforts, a significant number of children with BCLP will not make themselves available for mixed-dentition grafting (i.e., before the eruption of the maxillary canines).[33,137] In addition, a percentage of those who do will undergo unsuccessful grafting procedures and require other means of reconstruction and dental rehabilitation. For all of these reasons, a subgroup of individuals with BCLP continues to present at or after adolescence with multiple cleft-related challenges that may include the following:

1. *Maxillary hypoplasia.* The anterior maxilla may be either vertically long, which results in a gummy smile, or vertically short, which leaves an edentulous appearance. Horizontal deficiency (negative overjet) with a flat midface and an Angle class III malocclusion is also part of the usual presentation. The arch width of the lateral segments is frequently deficient (or at least pseudodeficient as a result of the negative overjet), with posterior crossbites. The dental midline may be shifted off of the facial midline, and the maxillary lateral segments may be canted. For the individual who was not effectively grafted during the mixed dentition, the two lateral segments and the premaxillary segments are separate components of the upper jaw. In general, each segment will vary with regard to its degree of vertical and horizontal dysplasia. These patients are best treated via the differential repositioning of each of the three segments to achieve a satisfactory arch form, occlusion, and facial appearance.
2. *Residual oronasal fistula.* Although the preference is to complete fistula closure and bone grafting during the mixed dentition (i.e., before the eruption of the canine at each cleft site), some patients with BCLP will have residual labial and palatal fistulas, with leakage of fluid through the nose while drinking and air leakage while speaking. Previous attempts at fistula closure may have failed as a result of inadequate available (palatal) soft tissue for wound closure in the incisal foramen region.
3. *Cleft–dental gaps.* Approximately 93% of the time, useful lateral incisors are absent or inadequate at each cleft site. A rudimentary lateral incisor or supernumerary tooth may be impacted or adjacent to each cleft; if so, they are of no functional value. If hypoplastic lateral incisors do erupt in the posterior or premaxillary segments, they rarely have the root support needed for long-term function. The result is often a dental gap at the cleft site between the central incisor and the canine on each side. If bone grafting and fistula closure during the mixed dentition were successfully carried out, then canine eruption followed by orthodontic closure of the cleft dental gaps will have likely been undertaken.
4. *Residual bony defects.* In the patient with BCLP who has not had successful or adequate bone grafting during the mixed dentition, significant residual defects through the alveolus, the floor of the nose, and the palate remain. A lack of continuity across the cleft alveolar ridges results in a mobile premaxilla that is only attached to the nasal septum and the labial soft tissues. It is also not uncommon to see only a limited bridge of bone (on one or both sides) connecting the lateral and premaxillary ridge after a previous bone graft. Unfortunately, this is inadequate bone volume into which the canine will erupt. In addition, a limited bone bridge cannot be relied on

Text continued on p. 1428

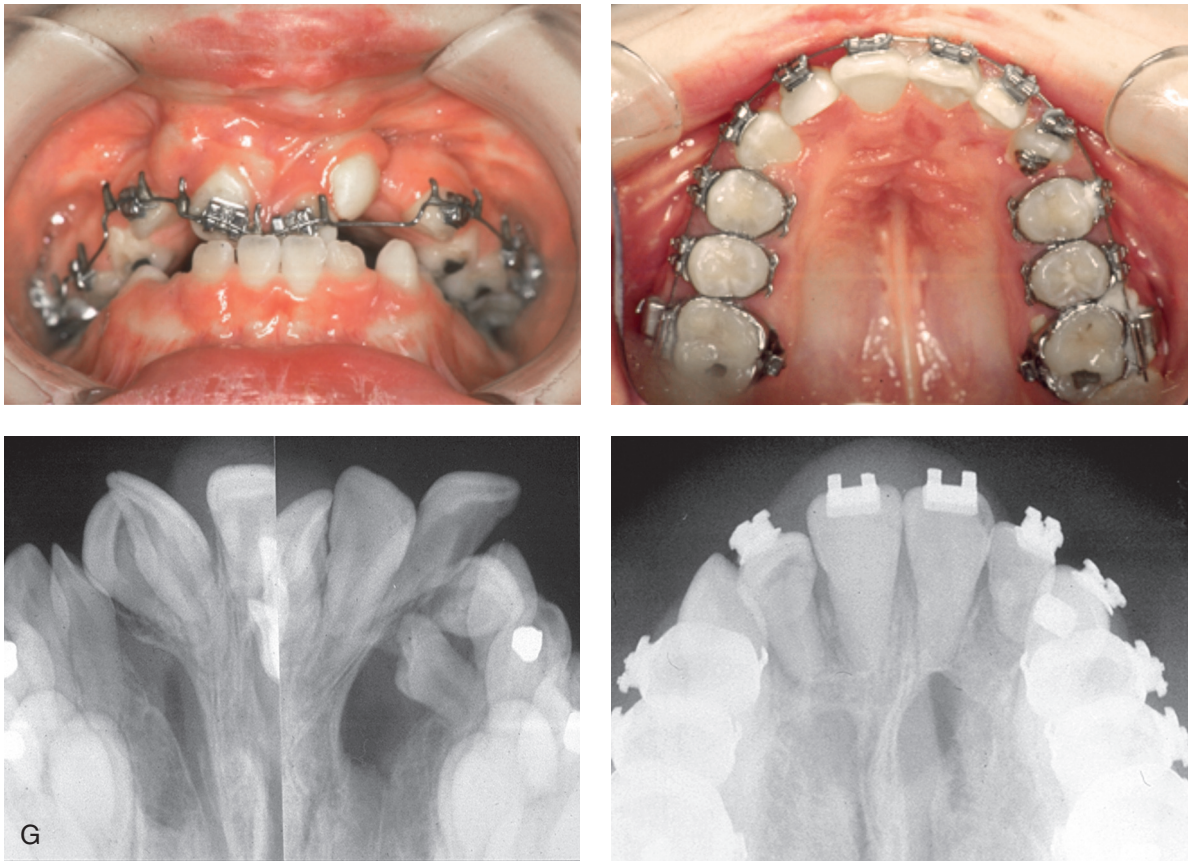
• **Figure 33-1** A child who was born with bilateral cleft lip and palate underwent lip and palate repair during early childhood. During the mixed dentition, she presented to this surgeon with a mobile premaxilla, alveolar and palatal defects, and residual (bilateral) labial and palatal fistulas. She then underwent a short phase of orthodontic treatment to expand the maxillary arch width. This was followed by iliac grafting of the skeletal defects and fistula closure. She was fortunate to have adequately developed lateral incisors. **A,** Facial views during infancy and early childhood. **B,** Facial and occlusal views during the mixed dentition with interceptive orthodontic treatment in progress and before bone grafting.



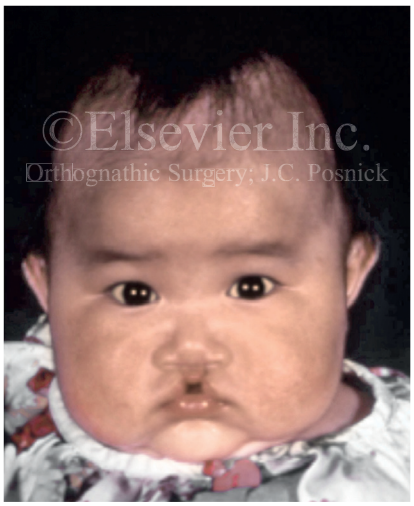
• **Figure 33-1, cont'd C,** Illustration of incisions for the elevation of flaps, fistula closure, and grafting during the mixed dentition in a child with BCLP. Maintenance of the labial mucosa to the premaxilla is essential. **D,** Illustration of flap and wound closure after grafting for fistula closure. The occlusal splint is secured to the maxillary segments to immobilize the premaxilla during initial bone healing. *Parts C, D are modified from an original illustration by Bill Winn.*

Continued

• **Figure 33-1, cont'd E,** Frontal and occlusal views at 14 years of age after successful grafting, fistula closure, and stabilization of the premaxilla, with definitive orthodontic alignment in progress. **F,** Views of the healed skin incision used to harvest the iliac graft.



• **Figure 33-1, cont'd G,** Palatal and radiographic views before and after successful bone grafting and orthodontic alignment.


©Elsevier Inc.
Orthognathic Surgery; J.C. Posnick

©Elsevier Inc.
Orthognathic Surgery; J.C. Posnick

©Elsevier Inc.
Orthognathic Surgery; J.C. Posnick

• **Figure 33-2** A child who was born with bilateral cleft lip and palate and who had intact nasal sills. She was referred to this surgeon and underwent lip and palate repair during infancy. During the mixed dentition, she underwent successful bone grafting with stabilization of the premaxilla. The canines erupted through the grafted cleft sites and established a normal alveolar ridge. The lateral incisors were essentially normal and maintained within the arch form. With significant maxillary hypoplasia, the patient underwent a comprehensive orthodontic and orthognathic approach. Her procedures included Le Fort I osteotomy in three segments (correction of arch width and curve of Spee, horizontal advancement, vertical lengthening) with interpositional grafting; bilateral sagittal split ramus osteotomies (clockwise rotation); and septoplasty, inferior turbinate reduction, and nasal floor recontouring. **A,** Frontal facial views before lip repair, after primary lip and palate repair, and at 8 years of age just before mixed dentition grafting.

• **Figure 33-2, cont'd** **B,** Frontal views in repose as a teenager before and after reconstruction. **C,** Frontal views with smile before and after reconstruction.

Continued

• **Figure 33-2, cont'd** **D,** Oblique facial views before and after reconstruction. **E,** Profile views before and after reconstruction.

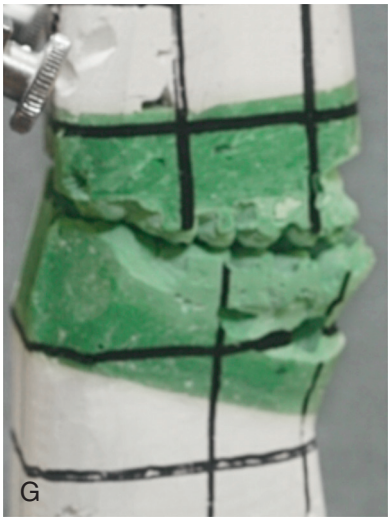
After successful mixed-dentition bone graft

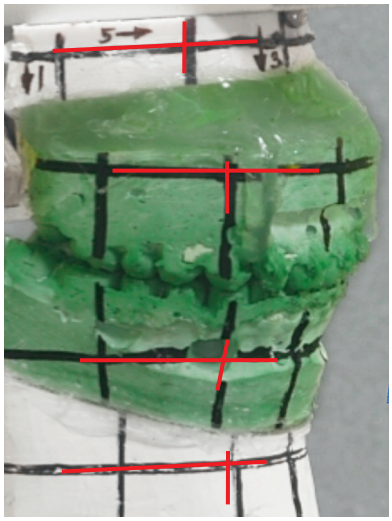
Pre surgery

After treatment

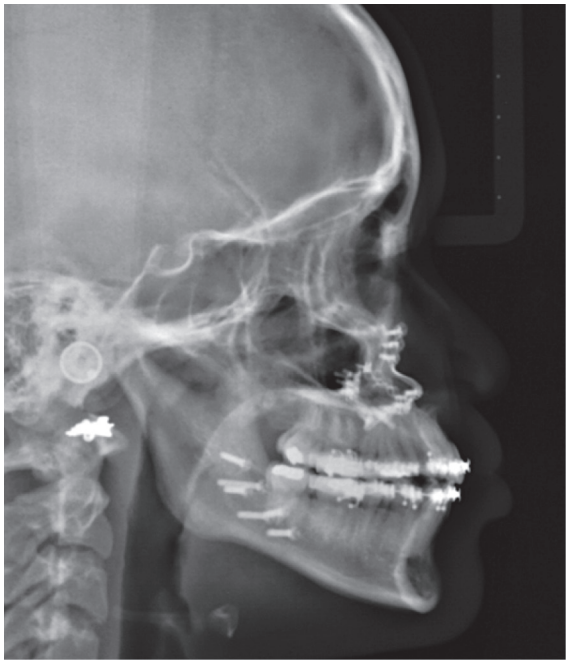
• **Figure 33-2, cont'd F,** Occlusal views during the mixed dentition, with orthodontics in progress before orthognathic surgery, and then after the completion of treatment.

Continued





Arch expansion



• **Figure 33-2, cont'd G,** Articulated dental casts indicate analytic model planning. **H,** Lateral cephalometric radiographs before and after reconstruction.

• **Figure 33-3** A child who was born with van der Woude syndrome that included bilateral cleft lip and palate and lower lip pits. He was referred to this surgeon and underwent lip and palate repair as well as lower lip pit excisions during childhood. He underwent successful mixed-dentition grafting and fistula closure. As a teenager, he demonstrated maxillary hypoplasia with secondary deformities of the mandible and the nasal cavity. He then underwent a comprehensive orthodontic and orthognathic/intranasal surgical approach. The patient's procedures included standard Le Fort I osteotomy (vertical lengthening, horizontal advancement, and clockwise rotation) with interpositional grafting; bilateral sagittal split ramus osteotomies (clockwise rotation); osseous genioplasty (horizontal advancement); and septoplasty, inferior turbinate reduction, and nasal floor recontouring. **A,** The patient is shown at the time of birth and then just before mixed-dentition grafting.

Continued

• **Figure 33-3, cont'd B,** Frontal views in repose before and after reconstruction. **C,** Frontal views with smile before and after reconstruction.

©Elsevier Inc.
Orthognathic Surgery; J.C. Posnick

• **Figure 33-3, cont'd** **D,** Oblique facial views before and after reconstruction. **E,** Profile views before and after reconstruction.

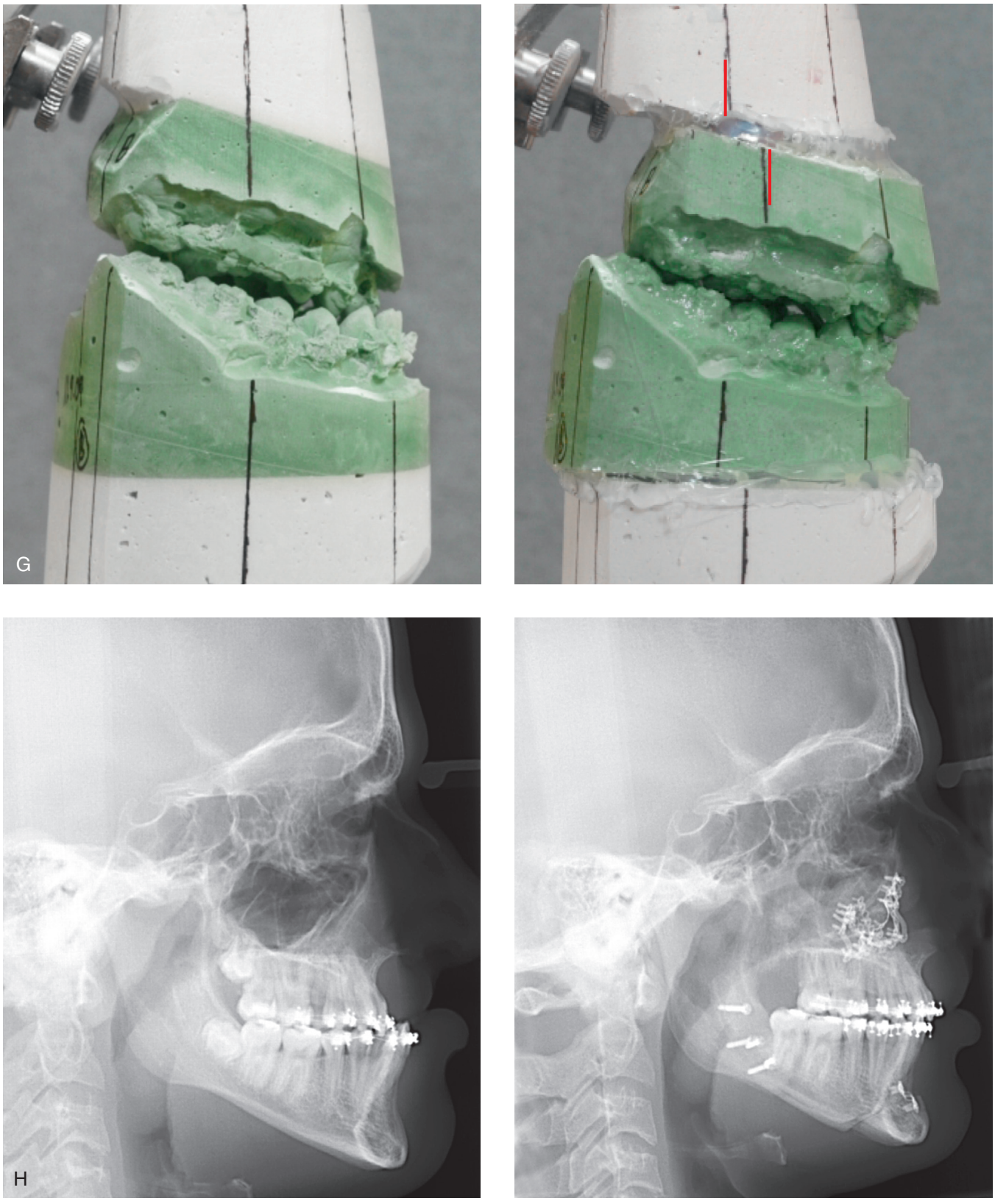
Continued

Prior to bone grafting

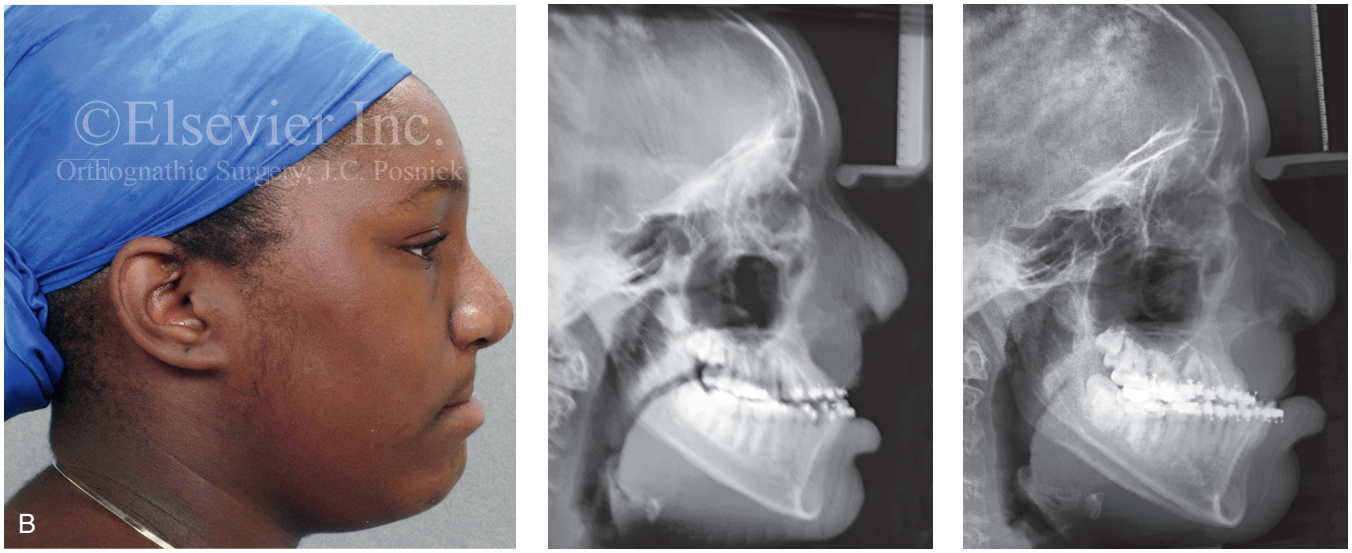
After successful mixed dentition bone graft and orthodontic cleft dental gap closure

After treatment

• **Figure 33-3, cont'd F,** Occlusal views are shown before mixed-dentition bone grafting and fistula closure, with orthodontics in preparation for orthognathic surgery, and after the completion of reconstruction. A dental implant is planned for the congenitally missing right maxillary bicuspid when the patient is 18 years old.



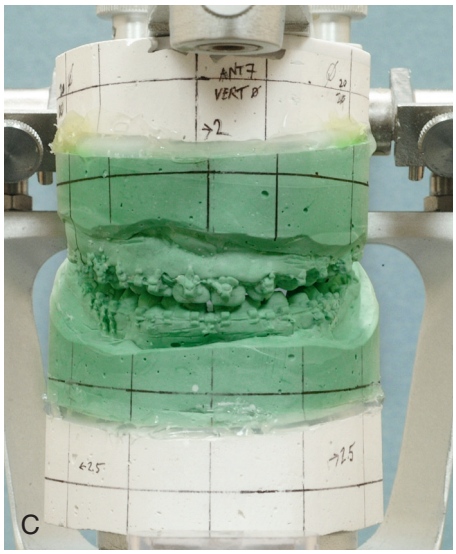
• **Figure 33-3, cont'd G,** Articulated dental casts indicate analytic model planning. **H,** Cephalometric radiographs before and after reconstruction.

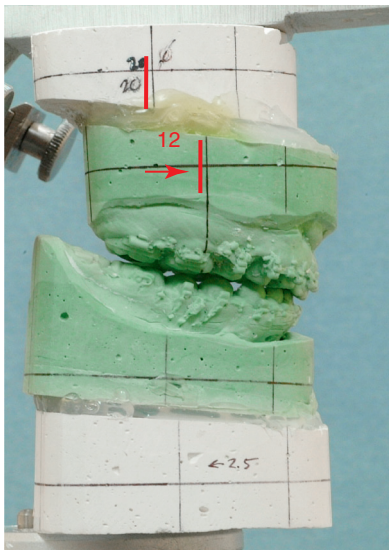


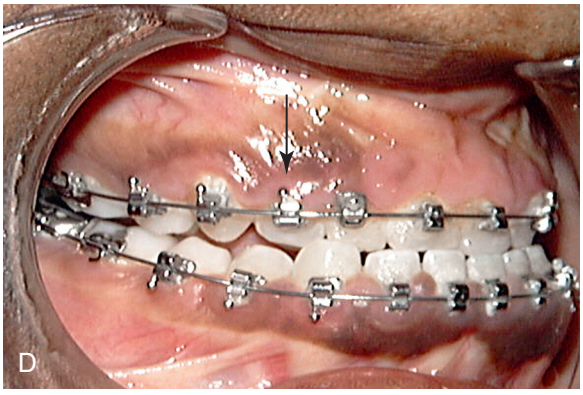
Prior to maxillary bicuspid extractions

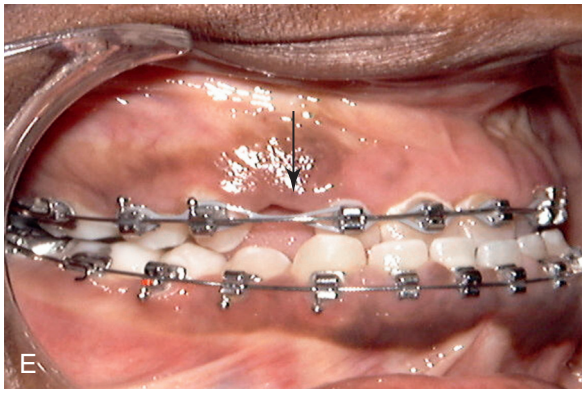
After maxillary bicuspid extractions

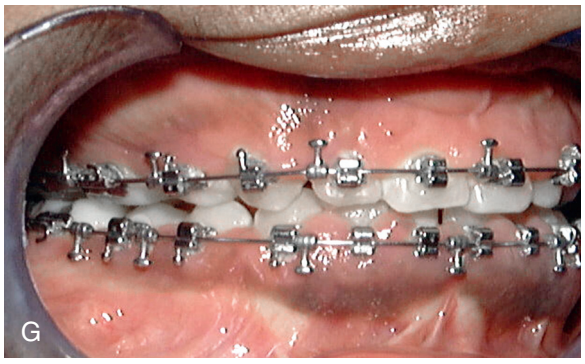
• **Figure 33-4** A child who was born with bilateral cleft lip and palate underwent lip and palate repair during infancy and early childhood by another surgeon. During the mixed dentition, she was referred to this surgeon with a mobile premaxilla, alveolar and palatal defects, and residual labial and palatal fistulas. She had the congenital absence of several permanent teeth. She underwent a short phase of orthodontic expansion and then successful iliac grafting and fistula closure during the mixed dentition. As a teenager, she underwent a comprehensive orthodontic and orthognathic/intranasal surgical approach. The patient's procedures included standard Le Fort I osteotomy (horizontal advancement and vertical shortening); bilateral sagittal split ramus osteotomies (clockwise rotation); osseous genioplasty (horizontal advancement); and septoplasty, inferior turbinate reduction, and nasal floor recontouring. **A,** The patient is shown at the time of birth. **B,** Facial and lateral cephalometric radiographic views during the adult dentition before and then after maxillary extractions with orthodontic retraction.





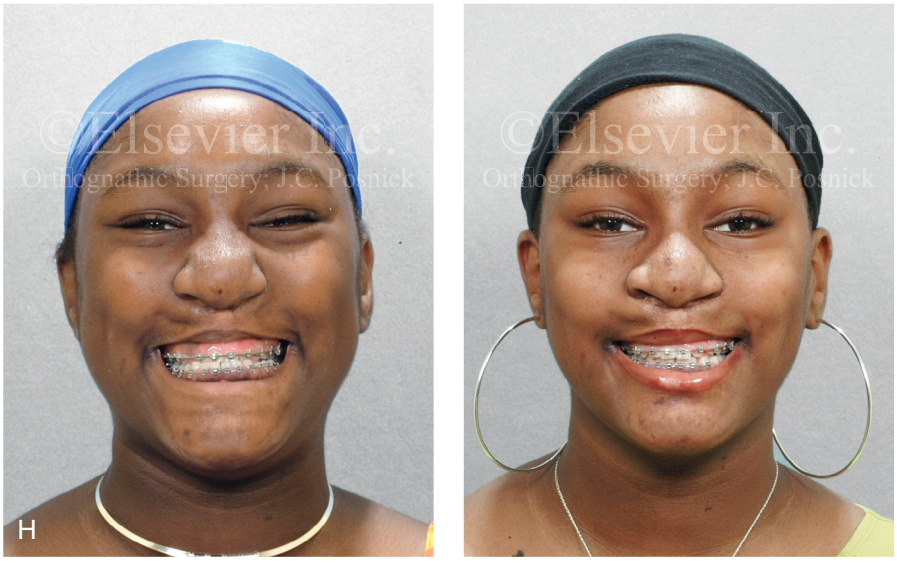






• **Figure 33-4, cont'd** **C,** Articulated dental casts that indicate analytic model planning. **D,** Occlusal view during the adult dentition before extractions. **E,** Occlusal view after extractions and before orthodontic decompensation. **F,** Occlusal view after orthodontic retraction and before surgery. **G,** Occlusal view early after surgical correction.

Continued



• **Figure 33-4, cont'd** **H,** Frontal views with smile before and after reconstruction. **I,** Oblique facial views before and after reconstruction. **J,** Profile views before and after reconstruction.

After successful mixed-dentition bone graft and maxillary bicuspid extractions - with orthodontics in progress

After treatment

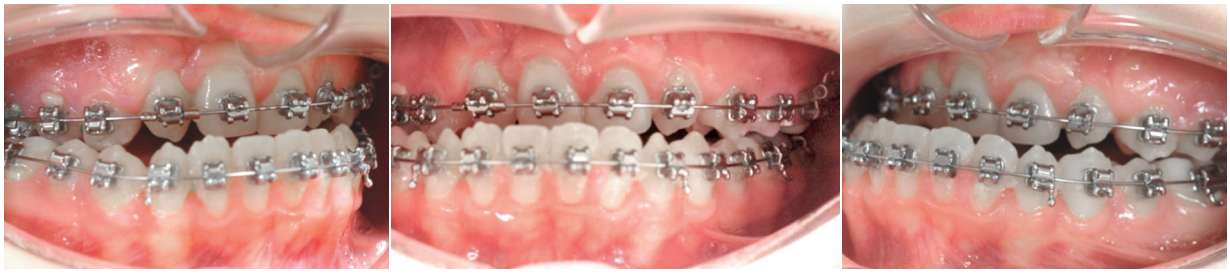
• **Figure 33-4, cont'd K,** Occlusal views before and after reconstruction.

• **Figure 33-5** A 17-year-old girl who was born with bilateral cleft lip and palate. She underwent lip repair by this surgeon during childhood. This was followed at another institution by cleft palate repair; a columella lengthening of the nasal soft tissues when she was 5 years old; a pharyngeal flap for the management of velopharyngeal insufficiency when she was 7 years old; and then bone grafting to the alveolar clefts when she was 8 years old. She was referred back to this surgeon when she was 17 years old and found to have cleft nasal deformities, chronic obstructed nasal breathing, severe maxillary hypoplasia, secondary deformities of the mandible, and retained wisdom teeth. The maxilla had been successfully grafted with orthodontic closure of the cleft dental gaps. The patient then underwent reconstruction by this surgeon, including maxillary Le Fort I osteotomy in two segments with differential repositioning (horizontal advancement +14 mm, vertical lengthening +5 mm, cant correction, and transverse narrowing) with interpositional grafting; bilateral sagittal split ramus osteotomies (asymmetry correction); osseous genioplasty (horizontal advancement); septoplasty, inferior turbinate reduction, and nasal base recontouring; and the removal of retained wisdom teeth. Before surgery, she underwent a nasoendoscopic speech assessment that demonstrated the satisfactory closure of the left lateral port with incomplete closure of the right lateral port (i.e., small air leakage). She maintained adequate velopharyngeal closure after surgery. Orthodontic maintenance and detailing continued and were followed by a removable maxillary retainer and a fixed lower anterior retaining wire. One year later, the patient underwent cleft rhinoplasty that included nasal osteotomies, the modification of the lower lateral cartilages, and a rib cartilage (caudal strut) graft. Note that ideal nasal aesthetics were compromised after a columella lengthening procedure that caused scarring of the soft tissues of the columella and distortions of the alar rims and tip. **A,** Frontal views in repose before and after reconstruction. **B,** Frontal views with smile before and after reconstruction.

©Elsevier Inc.
Orthognathic Surgery; J.C. Posnick

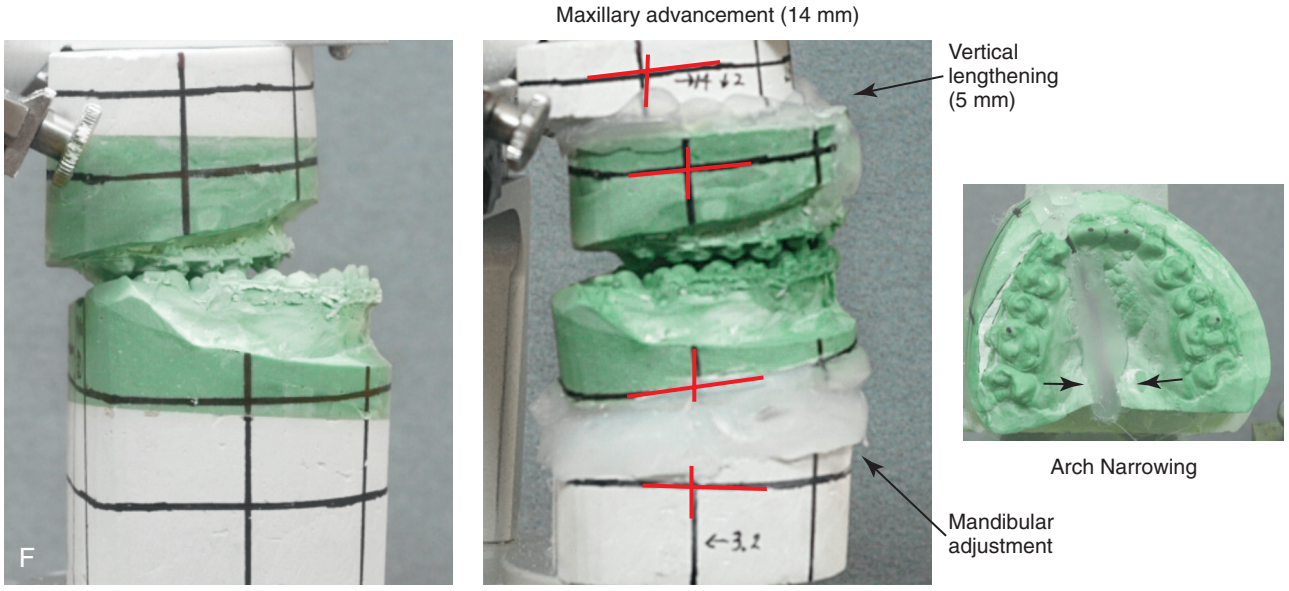
• **Figure 33-5, cont'd C,** Oblique facial views before and after reconstruction. **D,** Profile views before and after reconstruction.

Continued

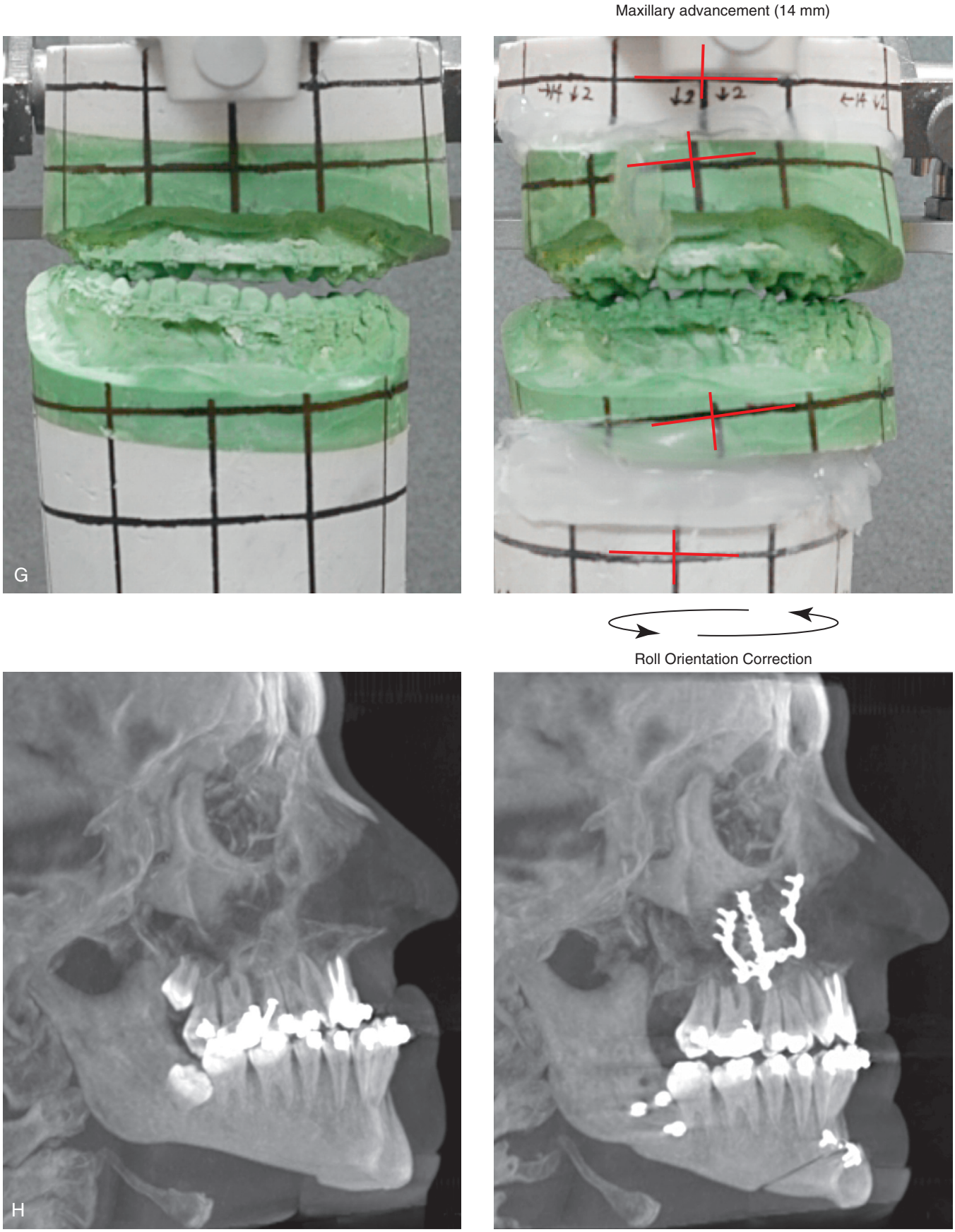


After successful mixed-dentition bone graft

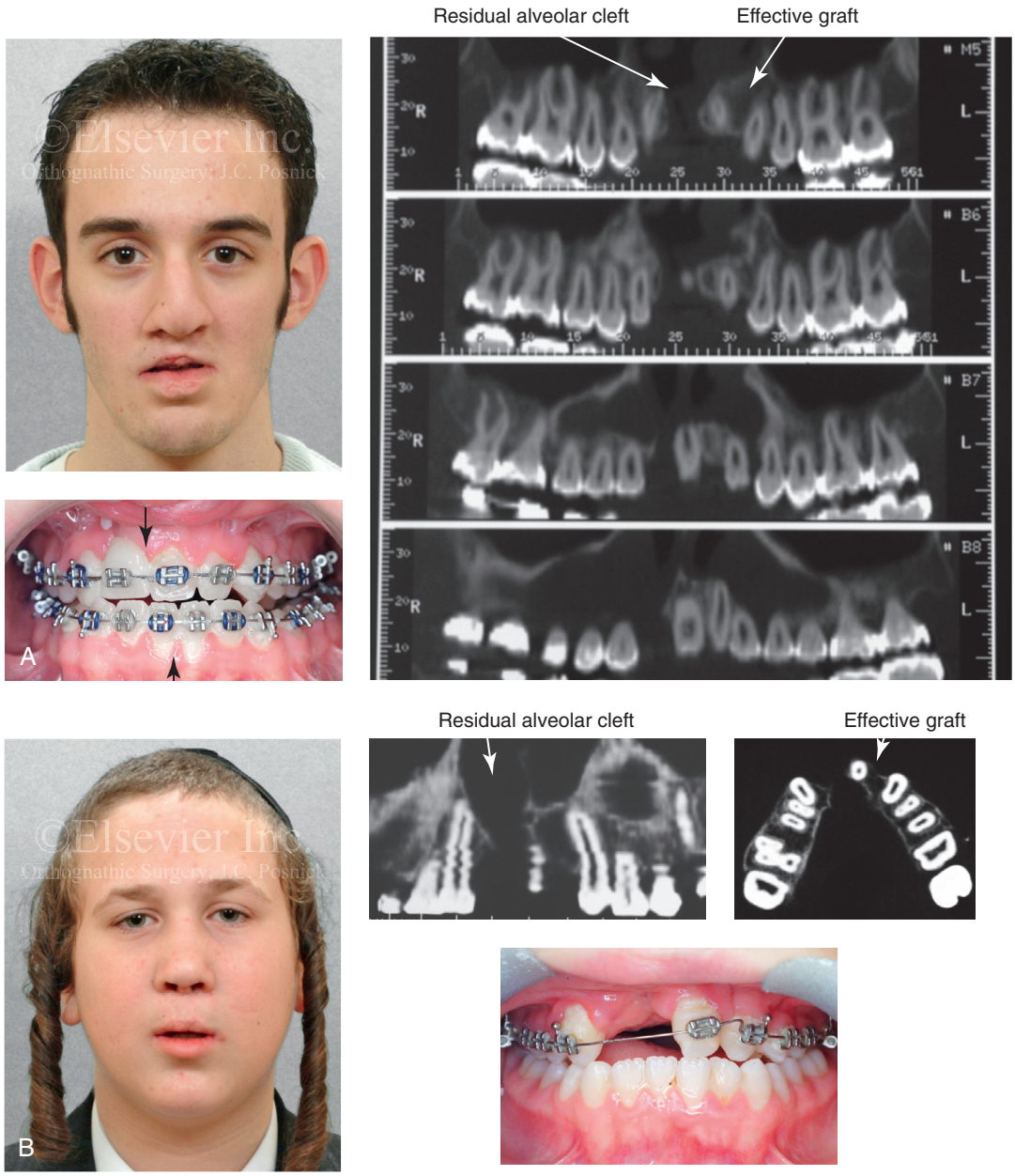
After treatment



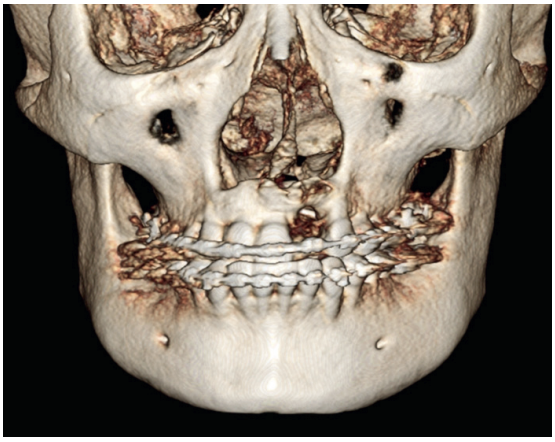
• **Figure 33-5, cont'd** **E,** Occlusal views with orthodontic appliances in place and then after reconstruction. **F** and **G,** Articulated dental casts that indicate analytic model planning.



• **Figure 33-5, cont'd H,** Lateral cephalometric radiographs before and after reconstruction.

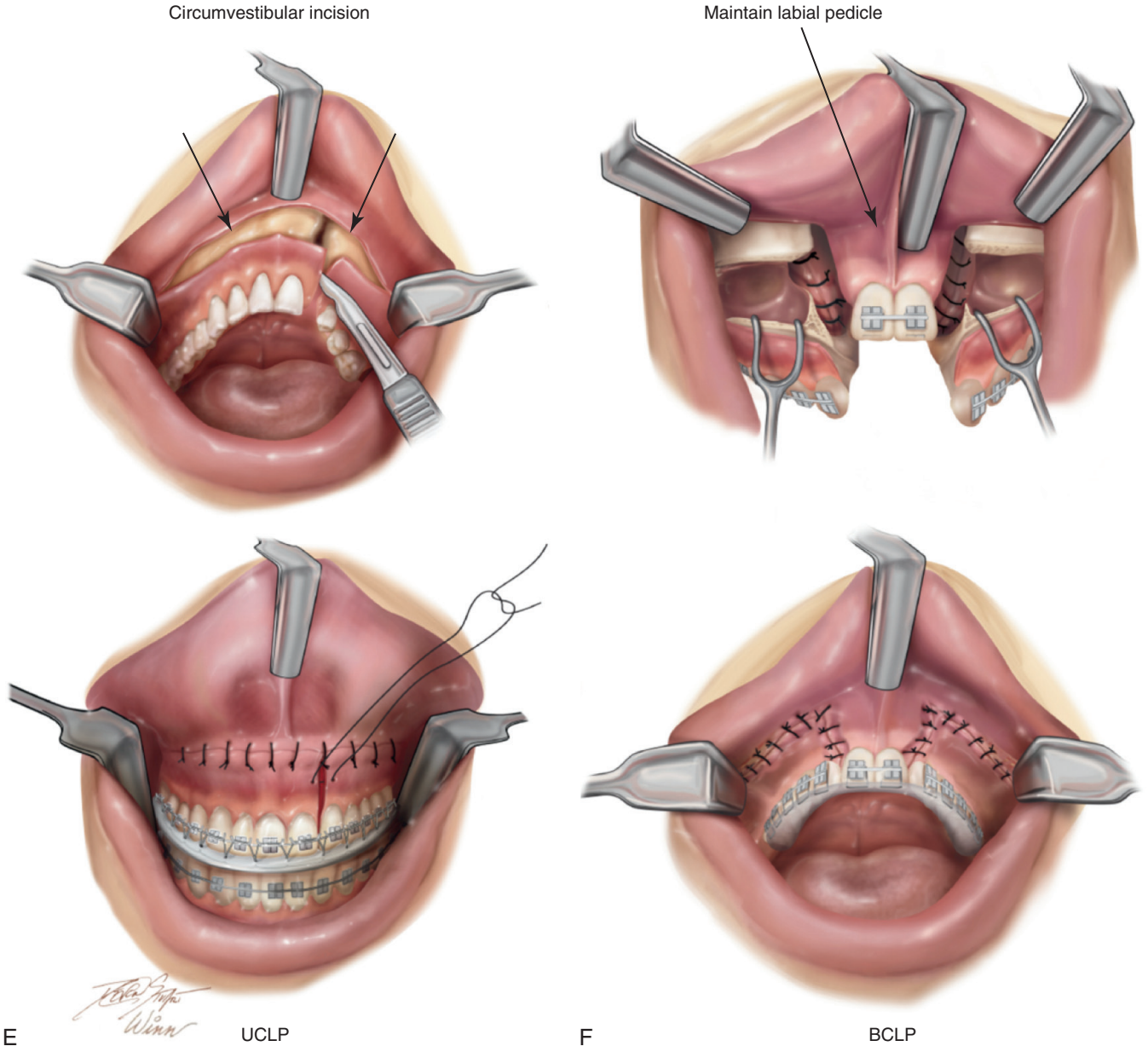


• **Figure 33-6** Three young adults who were born with bilateral cleft lip and palate are shown. Each had undergone bone grafting during the mixed dentition at another institution. Facial, occlusal, and computed tomography scan views indicate successful grafting at one cleft site but not at the other. It is important to make this determination when orthognathic surgery is required. If successful grafting was accomplished at one cleft site, then a circumvestibular incision can be made with full down-fracture, as one would for an ungrafted unilateral cleft alveolus. If successful grafting was not accomplished on either side, then a modified Le Fort I osteotomy in three segments would be required to maintain circulation to each of the three segments (i.e., the premaxilla, the left posterior segment, and the right posterior segment). This is done to avoid the complication of aseptic necrosis of the premaxilla (see Fig. 33-17). **A,** A teenager who was born with van der Woude syndrome and bilateral cleft lip and palate. He underwent successful grafting of one cleft site but not the other at another institution. He presented to this surgeon for orthognathic surgery. Facial, occlusal, and computed tomography scan views are shown. **B,** A teenager who was born with bilateral cleft lip and palate underwent successful alveolar grafting on the left side but not the right at another institution. He presented to this surgeon for orthognathic surgery. Facial, occlusal, and computed tomography scan views are shown and confirm these facts.



• **Figure 33-6, cont'd C,** A teenage girl who was born with bilateral cleft lip and palate underwent successful alveolar grafting on the right side but not the left. She then presented to this surgeon for orthognathic surgery. Facial and occlusal views when the previous patient was 7 years old, before grafting. **D,** Computed tomography scans of the same girl when she was 16 years old confirm successful grafting on right side but continued alveolar clefting on left side.

Continued



• **Figure 33-6, cont'd E,** Illustrations of incisions for a modified Le Fort I osteotomy in two segments either for a patient with unilateral cleft lip and palate and an open cleft or a patient born with BCLP and a successful graft on just one side. *Part E modified from an original illustration by Bill Winn.* **F,** Illustrations of the incisions required to maintain circulation to each of three segments in an unsuccessfully grafted case of bilateral cleft lip and palate.

• **Figure 33-7** An 18-year-old boy was born with bilateral cleft lip and palate (Video 14). He underwent lip and palate repair during childhood. Mixed-dentition grafting was not successful. He was then referred to this surgeon as a teenager and underwent a combined orthodontic and orthognathic surgical approach. The patient's procedures included modified Le Fort I osteotomy in three segments with closure of an oronasal fistula, alveolar defects, and cleft–dental gaps; and stabilization of the premaxilla with interpositional bone graft; bilateral sagittal split ramus osteotomies (correction of asymmetry); an osseous genioplasty (vertical reduction and horizontal advancement) and septoplasty, inferior turbinate reduction, and nasal floor recontouring. **A,** Frontal views at before cleft lip and palate repair. **B,** Profile and occlusal views during the mixed dentition. Iliac grafting by another surgeon was not successful.

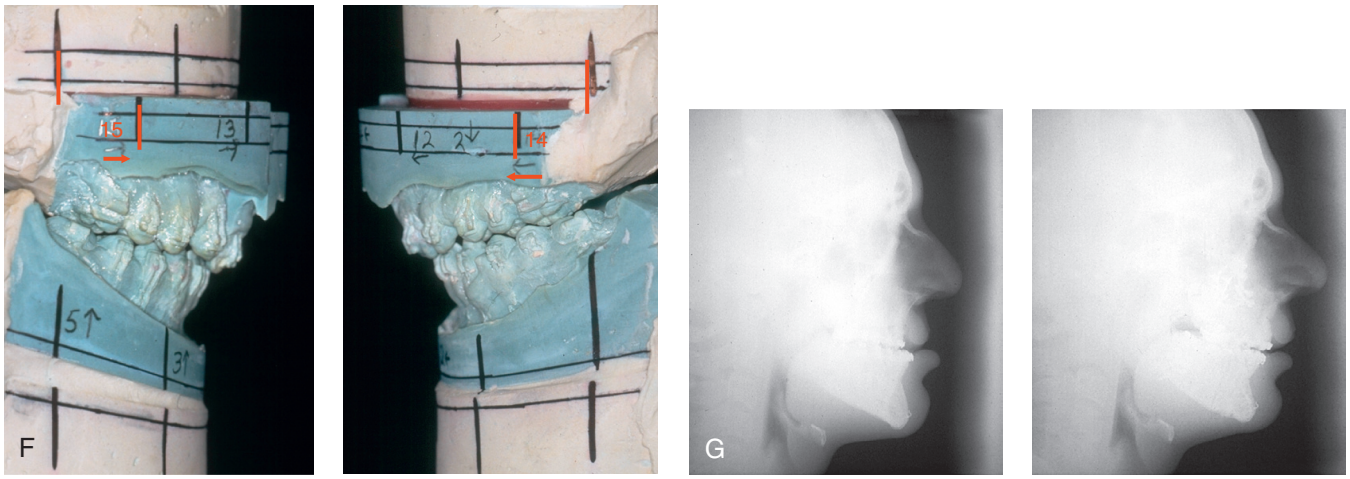
Continued

©Elsevier Inc. Orthognathic Surgery; J.C. Posnick

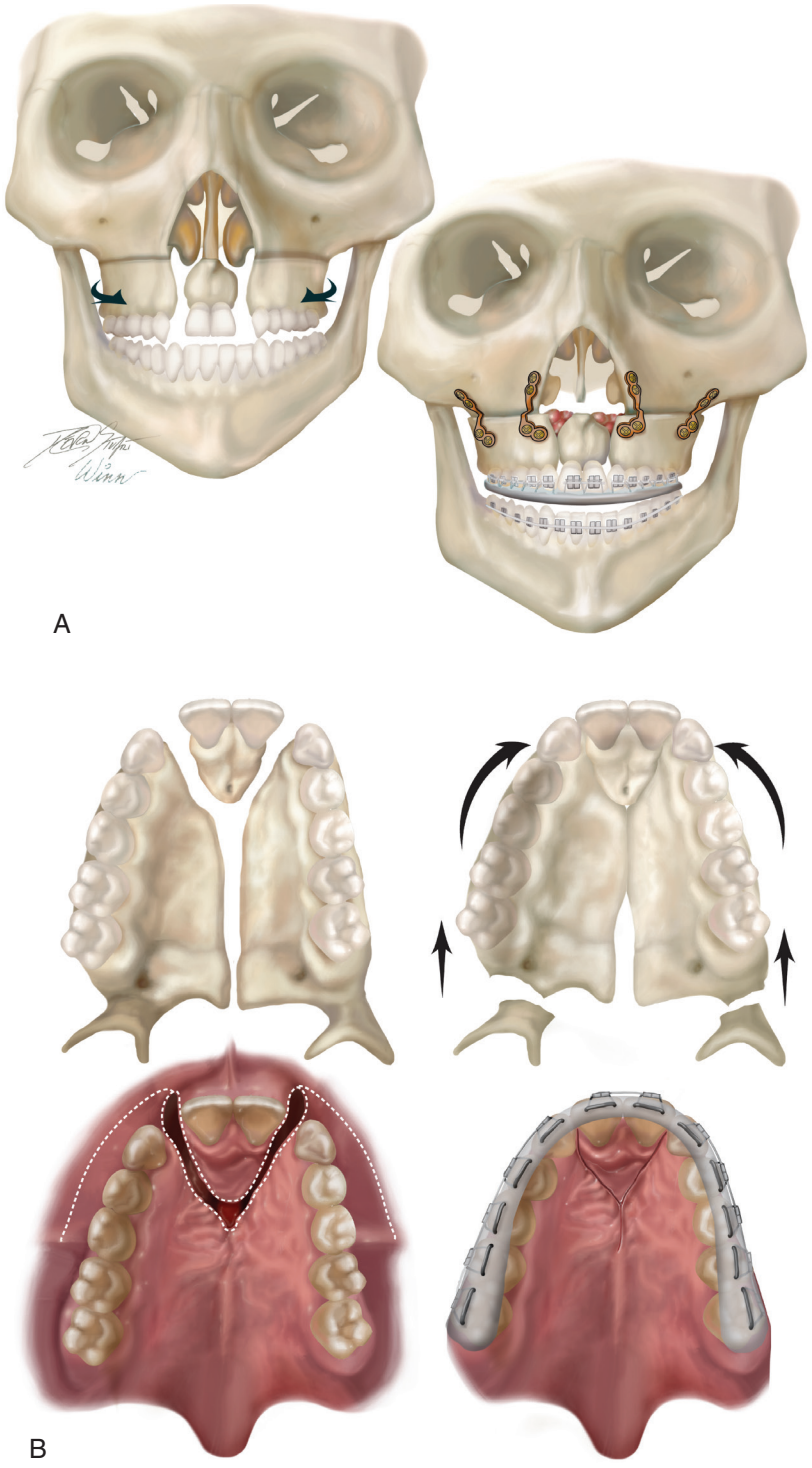
• **Figure 33-7, cont'd** **C,** Frontal views with smile before and after reconstruction. **D,** Profile views before and after reconstruction.

Residual cleft defects and mobile pre-maxilla

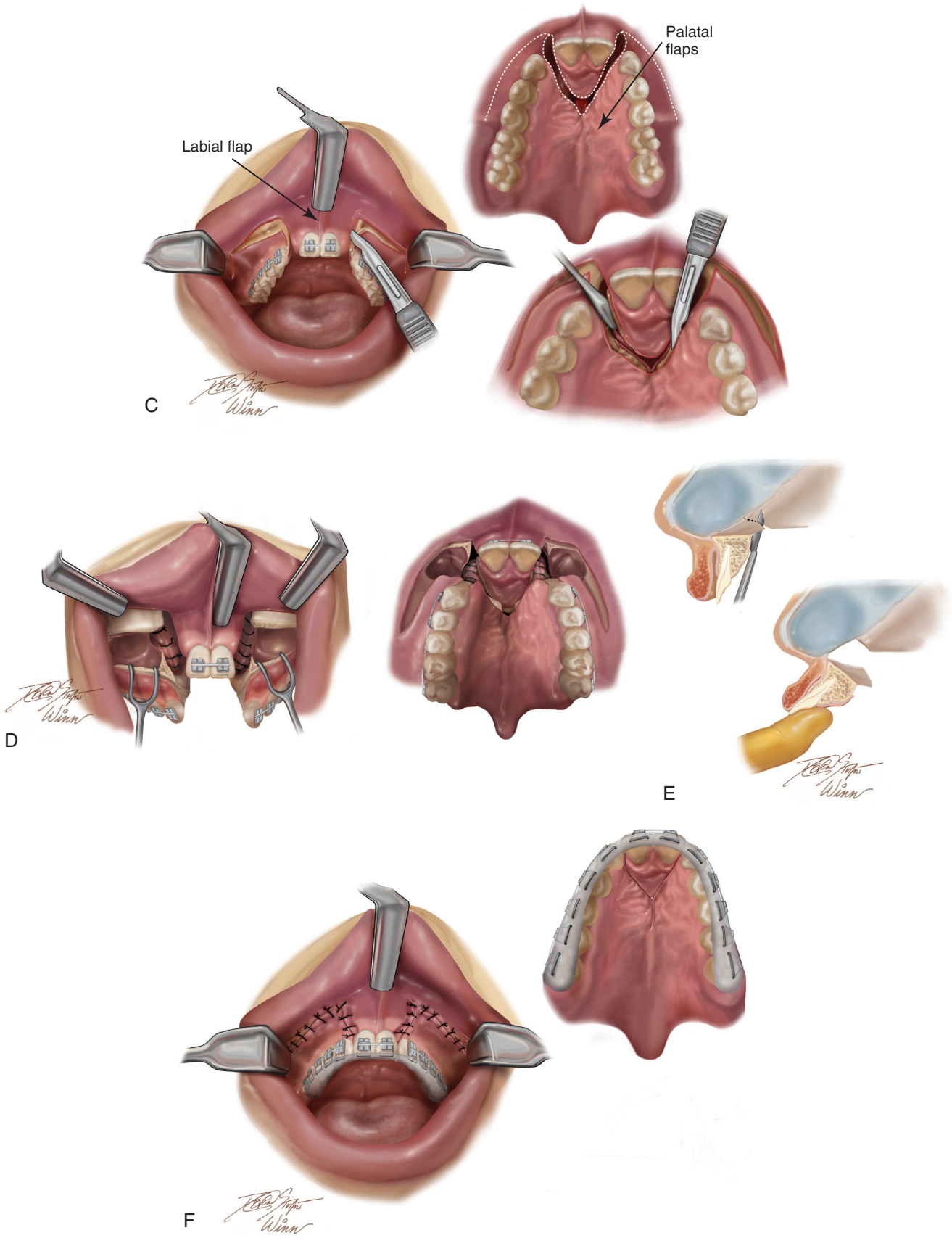
Residual cleft defects and mobile pre-maxilla



• **Figure 33-7, cont'd E,** Occlusal views before and 2 years after reconstruction. A degree of relapse (skeletal versus dental) has occurred. **F,** Articulated dental casts that indicate analytic model planning. **G,** Lateral cephalometric radiographs before and after reconstruction (Video 14). *C, D, E (top center, bottom center), from Posnick JC, Tompson B: Modification of the maxillary Le Fort I osteotomy in cleft-orthognathic surgery: the bilateral cleft and palate deformity,* J Oral Maxillofac Surg *51:2-11, 1993.*



• **Figure 33-8** Illustrations of modified Le Fort I osteotomy in three segments used when mixed-dentition grafting has not been successfully accomplished (i.e., residual fistula, alveolar clefts with mobile premaxilla, and cleft dental gaps) and maxillary hypoplasia requires orthognathic surgery. **A,** Illustrations of a patient with bilateral cleft lip and palate before and after three-part maxillary osteotomies with repositioning of the segments. Septoplasty and inferior turbinate reduction, are also shown. Skeletal views demonstrate anatomy before and after a modified Le Fort I osteotomy is three segments as described. **B,** Palatal views of bone segments before and after repositioning for the closure of cleft–dental gaps. Both skeletal and soft-tissue views are shown. Early after surgery, a prefabricated splint remains secured to the maxillary dentition. *Parts A, C-F, modified from an original illustration by Bill Winn.*

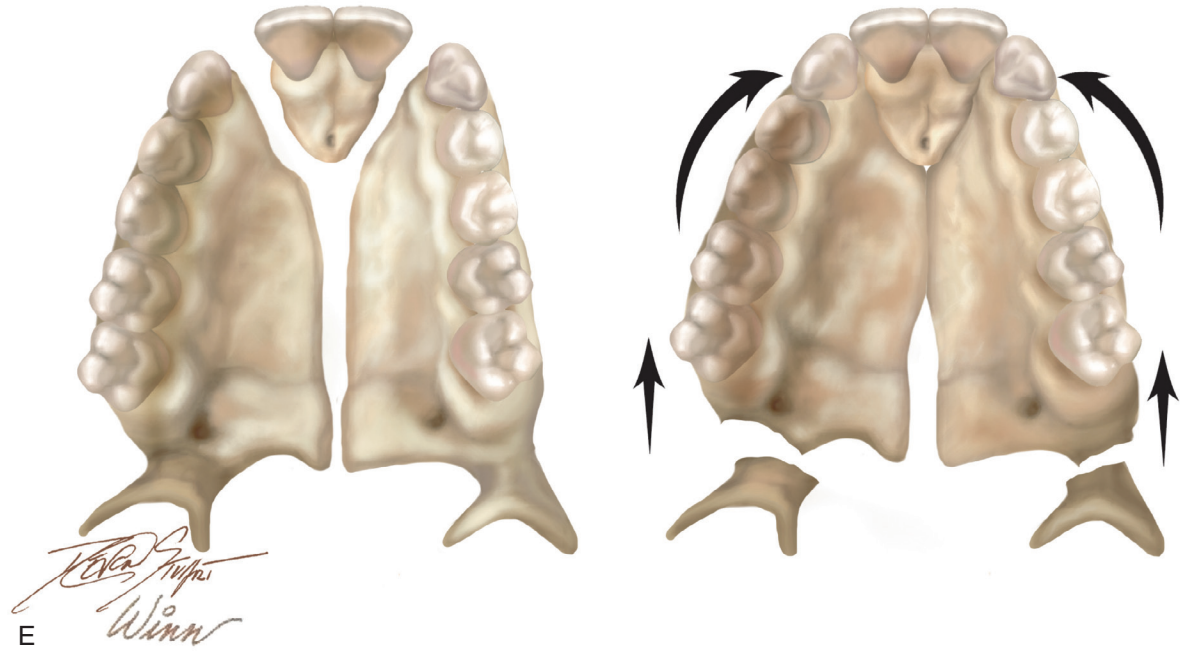


• **Figure 33-8, cont'd C,** Illustrations of incisions for modified Le Fort I osteotomy in three segments for the treatment of bilateral cleft lip and palate. **D,** Illustrations of down-fractured lateral segments demonstrating exposure for nasal-side closure of an oronasal fistula. **E,** Illustration of premaxillary osteotomy carried out on the palate side (vomer osteotomy) with the use of a reciprocating saw. **F,** Illustration demonstrating oral wounds sutured at the end of the procedure. Note that no sutures are required on the palate side. *(From Posnick JC: Cleft orthognathic surgery. In Russell RC (ed): Instructional Courses. Plastic Surgery Education Foundation, vol. 4, St. Louis, Mosby-Year Book, 1991, pp 129-157.)*

After palate repair
with large fistula

Residual cleft defects and mobile pre-maxilla

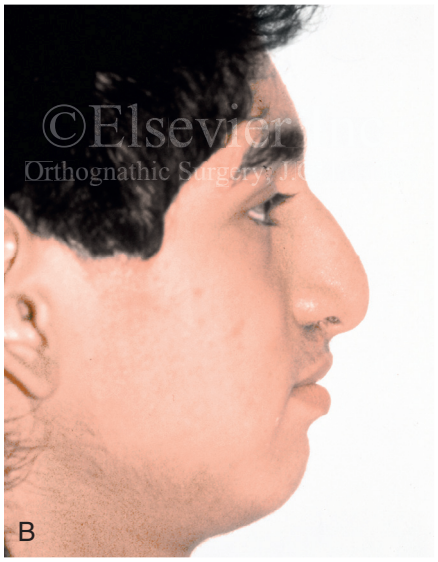
• **Figure 33-9** A 15-year-old girl was born with bilateral cleft lip and palate. She underwent lip and palate repair during infancy and early childhood by another surgeon. She retained a large oronasal fistula in the incisal foramen region from the time of the initial palate repair. She had worn a palatal prosthesis to obliterate the opening, but this interfered with speech articulation and oral hygiene. She was referred to this surgeon as a teenager and underwent a comprehensive orthodontic and orthognathic approach to close the large oronasal fistula, to stabilize the maxillary segments, and to restore her occlusion and periodontal health. As part of the preoperative orthodontic treatment, she underwent extraction of the five teeth for which there was inadequate alveolar bony support. During the operation, she underwent modified Le Fort I osteotomy in three segments with differential repositioning and interpositional grafting for closure of the large oronasal fistula, the cleft–dental gaps, and the alveolar defects. She also underwent septoplasty and inferior turbinate reduction to improve nasal airflow. Note that the maxilla was rehabilitated with the absence of the lateral incisors and a first bicuspid on the right side. **A,** Facial views before lip and palate repair. **B,** Palatal view during early childhood that indicates extensive oronasal fistula just after primary repair. **C** and **D,** Facial and palatal views at 15 years of age, when the patient was referred to this surgeon.

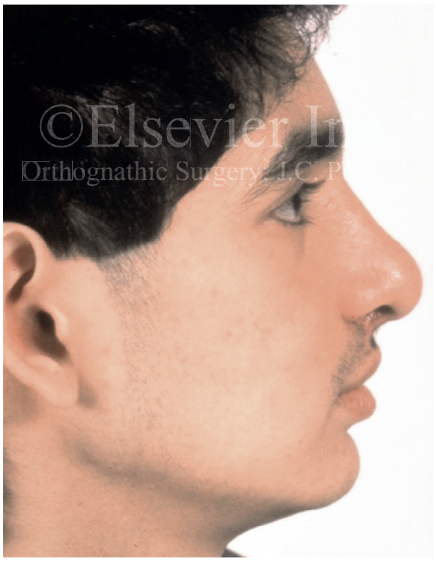


• **Figure 33-9, cont'd** **E,** Palatal view and illustrations demonstrating the patient's skeletal anatomy before and after reconstruction. The "X"s mark the teeth that are planned for extraction. The black dot and the two arrows indicate the location where the palatal mucosa will come together. **F,** Palatal views before and after reconstruction and dental rehabilitation via the technique just described.

Continued

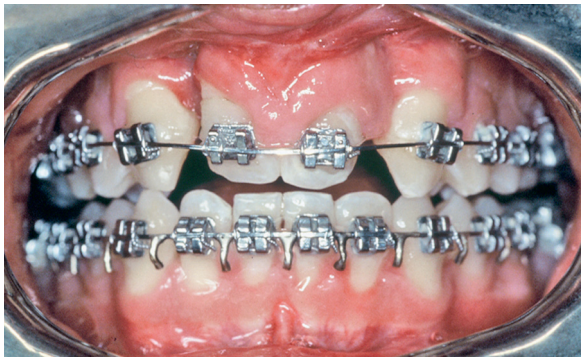
• **Figure 33-9, cont'd G,** Occlusal views before and after reconstruction. **H,** Occlusal and periapical radiographs before and after reconstruction and dental rehabilitation. **I,** Facial views before and after reconstruction.





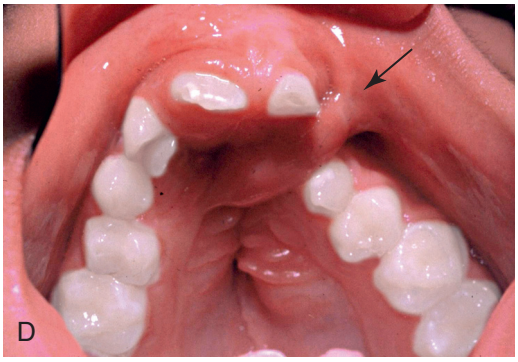
• **Figure 33-10** A 17-year-old boy of Hispanic descent was born with bilateral cleft lip and palate. He underwent lip and palate repair during childhood and unsuccessful grafting during the mixed dentition. He was referred to this surgeon as a teenager and underwent a comprehensive orthodontic and orthognathic approach. The patient's procedures included a modified Le Fort I osteotomy in three segments with differential repositioning and interpositional grafting (closure of the oronasal fistula, the alveolar defects, and the cleft–dental gaps; stabilization of the premaxilla); osseous genioplasty (vertical reduction and horizontal advancement); and septoplasty and inferior turbinate reduction. Six months later, he underwent an open rhinoplasty that included a rib cartilage (caudal strut) graft to improve nasal tip projection. **A,** Profile and occlusal views during the mixed dentition. **B,** Profile views during the teenage years before and after reconstruction. **C,** Frontal views during the teenage years before and after reconstruction and dental rehabilitation.

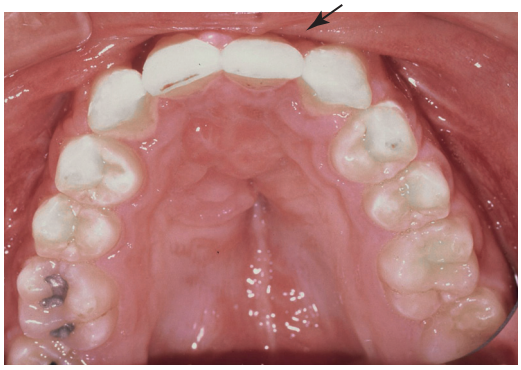
Continued



Unfavorable buccal flap

After flap take down and orthognathic correction





• **Figure 33-10, cont'd D,** Occlusal and palatal views before and after reconstruction and dental rehabilitation. *B, C ,D from Posnick JC, Witzel MA, Dagys AP: Management of jaw deformity in the cleft patient. In Bardach K, Morris HL, eds:* Multidisciplinary management of cleft lip and palate, *Philadelphia, 1990, W.B. Saunders, p 538.*

• **Figure 33-11** A 19-year-old man was born with bilateral cleft lip and palate. He underwent lip and palate repair during childhood but did not undergo grafting during the mixed dentition. He was referred to this surgeon as a teenager and underwent a comprehensive orthodontic and orthognathic approach. The procedures included a modified Le Fort I osteotomy in three segments with differential repositioning and interpositional grafting (closure of the oronasal fistula, the alveolar defects, and the cleft–dental gaps; stabilization of the premaxilla) as well as septoplasty and inferior turbinate reduction. **A,** Frontal views with smile before and after reconstruction. **B,** Oblique facial views before and after reconstruction. **C,** Profile views before and after reconstruction. *Continued*

• **Figure 33-11, cont'd** **D,** Occlusal views with orthodontics in progress and after reconstruction. **E,** Palatal views before and after reconstruction. **F,** Articulated dental casts that indicate analytic model planning.

• **Figure 33-12** A 15-year-old girl was born with bilateral cleft lip and palate. She underwent lip and palate repair during infancy. A superiorly based pharyngeal flap, lip scar revision, and tip rhinoplasty were carried out when she was 9 years old. She underwent iliac bone grafting to the alveolar clefts, without success. She was referred to this surgeon as a teenager and underwent a comprehensive orthodontic and surgical approach. The patient's procedures included modified Le Fort I osteotomy in three segments with differential repositioning and interpositional grafting (closure of the oronasal fistula, the alveolar clefts, and the cleft–dental gaps, with stabilization of the premaxilla); bilateral sagittal split osteotomies of the mandible (clockwise rotation without set-back); oblique osteotomy of the chin (horizontal advancement); removal of retained wisdom teeth; and septoplasty, inferior turbinate reduction, and recontouring of the pyriform rims. **A,** Frontal views in repose before and after reconstruction. **B,** Frontal views with smile before and after reconstruction.

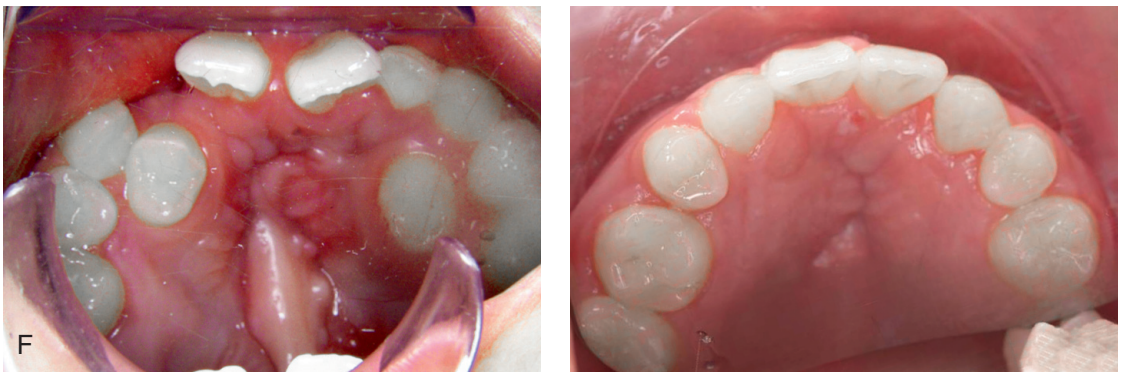
Continued

• **Figure 33-12, cont'd C,** Oblique facial views before and after reconstruction. **D,** Profile views before and after reconstruction.

S/P failed bone graft with residual cleft defects

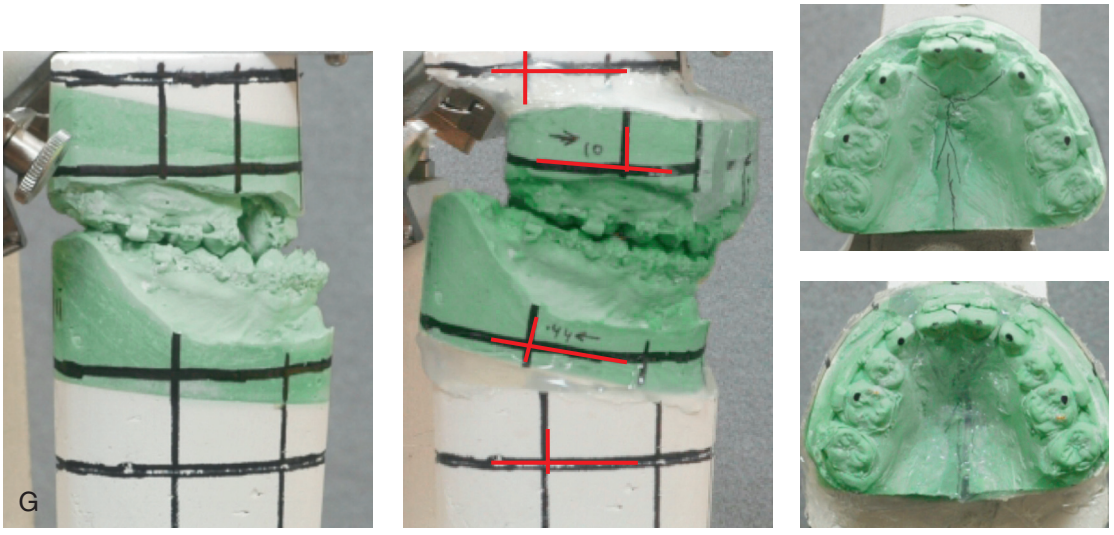
Pre surgery

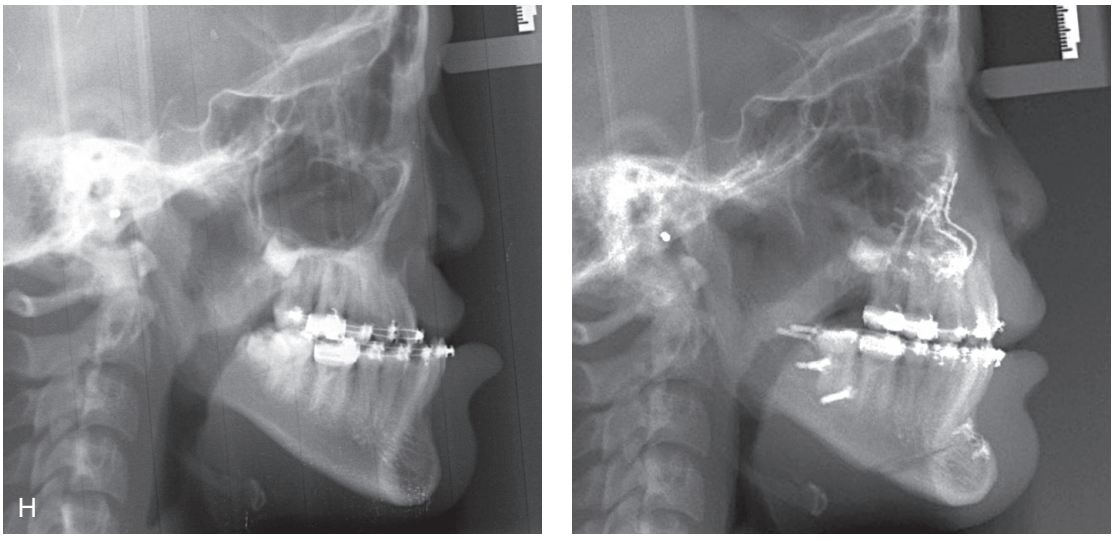
After treatment



• **Figure 33-12, cont'd E,** Occlusal views before the final phase of orthodontics, with orthodontics in preparation for surgery, and then after the completion of treatment. **F,** Palatal views before and after reconstruction.

Continued





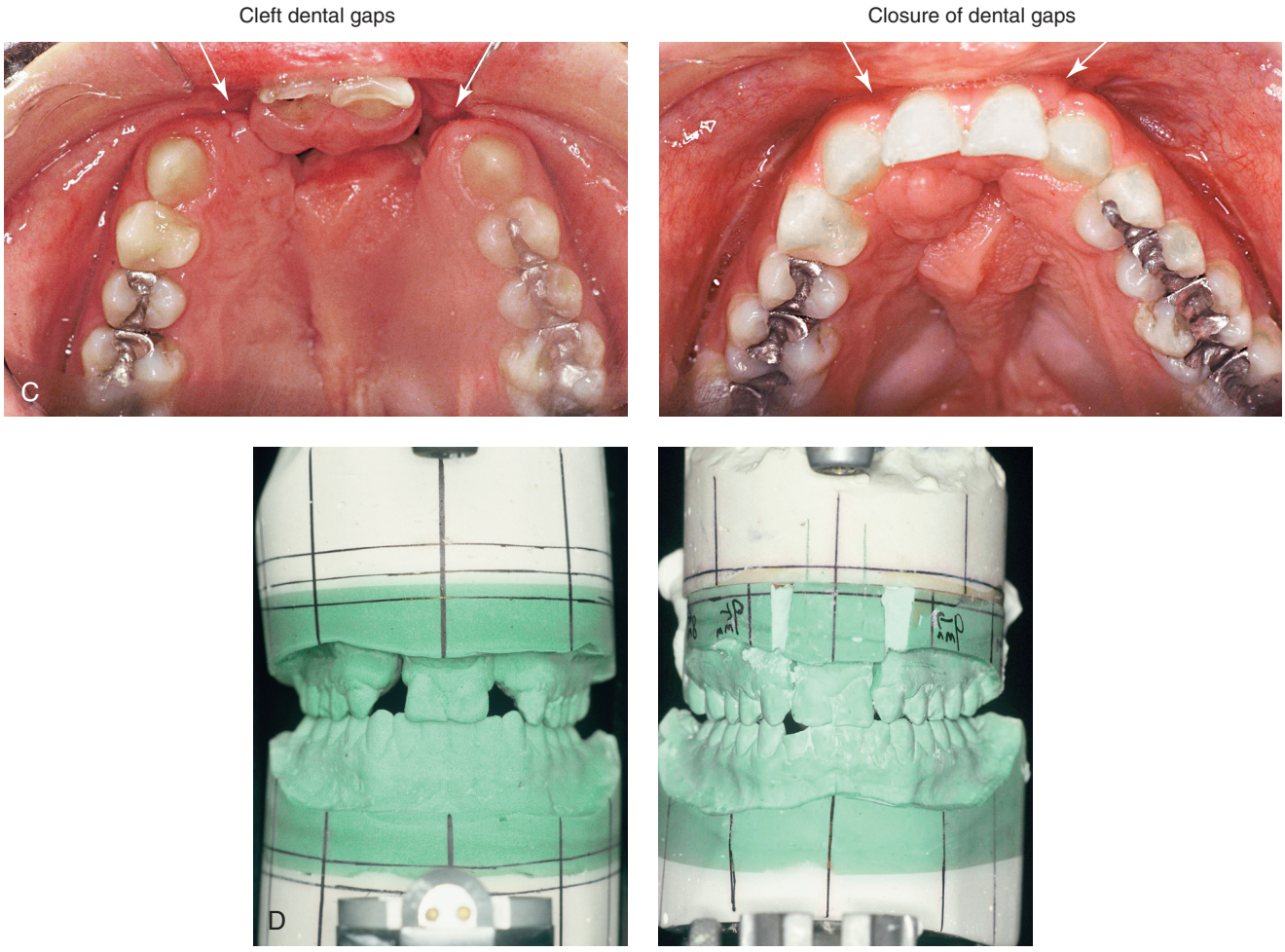
• **Figure 33-12, cont'd G,** Articulated dental casts that indicate analytic model planning. **H,** Lateral cephalometric views before and after surgery. Note that the maxillary dentition is reconstructed with only 10 teeth; there was a congenital absence of the lateral incisors and the removal of the first bicuspids to relieve crowding. The maxillary wisdom teeth were impacted and not functional and therefore also removed.

Residual cleft defects and mobile pre-maxilla

Residual cleft defects and mobile pre-maxilla

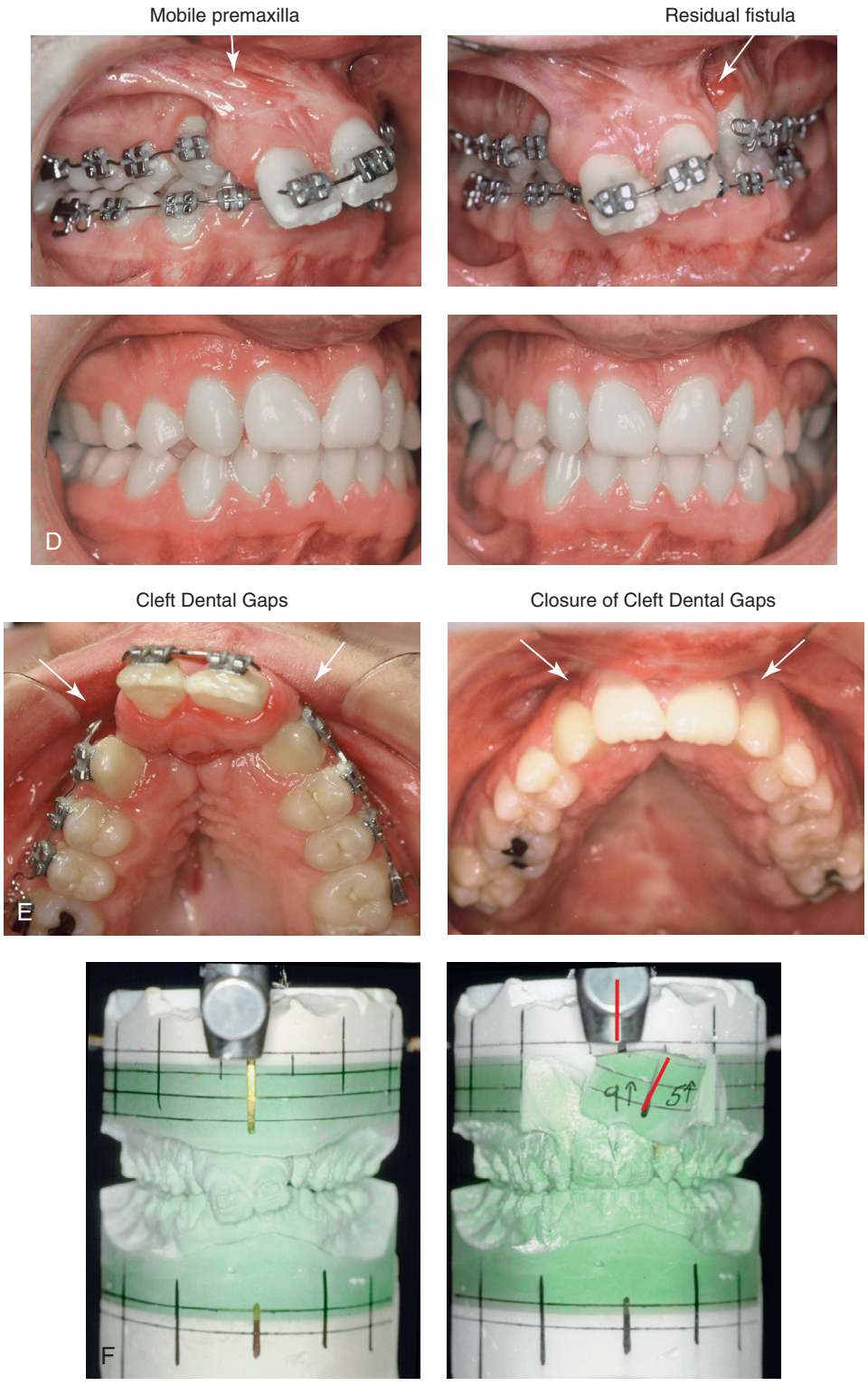
• **Figure 33-13** An 18-year-old woman who was born with bilateral cleft lip and palate underwent lip and palate repair during childhood but did not undergo successful grafting during the mixed dentition. She was referred to this surgeon as a teenager and underwent a combined orthodontic and surgical approach. The procedures included lateral segmental osteotomies of the maxilla (anterior advancement of the segments with closure of oronasal fistulas, alveolar defects, and cleft–dental gaps and the stabilization of premaxilla with interpositional grafting) as well as septoplasty and inferior turbinate reduction. **A,** Frontal views with smile before and after reconstruction. **B,** Occlusal views before and after reconstruction.

Continued

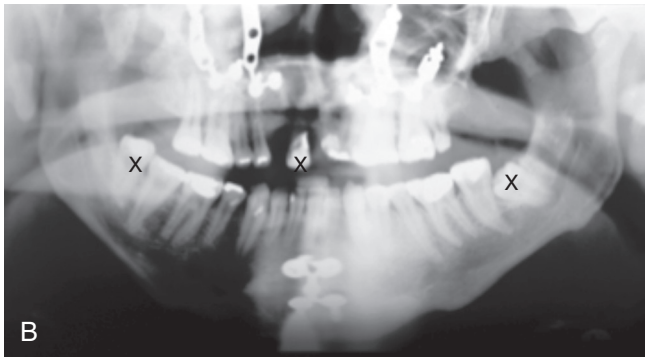


• **Figure 33-13, cont'd C,** Palatal views before and after reconstruction. **D,** Articulated dental casts that indicate analytic model planning. **A, B,** *and* **C** *from Posnick JC: Discussion of: Orthognathic surgery in cleft patients treated by early bone grafting,* Plast Reconstr Surg *87:840-842, 1991.*

• **Figure 33-14** A boy with bilateral cleft lip and palate underwent lip and palate repair during infancy. The premaxilla was distorted soon after birth, and it remained so into his teenage years. The patient presented to this surgeon when he was 16 years old for the management of residual dentoalveolar deformities. The maxillary (lateral) segments were well positioned, but the premaxilla was three-dimensionally distorted and mobile, bilateral labial and palatal fistulas were present, and the lateral incisors were congenitally absent. It was possible to surgically reposition (osteotomize) the premaxilla, to bone graft the defects of the palate and the floor of the nose, and to close the residual oronasal fistula during one procedure. **A,** Facial views in infancy after one side lip repair. **B,** Facial views during the mixed dentition after lip and palate repair. **C,** Frontal views with smile before and after premaxillary osteotomy and reconstruction. *Continued*

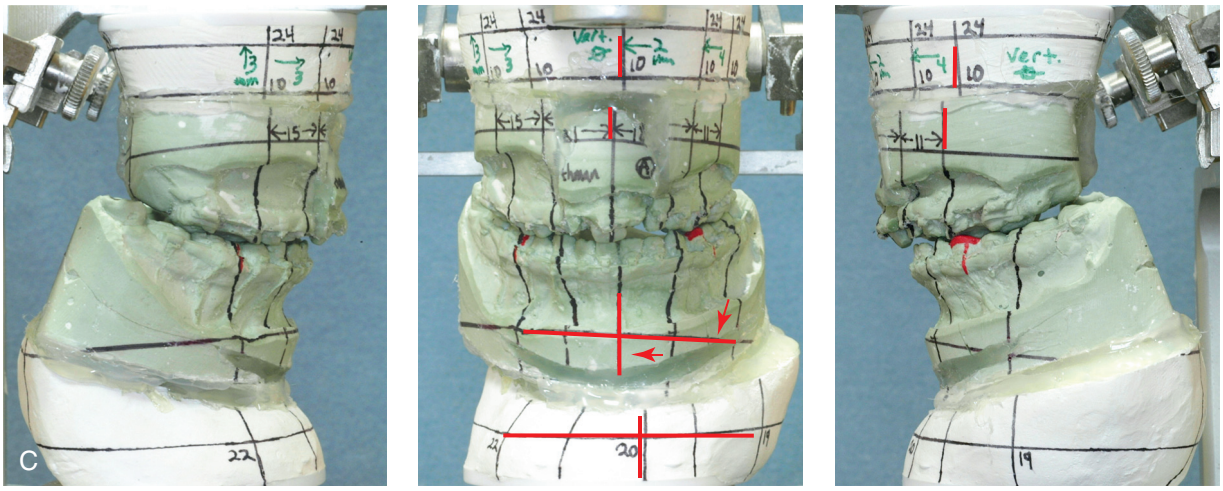


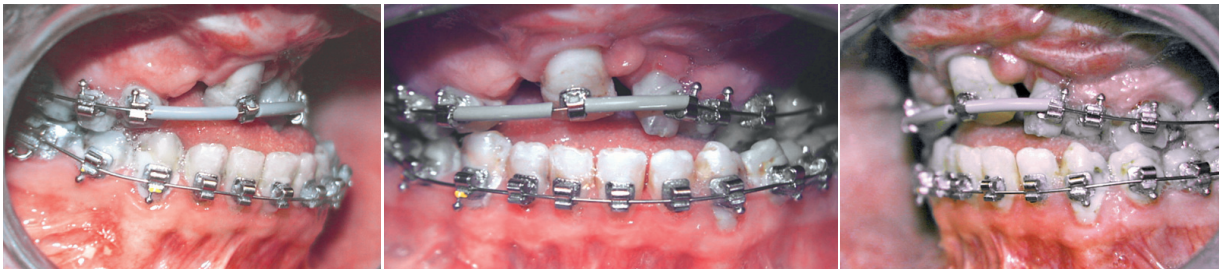
• **Figure 33-14, cont'd D,** Occlusal views before and after premaxillary osteotomy for reconstruction. **E,** Palatal views before and after premaxillary osteotomy for reconstruction. **F,** Articulated dental casts that indicate analytic model planning.



• **Figure 33-15** A 21-year-old man was born with bilateral cleft lip and palate. He underwent lip and palate repair, unsuccessful bone grafting, and unfavorable orthognathic surgery (Le Fort I and genioplasty) at another institution. He presented to this surgeon when he was 21 years old with a jaw deformity, malocclusion, and a limited dentition. He underwent evaluations by a periodontist, a prosthodontist, a surgeon, a speech pathologist, and an otolaryngologist. He then underwent a comprehensive surgical and dental rehabilitative approach. After periodontal treatment and further orthodontic (dental) decompensation, the patient's reconstruction included maxillary Le Fort I osteotomy in three segments with differential repositioning and interpositional grafting (cant correction, closing down the cleft gap, closing of the fistula, horizontal advancement, counterclockwise rotation, and vertical adjustment); bilateral sagittal split ramus osteotomies (asymmetry correction); and septoplasty and inferior turbinate reduction. This was followed by crown and bridge dental rehabilitation of the maxillary and mandibular dentition and ongoing periodontal surveillance. **A,** Frontal and occlusal views before redo orthodontics, surgery, and dental rehabilitation. **B,** Panorex and lateral cephalometric radiographs at time of presentation to this surgeon.

Continued





Pre surgery with residual cleft defects and mobile pre-maxilla

After treatment

• **Figure 33-15, cont'd C,** Articulated dental models that indicate analytic model planning. **D,** Facial views with smile before and after reconstruction and dental rehabilitation. **E,** Occlusal views before and after reconstruction and dental rehabilitation.

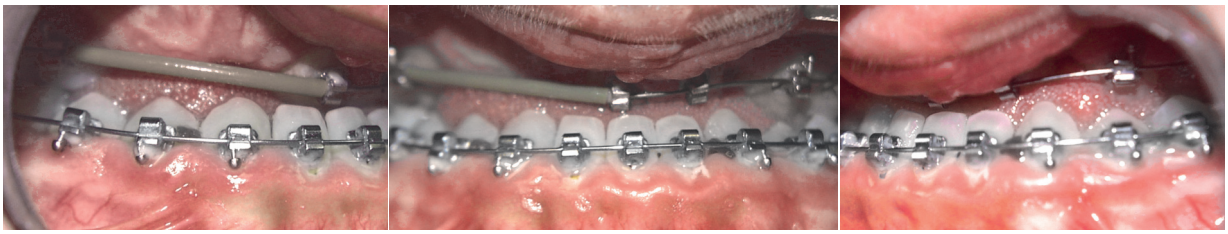
• **Figure 33-16** A boy who was born with bilateral cleft lip and palate underwent lip and palatal repair and unsuccessful bone grafting at another institution. He presented to this surgeon when he was 17 years old with a severe jaw deformity and a limited dentition. He underwent evaluations by an orthodontist, a periodontist, a prosthodontist, a surgeon, an otolaryngologist, and a speech pathologist. He agreed to a comprehensive surgical and dental rehabilitative approach. After periodontal treatment, orthodontic (dental) decompensation was accomplished. The patient's reconstructive surgery included maxillary Le Fort I osteotomy in three segments with differential repositioning and interpositional grafting, (clockwise rotation, vertical lengthening, and horizontal advancement), bilateral sagittal split ramus osteotomies (clockwise rotation), osseous genioplasty (bur recontouring to reduce pogonion); and septoplasty and inferior turbinate reduction. After initial healing and orthodontic completion, crown and bridge prosthetic rehabilitation was carried out. Six months later, the patient underwent an open rhinoplasty that included rib cartilage (caudal strut) placement. **A,** Frontal views in repose before and after reconstruction and rehabilitation. **B,** Frontal views with smile before and after reconstruction and rehabilitation.

Continued

©Elsevier Inc.
Orthognathic Surgery; J.C. Posnick

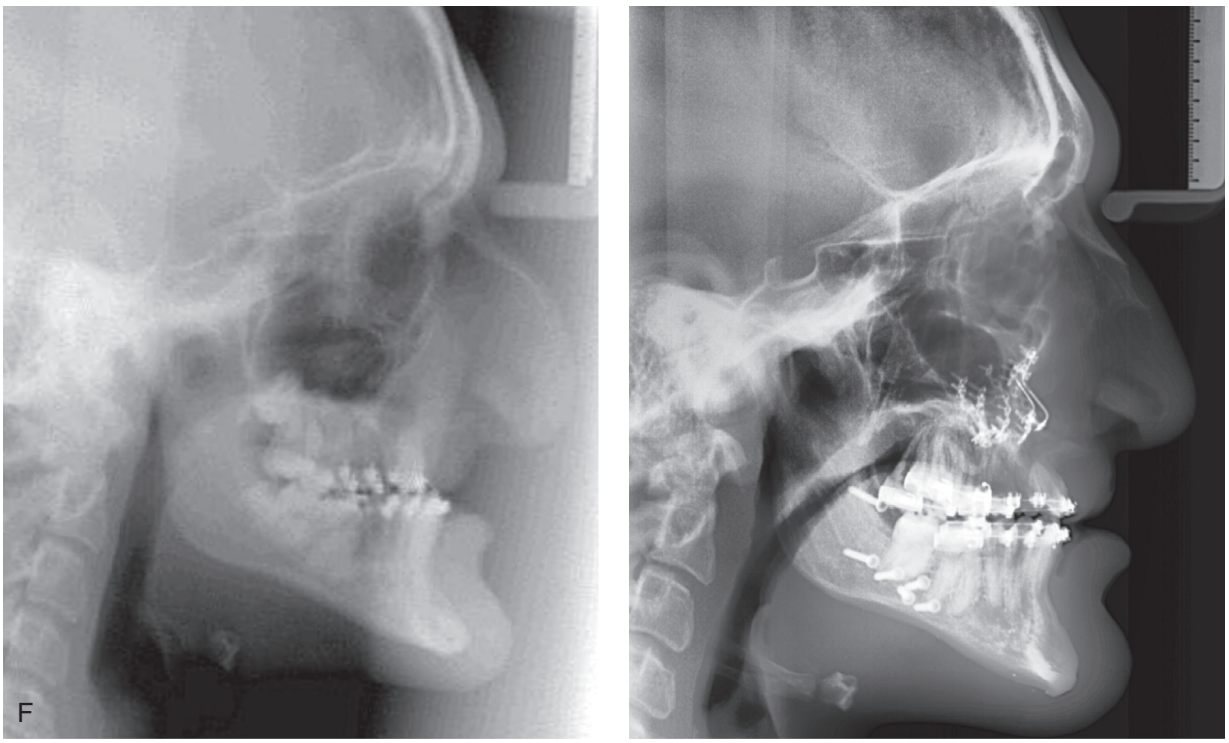
• **Figure 33-16, cont'd C,** Oblique facial views before and after reconstruction and rehabilitation. **D,** Profile views before and after reconstruction and rehabilitation.

Prior to treatment with residual cleft defects and mobile pre-maxilla



Pre surgery

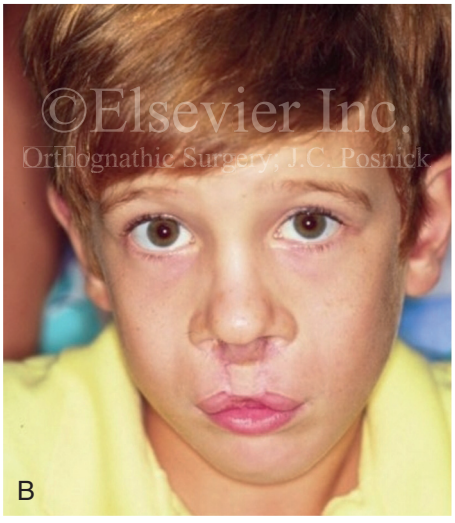
After treatment with Anterior bridge in place



• **Figure 33-16, cont'd E,** Occlusal views before retreatment, with orthodontic appliances in place, and then after reconstruction and rehabilitation. **F,** Lateral cephalometric radiographs before and after reconstruction.

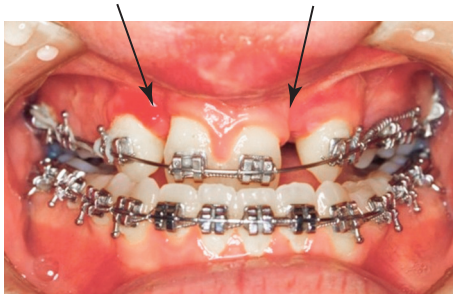
Prior to bone graft

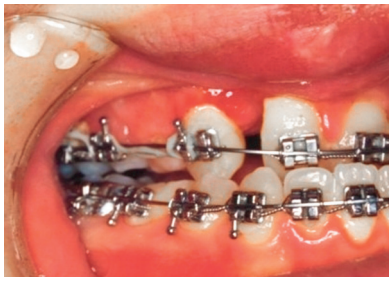
2 weeks after unsuccessful bone graft



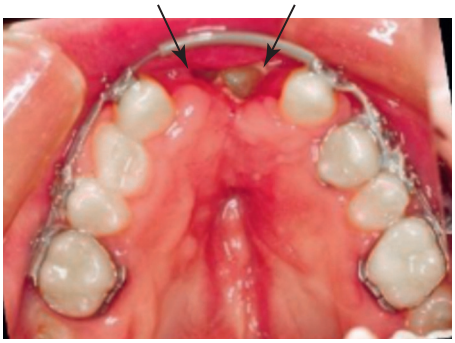
• **Figure 33-17** A child who was born with bilateral cleft lip and palate underwent lip and palate repair followed by unsuccessful mixed-dentition bone grafting and unfavorable orthognathic surgery with a loss of the premaxilla to aseptic necrosis at another institution. He also underwent unfavorable secondary lip and nasal soft-tissue procedures (including columella lengthening) at another institution. He was referred to this surgeon during his college years for the management of the jaw deformities and the fistula and for the coordination of his dental rehabilitative needs. He underwent evaluations by a periodontist, an orthodontist, a prosthodontist, a surgeon, an otolaryngologist, and a speech pathologist. After further orthodontic (dental) decompensation, the patient's surgery included maxillary Le Fort I osteotomy in two segments (anterior cleft gap narrowing, fistula closure, horizontal advancement, vertical lengthening, and interpositional grafting); bilateral sagittal split ramus osteotomies (clockwise rotation); osseous genioplasty (vertical shortening and horizontal advancement); and septoplasty, inferior turbinate reduction, and nasal recontouring. The dental gap was surgically narrowed from a four-tooth gap to a two-tooth gap. With successful healing and the completion of orthodontics, prosthetic rehabilitation of the maxillary arch was carried out. **A,** During the early mixed dentition, before grafting. **B,** During the mixed dentition, after unsuccessful grafting.

Residual alveolar clefts and mobile pre-maxilla despite bone graft



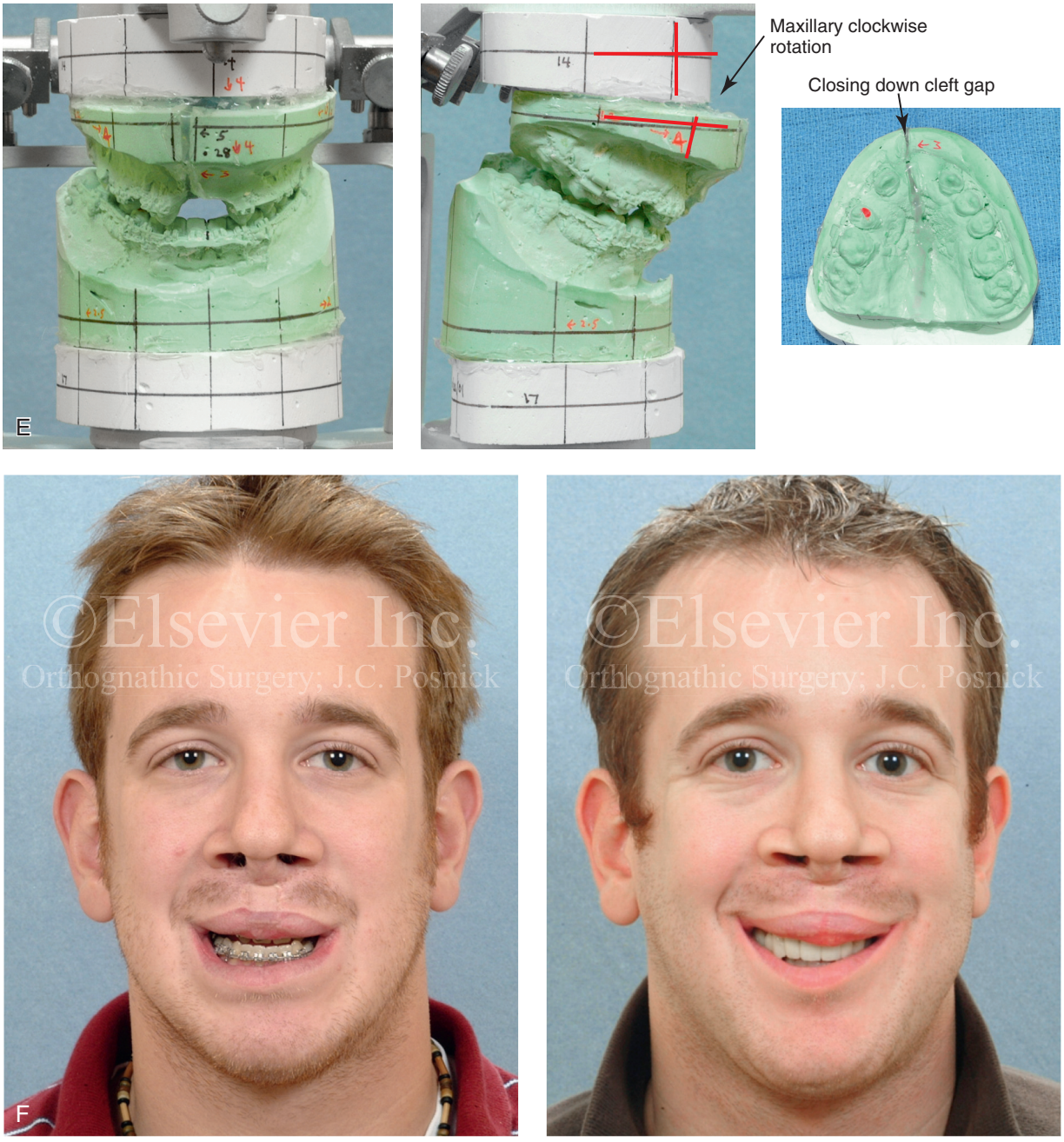


Aseptic necrosis/loss of premaxilla after circumvestibular incision



• **Figure 33-17, cont'd C,** As a teenager, before orthognathic surgery. **D,** After unsuccessful orthognathic surgery at another institution, including aseptic necrosis of the premaxilla and fistula formation.

Continued



• **Figure 33-17, cont'd E,** Articulated dental casts that indicate analytic model planning for redo orthognathic surgery. **F,** Frontal views with smile before and after redo orthognathic surgery and dental rehabilitation.

• **Figure 33-17, cont'd G,** Occlusal views after unsuccessful orthognathic surgery at another institution with the loss of the premaxilla and after redo orthognathic surgery and dental rehabilitation. **H,** Palatal views before and after redo orthognathic surgery and rehabilitation. **I,** Profile views before and after reconstruction and rehabilitation. **J,** Lateral cephalometric radiographs before and after redo orthognathic surgery.

to provide circulation to the premaxilla at the time of a Le Fort I down-fracture through a circumvestibular incision.

5. *Chin dysplasia.* The patient with BCLP is often a forced mouth breather with an open-mouth posture. In addition, if a pharyngoplasty was placed for management of VP insufficiency, then a chronic open mouth posture may also be present. Secondary deformity of the chin often occurs as a result of the longstanding open-mouth posture during growth (see Chapter 4). The deformity is characterized by excess vertical height and horizontal deficiency (see Chapter 37).
6. *Mandibular dysplasia.* True mandibular prognathism is uncommon in the patient with BCLP. Mandibular ramus osteotomies should be used to correct secondary deformities such as facial asymmetry, skeletal disproportions, and the occasional true anteroposterior malformation.
7. *Nasal obstruction and sinus blockage.* Obstructed breathing through the nose and frequent sinusitis are typical in the individual who is born with BCLP. This results from a combination of septal deviation, enlarged inferior turbinates, tight nasal apertures, and scarred (collapsed) internal and external nasal valves (see Chapter 10). A pharyngeal flap may also contribute to nasal obstruction, mucous trapping, and sinusitis. The presence of obstructive sleep apnea should be considered and ruled out (see Chapter 26).
8. *VP dysfunction.* It is estimated that approximately 20% of patients with a repaired cleft palate demonstrate VP dysfunction by the time they are 5 years old. The adolescent patient with BCLP who arrives for the evaluation of a jaw deformity may have a pharyngeal flap in place. Despite the presence of a pharyngeal flap, the patient still may have VP dysfunction (see Chapter 8). Although a Le Fort I osteotomy with advancement is likely to improve the upper airway and speech articulation, it can have a negative impact on VP function. These disparate effects of the surgical plan should be taken into account and shared with the patient and the family (see chapter 8).

Orthodontic Considerations in the Bilateral Cleft Lip and Palate Patient with a Jaw Deformity

The patient with BCLP who has not undergone effective bone grafting during the mixed dentition will have three separate maxillary segments, each with a degree of dysplasia in all three planes. Each segment should be assessed and treated individually by the orthodontist in anticipation of segmental surgical repositioning. Radiographic analysis is essential before any orthodontic movement is initiated in the patient with BCLP. The Panorex film is useful for assessing the overall jaw, the dental anatomy, and the tooth angulation. Maxillary occlusal and periapical radiographs through the cleft sites are of some help, but a cone beam computed tomography scan is ideal to assess alveolar bone volume, including the height of the alveolar crest, the fenestrations and dehiscence of the walls, and, more importantly, the integrity of the alveolus across each cleft site.

Both the number of permanent incisors and the amount of dentoalveolar bone in the premaxilla differ widely. Lateral incisor-like teeth are frequently found along the edges of the premaxilla or the lateral segments. These are generally rudimentary, with poor root form, and they are best removed. Unerupted supernumerary teeth should be extracted either at the time of bone grafting during the mixed dentition or in conjunction with orthognathic surgery. A useful lateral incisor at the cleft site is only found in 7% of patients. The lateral incisor's usefulness should be assessed in accordance with root (rather than crown) morphology (see the discussion of orthodontic space closure versus prosthetic replacement later in this chapter).[13]

The decision to extract additional teeth in the lateral segments depends on the width and height of available alveolar bone and the degree of dental root crowding in each segment. Extractions of the first bicuspids may be necessary to ensure that there is adequate bone for leveling and aligning teeth in each lateral segment without irreversibly weakening the periodontal support of the dental roots next to the clefts.

The placement of preangulated brackets or artistic positioning bends can displace the root apices from their alveolar housing, with a rapid loss of crestal bone height along the mesial of the cuspid and distal of the incisor. Excessive mesial tipping of the canine should also be avoided. Some patients lack sufficient bone in the premaxilla to fully align the incisors preoperatively. In these cases, more complete orthodontic straightening is best accomplished after the cleft defects are surgically closed and healed.

The incorporation of all erupted and maintained maxillary teeth (including the second molars) into the orthodontic mechanics will facilitate the development of the desired postoperative arch form. A lingual attachment on the first molar band allows for the use of a transpalatal appliance for indirect anchorage to assist with bringing the second molars into alignment.

The development of an ideal mandibular arch is critical to the effective repositioning of the upper maxillary segments in the most favorable position at the time of orthognathic surgery. Extractions in the mandibular arch are generally not required, but this depends on the space requirements needed to position the incisors ideally over the basal bone.

Immediate Presurgical Assessment

Two to six weeks before the operation, the orthodontist will confirm that the preoperative orthodontic objectives have been met and place the passive surgical wires. The surgeon

then takes final records, including alginate impressions of the maxillary and mandibular arches, a centric bite registration, a face-bow registration, and direct facial measurements. Past medical and dental records (e.g., radiographs, consultation reports, facial and occlusal photographs, dental models, special studies) are reviewed. Computed tomography scan views of the cleft alveolus can be helpful to assess bone volume. Decisions are finalized regarding preferred vector changes of the jaws and the precise linear distances and angles to be accomplished in each jaw for the desired results (see Chapter 12).[2,37,38,74,75] Analytic model planning is carried out on the articulated dental casts, and splints are fabricated. The splints assist with the achievement of the precise occlusion and the preferred facial aesthetics that were determined preoperatively (see Chapter 13).

Orthognathic Approach for Bilateral Cleft Lip and Palate Deformities

Evolution of Surgical Technique

Surgical attempts to correct jaw disharmonies in patients with BCLP date back to Steinkamm's 1938 description of a Le Fort I osteotomy in a patient with BCLP.[123] The early literature warned of complications with maxillary osteotomy in patients with BCLP and offered incomplete descriptions of surgical techniques to guide the surgeon in the performance of safe and reliable osteotomies.[9,26-30,41,42,68] However, Hugo Obwegeser's milestone contributions to cleft skeletal reconstruction are important (see Chapter 2).[82-88] Although he succeeded in advancing a cleft maxilla to the preferred occlusion without the need for a mandibular set-back, other clinicians did not share his enthusiasm. Jackson and colleagues stated that patients with BCLP who required jaw surgery must first undergo successful fistula closure and bone grafting, and they postulated that "only then should a Le Fort I or Le Fort II procedure be considered."[61] Ward-Booth and others described a Le Fort II osteotomy that was designed to protect the circulation to the premaxillary segment.[135] In 1974, Willmar described the complications associated with the Le Fort I osteotomy.[138] Eight of the 106 patients in this study had BCLP, and one died after surgery. However, details of the operative technique were not mentioned. In 1980, Sinn described modifications for bilateral maxillary alveolar cleft repair in combination with Le Fort I osteotomy.[122] He suggested mobilization of the lateral segments and closure of the bilateral fistula with cheek flaps. Tideman and colleagues described at least one patient with BCLP who underwent a Le Fort I advancement through limited vertical incisions and subperiosteal tunneling for osteotomy exposure.[130] Westbrook and associates described simultaneous maxillary advancement and the closure of bilateral clefts and oronasal fistulas.[136] They used limited incisions with the objective of maintaining maximum blood supply to the lateral maxillary segments, but this prevented direct exposure of the maxillary walls and full mobilization. The authors did not mention surgical dental gap closure in the single patient with BCLP. James and Brook,[62] Poole and colleagues,[97] and others also described cleft–orthognathic techniques that involved the use of limited incisions and indirect exposure with subperiosteal tunneling. They believed that the staging of the fistula closure and the Le Fort I osteotomy was necessary to avoid premaxillary segment necrosis.

Posnick clarified the safety of segmental Le Fort I osteotomy techniques for patients with a BCLP jaw deformity. The documented favorable results confirm its value for the routine, one-stage management of those with a residual fistula, alveolar clefts, and dental gaps.[98-113] The technique is based on an understanding of the circulatory requirements of each of the three maxillary units in a patient who has not undergone a successful bone graft to unite the clefted segments. The authors confirmed that each of the three segments can be safely osteotomized, fully mobilized, and relocated in accordance with the patient's functional needs and aesthetic objectives. The premaxilla can be osteotomized (i.e., Vomer osteotomy) and raised as a dento–osseous–musculo–mucosa flap that is based on the labial mucosal pedicle. This technique was first described by Wunderer in 1962 and used extensively in patients without clefts.[144] Attention to detail is necessary to preserve the attachments of the labial soft-tissue–mucosal pedicle to the premaxilla. The validity of this flap's circulation was proven by Bell and colleagues in rhesus monkeys (see Chapter 2).[5] The labial soft-tissue pedicle ensures flap survival and safe repositioning of the premaxillary (dento–osseous–musculo–mucosa) segment.[7] The technique of maintaining a blood supply to the down-fractured posterior (lateral) segments after a direct circumvestibular incision was originally described by Schuchardt and later confirmed experimentally by Bell.[6]

Standard Le Fort I Osteotomy

The subgroup of patients with BCLP with intact alveolar ridges on one or both sides may have been born with bilateral clefts of their lips but with intact alveolar ridges on both sides, an intact alveolar ridge on just one side, or alveolar clefts that have been successfully grafted during the mixed dentition on one or both sides. For the adolescent or adult with BCLP who presents with a jaw deformity and intact alveolar ridges on both sides, a standard Le Fort I down-fracture can be carried out to correct the maxillary deformity (see Chapter 15) (Figs. 33-2 through 33-5). The adolescent with BCLP with an intact alveolar ridge on one side (the other side having residual alveolar clefting and an oronasal fistula) is essentially presenting with the same anatomy as a patient with unilateral cleft lip and palate who has not undergone any grafting. The surgical approach (technique) for this patient is the same as that described for the individual with an un-grafted unilateral cleft lip and palate (see Chapter 32) (Fig. 33-6). For all others born with BCLP and who present with un-grafted (open) alveolar clefts, the modified Le Fort I osteotomy in three segments

described in the next section should be considered (see Figs. 33-7 through 33-17).

Modified Le Fort I Osteotomy in Three Segments

Unfortunately, in current clinical practice, a percentage of patients with BCLP jaw deformities continue to present with alveolar clefts, residual oronasal fistulae, and mobile premaxillae. When completing a Le Fort I osteotomy in the BCLP patient with open alveolar clefts, specific incision placement is critical to maintain circulation to each of the three segments (see Figs. 33-7 through 33-17).

A buccal (labiolateral) incision is made on each side in the depth of the vestibule, which extends from the zygomatic buttress (just anterior and gingival to the parotid duct) forward to the location of the residual oronasal fistula. A vertical incision then continues down the mesial line angle of the canine (or, if the canine is missing, the most mesial tooth in each lateral segment). This mesial line angle vertical incision begins to separate the oral and nasal mucosa through the fistula. On the labial aspect of the premaxilla, a vertical incision then joins the more posterior vestibular incision and continues down along the distal line angle of the central incisor on each side to further separate the oral and nasal mucosa of the fistula. Care is required to prevent any disruption of or incision into the mucosa within the labial vestibule of the premaxilla.

The nasal and oral mucosa are also sharply incised and separated on the palatal aspect of the premaxilla and on the palatal aspect of each lateral segment. When this occurs, there is complete separation of all oral and nasal mucosa of the fistula. Care is taken to prevent any disruption or separation of the palatal mucosa from its underlying hard palate. Each lateral maxillary segment depends on this palatal soft tissue for circulation into the palatal bone.

Next, on the labial side, subperiosteal soft-tissue dissection exposes the anterolateral aspect of the maxilla of each lateral segment. A maxillary (Le Fort I type) osteotomy is then performed through the lateral, anterior, and medial maxillary walls using a reciprocating saw with a long, straight blade. The pterygomaxillary sutures are separated with a curved osteotome. The vomer is often not attached to either lateral segment. However, if it is, a guarded osteotome is used to separate it from the maxilla before the lateral segments can be successfully down-fractured. The lateral segments are then down-fractured and disimpacted for three-dimensional repositioning.

Next, attention is turned to completing an "out-fracture" of the premaxillary segment. The nasal mucosa is further dissected from the palatal mucosa on the posterior side of the premaxilla. The nasal mucosa and the soft tissues are pushed superiorly into the nose. An osteotomy is performed to free the premaxilla from the vomer. The osteotomy is generally performed using a reciprocating saw with a short, straight blade from the palatal side. It is imperative that the labial vestibule mucosa (pedicle) to the premaxilla remain connected to its underlying bone, because blood flows through this mucosa pedicle into the premaxillary bone and teeth. The nasotracheal tube is visualized and protected during the osteotomy that separates the vomer from the premaxillary flap. With the lateral segments down-fractured and the premaxillary segment out-fractured, the internal structures of the nose are directly visualized.

If the nasal septum is deviated, submucosal exposure allows resection of the obstructing portions of the vomer, the perpendicular plate of the ethmoid, and the quadrangular cartilage using a rongeur. The structural components of the quadrangular cartilage (i.e., the dorsal and caudal strut) are preserved to prevent a late saddle deformity. The inferior turbinates, if enlarged, are reduced along the whole inferior aspect of the turbinate. The raw surfaces are then cauterized for hemostasis. The reduction of hypertrophic inferior turbinates will both facilitate nasal-side mucosal flap closure and improve nasal airflow after healing. The nasal mucosal flaps are sutured to obtain as watertight of a nasal-side closure as possible.

The lateral maxillary segments are ligated into the prefabricated acrylic occlusal splint. This is followed by the ligation of the premaxillary segment. Through this procedure, the cleft–dental gap on each side and the dead space associated with the bony clefts are closed. To accomplish dental gap closure, it is generally necessary to remove spurs of alveolar bone at the interface of the lateral and premaxillary segments using a rotary drill with a small watermelon bur. This is done without perforation through the lamina dura, which could injure the dental roots. The differential advancement of the lateral segments also approximates the labial and palatal mucosal flaps for effective oral-side fistula closure. The suturing of the palatal mucosa is not necessary, because the segmental repositioning approximates the freshened mucosal edges along the palate. The palatal mucosa must remain adherent to the underlying bone to ensure adequate circulation to each of the lateral segments. All three segments are now secure in the prefabricated acrylic splint.

The maxilla, which has been secured to the splint, is advanced and repositioned to the mandible. The jaws are then secured together with intermaxillary fixation. The preferred vertical dimension, which was determined preoperatively, is achieved to improve the facial height and the lip-to-tooth relationships. Titanium plates are conformed across the osteotomy sites at each zygomatic buttress and each pyriform aperture and then secured with screws. The intermaxillary fixation is released, and the occlusion is checked.

The intermediate splint is removed and the lateral segments are secured with wires into the final splint. A small amount of autogenous (iliac) cancellous bone graft is packed along the floor of the nose and the cleft (hard) palate on each side before securing the premaxilla into the final splint. The dead space associated with the alveolar cleft will be effectively closed once the premaxilla is secured into the splint.

A bloc of corticocancellous iliac bone is crafted and interposed along the bony gap created on each side when the lateral segments were advanced. The graft is placed between the zygomatic and pyriform plates. An additional microplate is placed from the superior maxilla, across the graft to the inferior maxilla, and then secured with screws. With the cleft–dental gaps surgically closed, the labial gingival mucosa on each side is directly approximated without the need for mucosal advancement flaps. If indicated, mandibular and chin osteotomies and removal of wisdom teeth are completed in the usual sequence and then fixed in place (see Chapter 15).

After wound closure, the occlusion is maintained with elastics. The elastics may be removed for immediate postoperative airway management and then reapplied as indicated. Maintenance of the occlusion and retraining of the muscles are accomplished with intermittent elastics during the next 5 weeks. For the modified Le Fort I osteotomy in three segments, the splint frequently remains ligated to the maxillary orthodontic brackets for a total of 7 weeks. At the time of splint removal, the orthodontist replaces the segmented maxillary surgical wires for a continuous rigid one. The treating orthodontist actively maintains the surgically achieved arch form and cleft–dental gap closures. The placement of a transpalatal appliance secured into the lingual tubes on the maxillary first molar bands may be a useful adjunct to maintain the arch form after the splint is removed.

Avoiding Pitfalls

Mobilization of the Down-Fractured Cleft Maxilla

Full mobilization of the Le Fort I osteotomy in the patient with BCLP is the critical step needed for the achievement of accurate intraoperative positioning and then long-term maintenance of the maxilla. The intraoperative use of both pterygoid and nasomaxillary disimpaction forceps is important for full mobilization. Taking the necessary time to slowly complete the disimpaction (10 to 15 minutes) is often crucial to the achievement of the desired results. Gaining experience—first through observing and assisting an experienced cleft orthognathic surgeon and then by using one's own hands—is essential to achieving confidence in the mobilization process.

Horizontal Advancement and Vertical Lengthening of the Cleft Maxilla

The surgeon must often carry out significant advancement and vertical lengthening of the BCLP maxilla to fully enhance facial aesthetics, to correct the occlusion, and to open the airway. Scarring of the palatal soft tissues from previous repairs may be of concern when considering significant advancement.

In general, to achieve full skeletal correction, all or most of the negative overjet should be managed in the maxilla. Most deficient BCLP maxilla will also require vertical lengthening. A repaired bilateral cleft lip that has minimal elevation with smile or that appears short and hypotonic may caution the surgeon against the vertical lengthening that the midface requires. The surgeon should fight the tendency to "underlengthen" the midface.

After the maxilla is fully mobilized and positioned, as described previously, stabilization is with titanium plates and screws (a plate with screws at each zygomatic buttress and pyriform rim).[71] The placement of a bloc corticocancellous graft wedged into the dead space between the zygomatic buttress and the pyriform rim plate on each side is generally necessary (see Chapter 18). Further stabilization across each graft with a titanium microplate and screws is also required (see Chapter 15). When a modified (three-segment) Le Fort I osteotomy is carried out, the soft-tissue pedicle that provides circulation to each bone flap must be protected during mobilization, graft placement, and stabilization, as described previously.

Closure of Residual Oronasal Fistula and Alveolar Cleft Management

Ideally, full closure of residual oronasal fistula and adequate alveolar bone grafting will have been carried out during the mixed dentition. If not, the modified Le Fort I in three segments (as described previously) for the patient with BCLP provides the opportunity for complete oronasal fistula closure on both the palatal and labial sides, the management of the cleft skeletal deficiencies, and the stabilization of the premaxilla without the need for direct alveolar grafting. Not uncommonly, grafting and fistula closure were carried out during the mixed dentition, but with only partial success. A residual fistula may remain, and the premaxilla may not have union with one or both lateral segments. Unfortunately, when completing a Le Fort I procedure in the patients with BCLP with residual fistula and non-bridging alveolar ridges, fistula closure and union across the ridge can only be accomplished with the use of the modified segmental approach, as described previously. Augmentation of an intact but hypoplastic alveolar ridge at the time of down-fracture is not advisable, because additional soft-tissue dissection around the alveolar ridge would be required with a risk of compromised circulation and aseptic necrosis. The option of placing a corticocancellous bloc graft into the alveolar defect at the time of Le Fort I down-fracture is also not feasible for the same reasons.

Managing the Nasal Cavity and the Nose

In the patient with BCLP, the inferior turbinates are generally enlarged and partially blocking the nasal cavity. If so, the reduction of the inferior turbinates will be beneficial for breathing and sinus drainage; this is simultaneously accomplished through the down-fracture (see Chapter 15 and illustrations in this chapter).

In the patient with BCLP, the septum (bone and cartilage) may be significantly deviated, thus further obstructing the airway. The completion of a submucosal resection to open the airway with preservation of the caudal and dorsal cartilaginous struts to prevent a saddle deformity is beneficial. This is accomplished through the Le Fort down-fracture (see Chapter 15 and illustrations in this chapter).

In the patient with BCLP, the pyriform rims and the nasal floor may be irregular, which results in negative effects on the airway and the nasal aesthetics. If successful grafting had previously been accomplished, recontouring of the pyriform rims, the nasal floor, and the nasal spine using a rotary drill with a watermelon bur through the circumvestibular incision is helpful to open the airway and to improve nasal aesthetics. This cannot be simultaneously accomplished with the three-segment modified Le Fort I procedure in these patients (see Chapter 15 and illustrations in this chapter).

Surgical attempts to control the nasal soft-tissue envelope with suturing techniques (e.g., the alar cinch stitch) after Le Fort I osteotomy are often suggested. In my experience, manipulating the nasal soft tissues with suturing techniques at the time of Le Fort I osteotomy in the patient with BCLP is often counterproductive to both the airway and the aesthetics, because it does not address the primary problem, as described previously. We anticipate that the nasal soft tissues will redrape over the repositioned and recontoured maxilla and the nasal base to result in an overall improvement in aesthetics. A definitive rhinoplasty is usually planned for 6 to 12 months after jaw surgery (see Chapter 38).

Immediately after surgery, the nasal cavity is not packed, and the septum is not stented. It is expected that coagulated blood will partially block the airway. The clots will dislodge and be self-eliminating by approximately 5 to 7 days after surgery. The use of saline nasal sprays and the institution of sinus precautions are both helpful during this time (see Chapter 11).

Management of the Mandibular Deformity

In the patient with BCLP, the mandible is often secondarily deformed. In a previously published study, we documented that, in a significant percentage of individuals with BCLP, the lower jaw will also benefit from sagittal split ramus osteotomies to improve facial symmetry and proportions.[108,112,113] The mandibular repositioning is not carried out in an attempt to avoid maxillary advancement but rather to improve overall morphology (see Chapter 15).

Management of the Chin Deformity

In the BCLP patient with maxillary hypoplasia, the chin is often secondarily deformed. Increased vertical length and a flat pogonion are typical. If these are present, an intraoral oblique inferior border osteotomy is simultaneously carried out with repositioning of the distal chin to accomplish the preferred morphology (see Chapters 15 and 37).

Clinical Management after Initial Surgical Healing

Managing the details of the in-hospital and at-home convalescence during initial healing is essential for a successful outcome after BCLP orthognathic surgery (see Chapter 11). Cephalometric and dental radiographs and facial and occlusal photographs are obtained at standard postoperative intervals to document clinical progress. The orthodontist sees the patient within 24 hours of splint removal (approximately 5 to 7 weeks after surgery) and replaces the maxillary sectional arch wires with a rigid continuous arch wire. The maxillary teeth are ligated together to maintain the surgical dental–gap closure and the overall arch form. Active orthodontic maintenance and finishing are started. The use of a transpalatal appliance (i.e., a wire or a palatal plate) may also be used to further stabilize the new arch form. Close monitoring by an orthodontist for skeletal and dental shifts during the first 6 months after surgery is essential.

Speech can be objectively reassessed 3 to 6 months after surgery. We prefer to evaluate VP function with the use of nasoendoscopic instrumentation when feasible. Definitive cleft soft-tissue procedures (e.g., cleft rhinoplasty, lip scar revision, pharyngeal flap or flap revision) can be carried out as early as 6 months after orthognathic surgery. After the orthodontic appliances are removed, any planned definitive dental restorative work can be finalized.

Orthognathic Surgery for Bilateral Cleft Lip and Palate: Review of Study

Patients and Methods

Posnick and Tompson assessed the clinical results of 33 consecutive adolescents (age range, 16 to 24 years; mean, 18 years) with BCLP who underwent orthognathic surgery by a single surgeon (Posnick) over a 6-year period using the modified Le Fort I osteotomy as described previously.[113] All patients underwent perioperative orthodontic treatment, and all were judged to be skeletally mature at the time of jaw surgery. The clinical follow-up period ranged from 1 to 7 years (mean, 40 months) at the close of the study. The country of origin of the patients varied (e.g., Canada, United States, Eastern Europe, Southeast Asia), as did their race (e.g., Caucasian, Asian, African). The patients' initial surgeons varied, as did their treatment protocols. All patients had undergone primary lip and palate repair during infancy. The number and extent of other operations (i.e., soft-tissue, lip, nasal, and palatal procedures) varied greatly (range: 1 to 10 procedures). Many patients had undergone additional

attempts to close the residual oronasal fistula as well as bone grafting to fill the alveolar clefts. The basic orthognathic procedures that were carried out included a modified Le Fort I osteotomy (n = 33). Ten of these patients also underwent simultaneous sagittal split ramus osteotomies of the mandible to correct secondary deformities. Fourteen underwent osseous genioplasty (vertical reduction and horizontal advancement) of varying degrees to improve profile aesthetics and lip posture.

In the study group, all of the patients with BCLP required closure of residual bilateral labial and palatal fistulas. Twenty-six of the 33 patients had a negative overjet of the incisors, which indicated horizontally maxillary hypoplasia. Twenty-eight of the 33 patients also had posterior crossbites in the lateral (lesser) segments, which indicated constriction of the maxillary arch despite orthodontic treatment. Thirty patients had a cleft–dental gap on each side, and one to three teeth were missing at each cleft site; one patient had a cleft–dental gap on just one side; and the remaining two patients had undergone successful gap closure via orthodontic treatment but had residual fistula and alveolar defects. For the 30 patients with bilateral cleft–dental gaps, simultaneous surgical closure via the differential advancement of the maxillary segments was planned. All 33 patients had mobile premaxillae, thereby indicating inadequate bone bridging across the cleft alveolar regions on each side. Of the 66 cleft sites, 59 had either congenitally absent or inadequate lateral incisors. Ninety-three percent of patients had an Angle Class III malocclusion on each side.

Results

All 33 adolescent patients with BCLP patients presented with residual oronasal fistulae, mobile premaxillae, and maxillary hypoplasia.[113] Twenty-six patients underwent successful one-stage complete fistula closure and full stabilization of the premaxillary segment through modified Le Fort I osteotomy, with differential maxillary segmental repositioning. The seven patients who retained small fistulas (all in the incisive foramen region) also retained a degree of mobility of the premaxillary segment. Final fistula closure involved the use of local flaps with additional bone grafting to achieve bony union of the premaxilla to the lateral segments.

All 33 patients successfully achieved and maintained keratinized gingiva over the cleft site and the cleft-adjacent teeth on each side. A degree of gingival recession occurred along the medial aspect of the canine (or the first premolar when the canine was absent) or the distal aspect of the incisor in 7 of the 132 cleft-adjacent teeth. None of the cleft-adjacent teeth required root canal therapy. Five patients required fixed prostheses to either replace missing teeth or to improve the aesthetics of the incisors as a result of poor enamel quality or congenital dysplasia. Thirty-one patients who had cleft–dental gaps underwent successful closure. In three patients, both the lateral incisor and the canine were missing, and a "closed down" but residual gap was planned. Dental restorative work was then carried out. Two other patients had undergone the orthodontic completion of gap closure.

The long-term maintenance of overjet and overbite was measured directly for each patient from the late (>1 year) postoperative lateral cephalometric radiograph. Most patients (31 of 33; 94%) maintained a positive overjet. Two of the 33 patients (6%) shifted into neutral overbite, and 4 of the 33 patients (12%) relapsed into a negative overbite. There were no cases of infection that required the extended use of antibiotics or drainage, and there was no postoperative hemorrhage that required a return to the operating room. No teeth or dentoalveolar segments were lost to aseptic necrosis, direct surgical trauma, infection, or any other cause. No patients underwent reoperation (orthognathic procedures) for suboptimal facial aesthetics or significant residual malocclusion.

Controversies and Unresolved Issues

Staging of Maxillary Reconstruction

The described modified Le Fort I osteotomy method for the management of the maxillary deformity in an adolescent or adult patient with BCLP who also presents with missing teeth, oronasal fistulae, alveolar defects, cleft–dental gaps, and nasal obstruction is not intended to replace standard techniques and the accepted sequencing of treatment earlier during childhood.[99] However, it does offer an effective reconstructive option after the opportunity for grafting the alveolus during the mixed dentition before the eruption of the permanent canines is lost and if a jaw deformity coexists. A two-stage approach for the adolescent with BCLP with maxillary hypoplasia, residual alveolar clefts, oronasal fistulae, cleft–dental gaps, a mobile premaxilla, and nasal obstruction is neither cost-effective nor time-efficient, and there may be increased morbidity. It is in these patients with BCLP that the modified Le Fort I osteotomy in three segments with the differential repositioning of each segment offers a reasonable opportunity for correction.[98-113]

Closure of Large Residual Palatal Fistula

Other indications for maxillary segmental osteotomies include the small group of adults (i.e., those in the permanent dentition) with BCLP who may have a satisfactory overjet or overbite but who have large residual palatal and bilabial fistulae, cleft–dental gaps, and mobile premaxillae. The difficulty in achieving successful fistula closure is the lack of oral-side soft tissue to secure a watertight palate closure in the incisal foramen region. In general, there are only two surgical options for management. One consideration is an anteriorly based dorsal tongue flap that is combined with an autogenous bone graft. If the size of the

fistula, the amount of dead space, and the tissue deficit is extensive, even a tongue flap and a bone graft will likely fail. The best option in these difficult cases is to obturate the fistula and to maintain the cleft–dental gaps with a temporary partial denture until early skeletal maturity (i.e., adult dentition). A modified Le Fort I osteotomy (either two or three segments) with differential repositioning is then performed. By advancing the lateral segments, closure of the fistula, dental gaps, and dead space can generally be achieved (Fig. 33-9). If there is horizontal, transverse, or vertical deficiency or deformity of the maxilla, it is simultaneously managed.

Management of Complex Dental Rehabilitation

Another indication for the modified Le Fort I osteotomy (two or three segments) in the adult with BCLP is to assist with complex dental rehabilitation when excessive cleft and congenital anodontia gaps exist, with or without residual fistula. There are times when horizontal maxillary projection may be satisfactory; however, a functional arch form can best be achieved through segmental Le Fort I osteotomies that are planned in conjunction with dental rehabilitation (Fig. 33-15).

Malocclusion after Modified Le Fort I Osteotomy

Despite the advantages of the described modified Le Fort I osteotomy for complex BCLP deformities, there are two occasional unresolved long-term shortcomings of this procedure. The first is the tendency for medial, superior, and posterior drift (relapse) of the lateral segments after surgical correction. To combat this relapse pattern, planned surgical overcorrection as well as rigid transverse maintenance early after surgery with an acrylic splint and then medium term (6 months) orthodontic retention techniques are essential. The second unresolved problem is the less than ideal success rate of 80% to 85% for complete fistula closure and premaxillary segment stabilization to the lateral segments. This is a result of the limited bone volume of the premaxilla in many patients and the inability to apply plate and screw stabilization (i.e., of the premaxilla to each lateral segment) without compromise of the anterior maxillary circulation, which is dependent on the labial mucosa pedicle.

Philosophy Concerning the Importance of a "28-Tooth Angle Class I Occlusion"

Another controversy for the reconstruction of the patient with BCLP centers on a philosophy of the importance of achieving a "28-tooth Angle Class I occlusion" with traditional "cuspid protection" rather than accepting "group function" during lateral excursions.[10,45,49,119,124,126,127] The achievement of a 28-tooth occlusion would require non-extraction therapy and the maintenance of each cleft–dental gap (i.e., lateral incisor region), followed by a single-tooth dental implant placement or the use of another prosthetic option. Unfortunately, in the maxillary-deficient patient with BCLP, dental crowding generally exists, and extractions or closure of the dental gaps will be required to avoid long-term periodontal sequelae.

In a non-cleft population, only a few studies compare the results of orthodontic space closure (OSC) with prosthetic replacement for the management of congenitally absent maxillary lateral incisors. The absence of high-quality clinical research to evaluate an implant replacement option (as apposed to a bridge) continues to fuel the discussion of what constitutes the optimal treatment of a missing lateral incisor in the patient with a cleft. It has been documented in several studies that, for an individual with an alveolar cleft, OSC results in a healthier periodontium and greater patient satisfaction than classic prosthetic replacement.[81] OSC in patients with clefts after successful alveolar bone grafting has the added advantage of allowing the canine to be positioned into the lateral incisor location, thereby resulting in an improved alveolar ridge morphology.[1,22] A single-tooth dental implant option for the management of the cleft–dental gap in the patient with BCLP is an attractive theoretical alternative, but it requires the presence of an intact maxilla (i.e., the stability of the premaxilla to each lateral segment) with adequate alveolar bone volume, interproximal crestal height, and sufficient attached gingiva at each cleft site for favorable implant placement, dental aesthetics, and long-term maintenance.[49,72,91,93,115,116,128,129,133,134,146]

Unfortunately, these anatomic parameters are not often met in patients with BCLP. Furthermore, these dental procedures are rarely covered by medical insurance, which adds to the family's financial burden. This approach also requires the meticulous coordination of a spectrum of dental specialists (e.g., orthodontist, periodontist, prosthodontist, surgeon) who have expertise and dedication in the area of cleft deformity treatment. Even if implant placement and crown restoration are successful, this will create a long-term high-maintenance dentition for the patient. This combination of factors usually favors OSC when feasible or surgical space (dental gap) closure (via modified Le Fort I osteotomy) when appropriate. OSC is preferably accomplished by successful mixed-dentition grafting and orthodontic dental gap closure. Oosterkamp and colleagues completed a retrospective study of adults with BCLP who were missing a permanent lateral incisor at each cleft site.[89] The patients were treated either with orthodontic space closure (n = 17) or via prosthetic replacement (n = 10). The purpose of the study was to compare the dental aesthetics and function of OSC versus those of the prosthetic replacement of the lateral incisors in patients with BCLP. The predominant mode of prosthetic replacement was resin-bonded bridges. Dental aesthetics were evaluated by the patients themselves and by a professional panel. Mandibular function was evaluated by means of the standardized

Mandibular Function Impairment Questionnaire. The level of mandibular impairment was calculated with the use of the standardized Functional Impairment Rating Scale. With respect to dental aesthetics, no significant differences between patients treated with OSC and prosthetic replacement were found. With respect to function, the level of mandibular impairment was significantly higher among patients who were treated with prosthetic replacement as compared with those treated with OSC.

Velopharyngeal Function after Le Fort I Advancement

Uncertainties about VP function and the management of an in-place pharyngeal flap should no longer be limiting factors when orthognathic surgery is necessary in a patient with BCLP. A nasoendoscopic guided examination by a speech pathologist and a surgeon who is familiar with cleft anatomy can closely predict current and expected VP function in a patient who is scheduled for a Le Fort I osteotomy (see Chapter 8). When postoperative VP deterioration is anticipated, the patient and the family are counseled about the sequencing of treatment. Clinical studies have now documented that VP function will deteriorate in a similar fashion when either DO or standard Le Fort I osteotomy techniques are used in the patient with a cleft patient.[14,15,44,47,63,67,73,76,94,131] Despite the frequent need for significant maxillary advancement to normalize the skeleton and the facial aesthetics of patients with BCLP, we have not had to or seen an advantage to transecting an in-place pharyngeal flap to achieve maxillary mobilization and the desired advancement. Our research and that of others confirms that a pharyngeal flap in place at the time of Le Fort I osteotomy does not increase complications nor does it result in a higher incidence of relapse.[113] The definitive reassessment of VP function after cleft Le Fort I advancement can be carried out 3 to 6 months after surgery. A primary or revision pharyngeal flap can be carried out 6 months after orthognathic correction in conjunction with cleft rhinoplasty or labial revision, if indicated.

Mixed-Dentition Le Fort I Osteotomy

By the mid 1980s, research clarified that, if jaw surgery is undertaken during the mixed dentition in the patient with a cleft palate, then another procedure to advance the maxilla will likely be required after skeletal maturity is reached.[140-144] More recently, several investigators again tested this theory by proceeding with mixed-dentition Le Fort I osteotomies in these patients using DO techniques.[16,34,35,46,48,59,95] All research to date indicates that the Le Fort I advancement carried out during the mixed dentition in the patient with clefting—whether by standard or DO techniques—results in no further significant horizontal growth. If the mandible continues to grow in length, an Angle Class III malocclusion will occur with the need for additional Le Fort I advancement or mandibular set-back.

Skeletal Relapse after Le Fort I Osteotomy in Patients with Clefting

There are more than 100 published articles reviewing skeletal stability and relapse in patients with clefting who have had Le Fort I advancement with the use of either standard osteotomies or DO techniques.[3,11,12,17-19,31,32,39,40,51,54-58,60,65,70,78,96,125,139] These studies do not demonstrate convincing differences in relapse patterns between the two techniques. Proponents of DO techniques frequently state that, when more than 10 mm of maxillary advancement is required, the use of standard Le Fort I osteotomy techniques with plate and screw fixation and bone grafts may lead to a greater degree of relapse. To put this in perspective, it should be noted that only 5% of patients with clefting who are undergoing Le Fort I advancement will require 10 mm or more of horizontal advancement at the incisors. All clinicians will agree that this subgroup of cleft patients (5%) is the most challenging. The reasons for this go beyond the degree of horizontal maxillary deficiency. These patients are also likely to present with multiple residual end-stage deformities (see the section about residual skeletal deformities earlier in this chapter) and previously failed surgical procedures. Aksu and colleagues documented a horizontal maxillary relapse rate of 22% after Le Fort I osteotomy in patients with clefting who were treated with DO techniques.[4] This was found at the 3-year follow-up evaluation in a series of adult patients with repaired cleft lips and palates who presented with maxillary hypoplasia.

He and colleagues reported their results of treating adolescents with repaired cleft lip and palate and maxillary hypoplasia with Le Fort I advancement via an external DO device.[50] The patients (n = 17) were treated at one center between 2000 and 2006 and had at least 1 year of follow up and a full set of records. DO treatment started on day 5 (1 mm/day) and continued until a Class II occlusion (overcorrection) was achieved. Consolidation time ranged from 4 to 12 weeks, and this was followed by several more months of face mask therapy. The first four treated patients—two with unilateral cleft lip and palate and two with BCLP—developed fibrous non-union; all four required reoperation and rigid fixation to achieve bony union. The authors then extended the consolidation period to a minimum of 12 weeks for the remaining patients (n = 13), at which point all patients had achieved bony union. In the group that achieved satisfactory bony union (13 out of 17 patients; 76%) the mean horizontal relapse was 11.9%, with 5 out of 13 patients (38%) developing no better than "end-to-end" occlusion. Another orthognathic procedure was necessary to obtain a satisfactory occlusion in 5 of the 13 patients (38%) who had initially achieved bony union. Overall, 9 out of 17 patients (53%) required two orthognathic procedures.

In 2011, Chen and colleagues reported a 30.7% incidence of horizontal relapse after Le Fort I osteotomy with the use of DO techniques in patients with clefting who

presented with Class III malocclusion.[16] Interestingly, Posnick and colleagues documented a more limited degree of horizontal relapse and overall success when using the modified Le Fort I techniques described for cleft patients.[113] Stability and relapse were measured by a review of postoperative (≥1 years) cephalometric analysis and clinical examination.[113] This was reported by cleft type: 94% of patients with BCLP maintained a positive overjet long term; those with unilateral cleft lip and palate achieved 6.9 mm of advancement, with 5.3 mm maintained for the long term; and those with isolated cleft palate (ICP) had 6.1 mm of advancement, with 5.1 mm maintained over the long term. All of the ICP patients maintained positive overjet long-term.

Standard Approach versus Distraction Osteogenesis Approach to Le Fort I Advancement

I agree with Obwegeser when he stated the following: "Today, many surgeons will resort to the use of distraction devices to gradually advance the cleft maxilla. Although in some circumstances this may be appropriate, it should be remembered that most cleft patients can be treated efficiently, even when requiring significant advancement with the classic Le Fort I type procedure as described."[87]

Standard thinking is for the surgeon to commit to an approach (DO versus standard technique) for Le Fort I advancement before he or she arrives in the operating room. The surgeon then sticks to the plan, no matter what happens intraoperatively. On the basis of a review of the literature and almost three decades of personal experience as a cleft jaw surgeon, I make the following observations and recommendations in this area.

Observations About the Distraction Osteogenesis Approach

- DO techniques are always the patient's second choice when compared with the standard approach of Le Fort I osteotomy and rigid internal fixation. The length of downtime after surgery involving DO is longer (≥3 months of limited diet and physical activity), and it is followed by several additional months of face mask therapy. There is no reduction in the perioperative complications when using DO techniques rather than standard techniques. The device is also awkward and socially embarrassing, and it typically blocks the patient's visual fields (e.g., the RED, BLUE, or GREEN external DO devices).
- If the cleft maxilla is down-fractured, adequately mobilized, and stabilized with interpositional bone grafts and plate and screw fixation, it has a high probability of healing as planned with a predictable and less extensive convalescence than DO.
- An experienced cleft orthognathic surgeon will be more confident in his or her ability to down-fracture and fully mobilize the BCLP maxilla than a less experienced surgeon. Therefore, the less experienced surgeon will be more likely to choose DO techniques to avoid the personal stress of the intraoperative maxillary mobilization process.
- Even for the experienced cleft orthognathic surgeon, there will be the occasional patient in whom the extent of maxillary hypoplasia and associated deformities (e.g., missing teeth, lack of alveolar bone, residual fistula) will lead him or her to choose DO techniques. Despite its protracted healing requirements and its limited ability to correct all of the cleft deformities, the DO device's gradual stretching ability can mobilize even the most recalcitrant maxilla (see Fig. 32-21).

Recommendations for the Distraction Osteogenesis Approach

For most patients with BCLP and maxillary hypoplasia, reserving DO techniques to serve as a "bail out" when the down-fractured maxilla cannot be adequately mobilized is the approach that I recommend. The patient and family at risk can be informed of this contingency plan in advance. This approach allows the surgeon to have flexibility in the operating room to make the right decision at the right time for the patient in need to achieve the most efficient convalescence and the most favorable long-term objectives.

Conclusions

Clinical studies document a high prevalence of jaw deformities in mature patients with BCLP. Safe and reliable surgical methods are now available to manage the presenting cleft jaw deformity, malocclusion, and other simultaneous residual defects (e.g., oronasal fistulae, bony defects, cleft–dental gaps, nasal obstruction). These techniques offer the surgeon an opportunity to improve the patient's head and neck function, to enhance the patient's facial aesthetics, and to positively affect the patient's long-term quality of life and well-being.

References

1. Abyholm FE, Bergland O, Semb G: Secondary bone grafting of alveolar clefts: A surgical/orthodontic treatment enabling a nonprosthodontic rehabilitation in cleft lip and palate patients. *Scand J Plast Reconstr Surg* 15:127, 1981.
2. Al-Waheidi EMH, Harradine NWT, Orth M: Soft tissue profile changes in patients with cleft lip and palate following maxillary osteotomies. *Cleft Palate Craniofac J* 35:535–543, 1998.

3. Araujo A, Schendel SA, Wolfort, LM, Epker BN: Total maxillary advancement with and without bone grafting. *J Oral Surg* 36:849–858, 1978.
4. Aksu M, Saglam-Aydinatay B, Akcan CA, et al: Skeletal and dental stability after maxillary distraction with a rigid external device in adult cleft lip and palate patients. *J Oral Maxillofac Surg* 68:254–259, 2010.
5. Bell WH: Revascularization and bone healing after anterior maxillary osteotomy: A study using adult rhesus monkeys. *J Oral Surg* 27:249–255, 1969.
6. Bell WH, Levy BM: Revascularization and bone healing after posterior maxillary osteotomy. *J Oral Surg* 29:313, 1971.
7. Bell WH, You ZH, Finn RA, Fields RT: Wound healing after multisegmental Le Fort I osteotomy and transection of the descending palatine vessels. *J Oral Maxillofac Surg* 53:1425, 1995.
8. Berkowitz S: State of the art in cleft palate orofacial growth and dentistry: A historical perspective. *Am J Orthod* 74:564–576, 1978.
9. Bloomquist DS: Intraoperative assessment of maxillary perfusion during Le Fort I osteotomy [discussion]. *J Oral Maxillofac Surg* 52:831, 1994.
10. Boyne PJ, Sands NR: Secondary bone grafting of residual alveolar and palatal clefts. *J Oral Surg* 30:87, 1972.
11. Braun TW, Sotereanos GC: Orthognathic and secondary cleft reconstruction of adolescent patients with cleft palate. *J Oral Surg* 38:425, 1980.
12. Braun TW, Sotereanos GC: Orthognathic surgical reconstruction of cleft palate deformities in adolescents. *J Oral Surg* 39:255, 1981.
13. Cassolato SF, Ross B, Daskalogiannakis J, et al: Treatment of dental anomalies in children with complete unilateral cleft lip and palate at Sick Kids Hospital, Toronto. *Cleft Palate Craniofac J* 46(2):166–172, 2009.
14. Chanchareonsook N, Samman N, Whitehill TL: The effect of cranio-maxillofacial osteotomies and distraction osteogenesis on speech and velopharyngeal status: A critical review. *Cleft Palate Craniofac J* 43:477–487, 2006.
15. Chanchareonsook N, Whitehill TL, Samman N: Speech outcome and velopharyngeal function in cleft palate: Comparison of Le Fort I maxillary osteotomy and distraction osteogenesis—early results. *Cleft Palate Craniofac J* 44:23–32, 2007.
16. Chen P, Por Y, Liou EJ, Chang FC: Maxillary distraction osteogenesis in the adolescent cleft patient: Three-dimensional computed tomography analysis of linear and volumetric changes over five years. *Cleft Palate Craniofac J* 40:445–454, 2011.
17. Cheung LK, Chua HD: A meta-analysis of cleft maxillary osteotomy and distraction osteogenesis. *Int J Oral Maxillofac Surg* 35:14, 2006.
18. Cho BC, Kyung HM: Distraction osteogenesis of the hypoplastic midface using a rigid external distraction system: The results of a one- to six-year follow-up. *Plast Reconstr Surg* 118:1201, 2006.
19. Cohen SR, Burnstein FD, Stewart MB, Rathburn MA: Maxillary-midface distraction in children with cleft lip and palate: A preliminary report. *Plast Reconstr Surg* 99:1421–1428, 1997.
20. Cohen SR, Corrigan M, Wilmot J, Trotman CA: Cumulative operative procedures in patients aged 14 years and older with unilateral or bilateral cleft lip and palate. *Plast Reconstr Surg* 96:267–271, 1995.
21. Correa Normando AD, da Silva Filho OG, Capelozza Filho L: Influence of surgery on maxillary growth in cleft lip and/or palate patients. *J Craniomaxillofac Surg* 20:111, 1992.
22. da Silva-Filho OG, Teles SG, Ozawa TO, Filho LC: Secondary bone graft and eruption of the permanent canine in patients with alveolar clefts: literature review and case report. *Angle Orthod* 2000; 70:174–178.
23. Daskalogiannakis J, Mehta M: The need for orthognathic surgery in patients with repaired complete unilateral and complete bilateral cleft lip and palate. *Cleft Palate Craniofac J* 46:498–502, 2009.
24. David DJ, Smith I, Nugent M, et al: From birth to maturity: A group of patients who have completed their protocol management. Part III. Bilateral cleft lip-cleft palate. *Plast Reconstr Surg* 128:475–484, 2011.
25. DeLuke DM, Marchand A, Robles EC, Fox P: Facial growth and the need for orthognathic surgery after cleft palate repair: Literature review and report of 28 cases. *J Oral Maxillofac Surg* 55:694–698, 1997.
26. Dodson TB, Neuenschwander MC: Maxillary perfusion during Le Fort I osteotomy after ligation of the descending palatine artery. *J Oral Maxillofac Surg* 55:51, 1997.
27. Dodson TB, Neuenschwander MC, Bays RA: Intraoperative assessment of maxillary perfusion during Le Fort I osteotomy. *J Oral Maxillofac Surg* 52:827, 1994.
28. Drommer R: Selecting angiographic studies prior to Le Fort I osteotomy in patients with cleft lip and palate. *J Maxillofac Surg* 7:264, 1979.
29. Drommer R: The history of the "Le Fort I-Osteotomy." *J Maxillofac Surg* 14:119–122, 1986.
30. Drommer R, Luhr HG: The stabilization of osteotomized maxillary segments with Luhr miniplates in secondary cleft surgery. *J Maxillofac Surg* 9:166–169, 1981.
31. Erbe M, Stoelinga PJW, Leenen RJ: Long-term results of segmental repositioning of the maxilla in cleft palate patients without previously grafted alveolo-palatal clefts. *J Craniomaxillofac Surg* 24:109, 1996.
32. Eskenazi LB, Schendel SA: An analysis of Le Fort I maxillary advancement in cleft lip and palate patients. *Plast Reconstr Surg* 90:779–786, 1992.
33. Felstead AM, Deacon S, Revington P: The outcome for secondary alveolar bone grafting in the southwest UK region post-CSAG. *Cleft Palate Craniofac J* 47:359–362, 2010.
34. Figueroa AA, Polley JW, Friede H, et al: Long-term skeletal stability after maxillary advancement with distraction osteogenesis using a rigid external distraction device in cleft maxillary deformities. *Plast Reconstr Surg* 114:1382, 2004.
35. Figueroa AA, Polley JW, Ko EW: Maxillary distraction for the management of cleft maxillary hypoplasia with a rigid external distraction system. *Semin Orthod* 5:46, 1999.
36. Filho LC: Isolated influences of lip and palate surgery on facial growth: Comparison of operated and unoperated male adults. *Cleft Palate Craniofac J* 33:51, 1996.
37. Freihofer HPM Jr: The lip profile after correction of retro-maxillism in cleft and non-cleft patients. *J Maxillofac Surg* 4:136–141, 1976.
38. Freihofer HPM Jr: Changes in nasal profile after maxillary advancement in cleft and non-cleft patients. *J Maxillofac Surg* 5:20–27, 1977.
39. Gaggl A, Schultes G, Karcher H: Aesthetic and functional outcome of surgical and orthodontic correction of bilateral clefts of lip, palate, and alveolus. *Cleft Palate Craniofac J* 36:407, 1999.
40. Georgiade NG: Mandibular osteotomy for the correction of facial disproportion in the cleft lip and palate patient. *Symposium on Management of Cleft Lip and Palate and Associated Deformities* 8:238, 1974.
41. Gillies HD, Millard DR Jr: *The principles and art of plastic surgery*, Boston, 1957, Little, Brown.
42. Gillies HD, Rowe NL: L'ostéotomie du maxillaire supérieur enoisagée essentiellement dans le cas de bec-de-lièvre total. *Rev Stomatol* 55:545–552, 1954.
43. Good PM, Mulliken JB, Padwa BL: Frequency of Le Fort I osteotomy after repaired cleft lip and palate or cleft palate. *Cleft Palate Craniofac J* 44:396–401, 2007.
44. Guyette TW, Polley JW, Figueroa A, Smith BE: Changes in speech following maxillary distraction osteogenesis. *Cleft Palate Craniofac J* 38:199–205, 2001.
45. Hall HD, Posnick JC: Early results of secondary bone grafts in 106 alveolar clefts. *J Oral Maxillofac Surg* 41:289, 1984.
46. Harada K, Baba Y, Ohyama K, et al: Maxillary distraction osteogenesis for cleft lip and palate children using an external, adjustable, rigid distraction device: A report of 2 cases. *J Oral Maxillofac Surg* 59:1492, 2001.
47. Harada K, Ishii Y, Ishii M, et al: Effect of maxillary distraction osteogenesis on velopharyngeal function: A pilot study. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 93:538, 2002.

48. Harada K, Sato M, Omura K: Long-term maxillomandibular skeletal and dental changes in children with cleft lip and palate after maxillary distraction. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 102:292, 2006.
49. Harrison JW: Dental implants to rehabilitate a patient with an unrepaired complete cleft. *Cleft Palate Craniofac J* 29:485, 1992.
50. He Dongmei, Genecov DG, Barcelo R: Nonunion of the external maxillary distraction in cleft lip and palate: Analysis of possible reasons. *J Oral Maxillofac Surg* 68:2402–2411, 2010.
51. Hedemark A, Freihofer HP Jr: The behavior of the maxilla in vertical movements after Le Fort I osteotomy. *J Maxillofac Surg* 6:244, 1978.
52. Heidbuchel KLWM, Kuijpers-Jagtman AM: Maxillary and mandibular dental-arch dimensions and occlusion in bilateral cleft lip and palate patients from 3 to 17 years of age. *Cleft Palate Craniofac J* 34:21, 1997.
53. Heidbuchel KLWM, Kuijpers-Jagtman AM, Freihofer HPM: Facial growth in patients with bilateral cleft lip and palate: A cephalometric study. *Cleft Palate Craniofac J* 31:210, 1994.
54. Henderson D, Jackson IT: Combined cleft lip revision, anterior fistula closure and maxillary osteotomy: A one-stage procedure. *Br J Oral Surg* 13:33, 1975.
55. Hirano A, Suzuki H: Factors related to relapse after Le Fort I maxillary advancement osteotomy in patients with cleft lip and palate. *Cleft Palate Craniofac J* 38:1–10, 2001.
56. Hochban W, Ganss C, Austermann KH: Long-term results after maxillary advancement in patients with clefts. *Cleft Palate Craniofac J* 30:237–243, 1993.
57. Horster W: Experience with functionally stable plate osteosynthesis after forward displacement of the upper jaw. *J Maxillofac Surg* 8:176, 1980.
58. Houston WJB, James DR, Jones E, Kavvadia S: Le Fort I maxillary osteotomies in cleft palate cases. Surgical changes and stability. *J Craniomaxillofac Surg* 17:9–15, 1989.
59. Huang CS, Harikrishnan P, Liao YF, et al: Long-term follow-up after maxillary distraction osteogenesis in growing children with cleft lip and palate. *Cleft Palate Craniofac J* 44:274, 2007.
60. Hui E, Hägg EU, Tideman H: Soft tissue changes following maxillary osteotomies in cleft lip and palate and non-cleft patients. *J Craniomaxillofac Surg* 22:182–186, 1994.
61. Jackson IT: Cleft and jaw deformities. In Whitaker LA, Randall P, editors: *Symposium on reconstruction of jaw deformities*, St Louis, 1978, CV Mosby.
62. James D, Brook K: Maxillary hypoplasia in patients with cleft lip and palate deformity—the alternative surgical approach. *Eur J Orthop* 7:231, 1985.
63. Janulewicz J, Costello BJ, Buckley MJ, et al: The effects of Le Fort I osteotomies on velopharyngeal and speech functions in cleft patients. *J Oral Maxillofac Surg* 62:308–314, 2004.
64. Jorgenson RJ, Shapiro SD, Odiner KL: Studies on facial growth and arch size in cleft lip and palate. *J Craniofac Genet Dev Biol* 4:33, 1984.
65. Kanno T, Mitsugi M, Hosoe M, et al: Long-term skeletal stability after maxillary advancement with distraction osteogenesis in nongrowing patients. *J Oral Maxillofac Surg* 66:1833–1846, 2008.
66. Kapp-Simon KA: Psychological interventions for the adolescent with cleft lip and palate. *Cleft Palate Craniofac J* 32:104, 1995.
67. Ko EW, Figueroa AA, Guyette TW, et al: Velopharyngeal changes after maxillary advancement in cleft patients with distraction osteogenesis using a rigid external distraction device: 1-year cephalometric follow-up. *J Craniofac Surg* 10:312–320, 1999.
68. Lanigan DT: Discussion of: Wound healing after multisegmental Le Fort I osteotomy and transection of the descending palatine vessels. *J Oral Maxillofac Surg* 53:1433, 1995.
69. Leonard BJ, Brust JD, Abrahams G, et al: Self-concept of children and adolescents with cleft lip and/or palate. *Cleft Palate Craniofac J* 28:347, 1991.
70. Lisson JA, Trankmann J: Comparative surgery of osteotomized and nonosteotomized BCLP patients. *Cleft Palate Craniofac J* 34:1997.
71. Luhr HG: Zur stabilen osteosynthese bei unterkiefer–frakturen. *Dtsch Zahnarztl Z* 23:754, 1968.
72. Lund TW, Wade M: Use of osseointegrated implants to support a maxillary denture for a patient with repaired cleft lip and palate. *Cleft Palate Craniofac J* 30:418, 1993.
73. Marrinan EM, LaBrie RA, Mulliken JB: Velopharyngeal function in nonsyndromic cleft palate: Relevance of surgical technique, age at repair, and cleft type. *Cleft Palate Craniofac J* 35:95–100, 1998.
74. McCance AM, Moss JP, Fright WR, et al: Three-dimensional analysis techniques. Part 1: Three-dimensional soft-tissue analysis of 24 adult cleft palate patients following Le Fort I maxillary advancement: A preliminary report. *Cleft Palate Craniofac J* 34:36, 1997.
75. McCance AM, Orth M, Moss JP et al: Three-dimensional analysis techniques. Part 4: Three-dimensional analysis of bone, and soft tissue to bone ratio of movements in 24 cleft patients following Le Fort I osteotomy: A preliminary report. *Cleft Palate Craniofac J* 43:58–62, 1997.
76. McComb R, Marrinan E, Nuss RC, et al: Predictors of velopharyngeal insufficiency after Le Fort I maxillary advancement in patients with cleft palate. *J Oral Maxillofac Surg* 69:2226–2232, 2011.
77. McKinstry RE: *Cleft palate dental care: A historical perspective*, Arlington, Va, 2000, ABI Publications.
78. Molina F, Ortiz Monasterio F, de la Paz Aguilar M, Barrera J: Maxillary distraction: Aesthetic and functional benefits in cleft lip-palate and prognathic patients during mixed dentition. *Plast Reconstr Surg* 101:951, 1998.
79. Mølsted K, Brattström V, Prahl-Andersen B, et al: The Eurocleft study: Intercenter study of treatment outcomes in patients with complete cleft lip and palate. Part 3: Dental arch relationships. *Cleft Palate Craniofac J* 42:78–82, 2005.
80. Nanda SK: Patterns of vertical growth in the face. *Am J Orthod Dentofacial Orthop* 93:103–116, 1988.
81. Nordquist GG, McNeill RW: Orthodontic vs restorative treatment of the congenitally absent lateral incisor—long-term periodontal and occlusal evaluation. *J Periodontol* 46:139–143, 1975.
82. Obwegeser HL: Correction of the facial appearance of harelip and cleft palate patients by surgery on the jaws. Excerpta Medica International Congress Series No 141. Reconstructive surgery. Thermal injury and other subjects, pp 110-117, 1966.
83. Obwegeser HL: Surgery as an adjunct to orthodontics in normal and cleft palate patients. *Rep Congr Eur Orthod Soc* 42:343–353, 1966.
84. Obwegeser HL: Surgical correction of deformities of the jaws in adult cleft cases. Paper read at the First International Conference on Cleft Lip and Palate, Houston, Tex, 14-17, April 1969.
85. Obwegeser HL: Surgical correction of small or retrodisplaced maxillae: The "dish-face" deformity. *Plast Reconstr Surg* 43:351, 1969.
86. Obwegeser HL: Surgical correction of maxillary deformities. In Grabb WC, Rosenstein SW, Bzoch KR, editors: *Cleft lip and palate*, Boston, 1971, Little, Brown, pp 515–556.
87. Obwegeser HL: Orthognathic surgery and a tale of how three procedures came to be: A letter to the next generations of surgeons. *Clin Plast Surg* 34:331–355, 2007.
88. Obwegeser HL, Lello GE, Farmand M: Correction of secondary cleft deformities. In Bell WH, editor: *Surgical correction of dentofacial deformities.* New Concepts, Vol III, Philadelphia, 1985, Saunders, 13, pp 592–638.
89. Oosterkamp BCM, Dijkstra PU, Remmelink HJ, et al: Orthodontic space closure versus prosthetic replacement of missing upper lateral incisors in patients with bilateral cleft lip and palate. *Cleft Palate Craniofac J* 47:591–596, 2010.
90. Padwa BL, Sonis A, Bagheri S, Mulliken JB: Children with repaired bilateral cleft lip and palate: Effect of age at premaxillary osteotomy on facial growth. *Plast Reconstr Surg* 104:1261–1269, 1999.
91. Parel SM, Branemark PI, Jansson T: Osseointegration in maxillofacial prosthetics: I. Intraoral applications. *J Prosthet Dent* 55:490, 1986.

92. Perko M: The history of treatment of cleft lip and palate. *Prog Pediatr Surg* 20:239–248, 1986.
93. Perrott D, Sharma AB, Vargevik K: Endosseous implants for pediatric patients. Unknown factors, indications, contraindications, and special considerations. *Oral Maxillofac Surg Clin North Am* 6:79, 1994.
94. Phillips JH, Klaiman P, Delorey R, MacDonald DB: Predictors of velopharyngeal insufficiency in cleft palate orthognathic surgery. *Plast Reconstr Surg* 115:681–686, 2005.
95. Polley JW, Figueroa AA: Management of severe maxillary deficiency in childhood and adolescence through distraction osteogenesis with an external, adjustable, rigid distraction device. *J Craniofac Surg* 8:181, 1997.
96. Polley JW, Figueroa AA: Rigid external distraction: Its application in cleft maxillary deformities. *Plast Reconstr Surg* 102:1360, 1998.
97. Poole MD, Robinson PP, Nunn ME: Maxillary advancement in cleft lip and palate patients: A modification of the Le Fort I osteotomy and preliminary results. *J Maxillofac Surg* 14:123, 1986.
98. Posnick JC: (Discussion of) Orthognathic surgery in cleft patients treated by early bone grafting. *Plast Reconstr Surg* 87:840, 1991.
99. Posnick JC: Orthognathic surgery in the cleft patient. In Russel RC, editor: *Instructional Courses, Plastic Surgery Education Foundation*, St Louis, 1991, CV Mosby Co, 4, pp 129–157.
100. Posnick JC: Orthognathic surgery for the cleft lip and palate patient. *Semin Orthod* 2(3):205–214, 1996.
101. Posnick JC: The treatment of secondary and residual dentofacial deformities in the cleft patient: Surgical and orthodontic therapy. *Clin Plast Surg* 24(3):583–597, 1997.
102. Posnick JC: Cleft lip and palate: Bone grafting and management of residual oro-nasal fistula. In Posnick JC, editor: *Craniofacial and maxillofacial surgery in children and young adults*, Philadelphia, 2000, WB Saunders Co, 33, pp 827–859.
103. Posnick JC: Cleft-orthognathic surgery: The bilateral cleft lip and palate deformity. In Posnick JC, editor: *Craniofacial and maxillofacial surgery in children and young adults*, Philadelphia, 2000, WB Saunders Co, 35, pp 908–950.
104. Posnick JC: The staging of cleft lip and palate reconstruction: Infancy through adolescence. In Posnick JC, editor: *Craniofacial and Maxillofacial Surgery in Children and Young Adults*, Philadelphia, 2000, WB Saunders Co, 32, pp 785–826.
105. Posnick JC, Agnihotri N: Managing chronic nasal airway obstruction at the time of orthognathic surgery: A twofer. *J Oral Maxillofac Surg* 69:695–701, 2011.
106. Posnick JC, Al-Qattan MM, Pron G: Facial sensibility in cleft and non-cleft adolescents one year after undergoing Le Fort I osteotomy. *Plast Reconstr Surg* 194(3):431–435, 1994.
107. Posnick JC, Dagys AP: Orthognathic surgery in the bilateral cleft lip patient: An integrated surgical and orthodontic approach. *Oral Maxillofac Surg Clin North Am* 3:693, 1991.
108. Posnick JC, Dagys AP: Orthognathic surgery in the bilateral cleft patient: An integrated surgical and orthodontic approach. *Oral Maxillofac Surg Clin North Am* 3(6):693–710, 1992.
109. Posnick JC, Getz SB: Surgical closure of end-stage palatal fistulas using anteriorly-based dorsal tongue flaps. *J Oral Maxillofac Surg* 45(11):907–912, 1987.
110. Posnick JC, Ricalde P: Cleft-orthognathic surgery. *Clin Plast Surg* 31(2):315–330, 2004.
111. Posnick JC, Ruiz R: (Discussion of) Repair of large anterior palatal fistulas using thin tongue flaps. *Ann Plast Surg* 45:114–117, 2000.
112. Posnick JC, Tompson B: Modification of the maxillary Le Fort I osteotomy in cleft-orthognathic surgery: The bilateral cleft lip and palate deformity. *J Oral Maxillofac Surg* 51(1):2–11, 1993.
113. Posnick JC, Tompson B: Cleft-orthognathic surgery: Complications and long-term results. *Plast Reconstr Surg* 96(2):255–266, 1995.
114. Pruzansky FH: Long-term effects of premaxillary setback on facial skeletal profile in complete bilateral cleft lip and palate. *Cleft Palate J* 22:97, 1985.
115. Robertsson S, Mohlin B: The congenitally missing upper lateral incisor: A retrospective study of orthodontic space closure versus restorative treatment. *Eur J Orthod* 22:697–710, 2000.
116. Ronchi P, Chiapasco M, Frattini D: Endosseous implants for prosthetic rehabilitation in bone grafted alveolar clefts. *J Craniomaxillofac Surg* 23:382, 1995.
117. Rosenstein SW: Facial growth and the need for orthognathic surgery after cleft palate repair: Literature review and report of 28 cases [discussion]. *J Oral Maxillofac Surg* 55:698, 1997.
118. Ross BR: Treatment variables affecting facial growth in complete unilateral cleft lip and palate. Part 7: An overview of treatment and facial growth. *Cleft Palate J* 24:71–77, 1987.
119. Samman N, Cheung LK, Tideman H: A comparison of alveolar bone grafting with and without simultaneous maxillary osteotomies in cleft palate patients. *Int J Oral Maxillofac Surg* 23:65–70, 1994.
120. Saperstein EL, Kennedy DL, Mulliken JB, Padwa BL: Facial growth in children with complete cleft of the primary palate and intact secondary palate. *J Oral Maxillofac Surg* 70:e66–e71, 2012.
121. Shaw WC, Asher-McDade C, Brattstrom V, et al: A six-center international study of treatment outcomes in patients with clefts of the lip and palate. *Cleft Palate Craniofac J* 29:393, 1992.
122. Sinn DP: Simultaneous maxillary expansion and advancement repair of oronasal fistula, and bone grafting of the alveolar cleft. In Bell WH, Proffit WR, White RP, editors: *Surgical correction of dentofacial deformities*, Philadelphia, 1980, WB Saunders.
123. Steinkamm W: *Die pseudo-progenie und ihre behandlung*, 1938, University of Berlin, inaugural dissertation.
124. Stoelinga PJ, Haers PE, Lennen RJ, et al: Late management of secondarily grafted clefts. *Int J Oral Maxillofac Surg* 19:91, 1990.
125. Stoelinga PJ, vd Vijver HR, Leenen RJ, et al: The prevention of relapse after maxillary osteotomies in cleft palate patients. *J Craniomaxillofac Surg* 15:325, 1987.
126. Suzuki A, Takahama Y: Maxillary lateral incisor of subjects with cleft lip and/or palate: Part 1. *Cleft Palate Craniofac J* 29:376, 1992.
127. Suzuki A, Takahama Y: Maxillary lateral incisor of subjects with cleft lip and/or palate: Part 2. *Cleft Palate Craniofac J* 29:380, 1992.
128. Takahashi T, Fukuda M, Yamaguchi T, et al: Use of an osseointegrated implant for dental rehabilitation after cleft repair by periosteoplasty: A case report. *Cleft Palate Craniofac J* 35:268, 1997.
129. Takahashi T, Fukuda M, Yamaguchi T, et al: Use of endosseous implants for dental reconstruction of patients with grafted alveolar clefts. *J Oral Maxillofac Surg* 55:576, 1997.
130. Tideman H, Stoelinga P, Gallia L: Le Fort I advancement with segmental palatal osteotomies in patients with cleft palates. *J Oral Surg* 38:196, 1980.
131. Trindale IE, Yamashita RP, Suguimoto RM, et al: Effects of orthognathic surgery on speech and breathing of subjects with cleft lip and palate: Acoustic and aerodynamic assessment. *Cleft Palate Craniofac J* 40:54–64, 2003.
132. Trotman CA, Ross RB: Craniofacial growth in bilateral cleft lip and palate: Ages six years to adulthood. *Cleft Palate Craniofac J* 30:261, 1993.
133. Turvey TA: Use of Branemark implant in the cleft palate patient [commentary]. *Cleft Palate Craniofac J* 28:304, 1991.
134. Verdi FJ Jr, Shanzi GL, Cohen SR, et al: Use of Branemark implant in the cleft palate patient. *Cleft Palate Craniofac J* 28:301, 1991.
135. Ward-Booth RP, Bhatia SN, Moos KF: A cephalometric analysis of the Le Fort II osteotomy in the adult cleft patient. *J Maxillofac Surg* 12:208, 1984.
136. Westbrook MT Jr, West RA, McNeil RW: Simultaneous maxillary advancement and closure of bilateral alveolar clefts and oronasal fistulas. *J Oral Maxillofac Surg* 41:257, 1983.

137. Williams AC, Bearn D, Mildinhall S, et al: Cleft lip and palate care in the United Kingdom—the Clinical Standards Advisory Group (CSAG) study. Part 2: Dentofacial outcomes and patient satisfaction. *Cleft Palate Craniofac J* 38:24–29, 2001.
138. Willmar K: On Le Fort I osteotomy: A follow-up study of 106 operated patients with maxillo-facial deformity. *Scand J Plast Reconstr Surg* 12(Suppl 1):1–68, 1974.
139. Wiltfang J, Hirschfelder U, Neukam FW, et al: Long-term results of distraction osteogenesis of the maxilla and midface. *Br J Oral Maxillofac Surg* 40:473, 2002.
140. Wolford LM: Effects of orthognathic surgery on nasal form and function in the cleft patient. *Cleft Palate Craniofac J* 29:546–555, 1992.
141. Wolford LM, Cassano DS, Cottrell DA, et al: Orthognathic surgery in the young cleft patient: Preliminary study on subsequent facial growth. *J Oral Maxillofac Surg* 66:2524–2536, 2008.
142. Wolford LM, Karras SC, Mehra P: Considerations for orthognathic surgery during growth: Part 1. Mandibular deformities. *Am J Orthod Dentofacial Orthop* 119:95, 2001.
143. Wolford LM, Karras SC, Mehra P: Considerations for orthognathic surgery during growth: Part 2. Maxillary deformities. *Am J Orthod Dentofacial Orthop* 119:102, 2001.
144. Wunderer S: Die Prognathieoperation mittels frontal gestieltem maxillafragment. *Osterreichische Z Stomatol* 59:98, 1962.
145. Yoshida H, Nakamura A, Michi K, et al: Cephalometric analysis of maxillofacial morphology in unoperated cleft palate patients. *Cleft Palate Craniofac J* 29:419–424, 1992.
146. Zachrisson BU, Stenvik A: Single implants—optimal therapy for missing lateral incisors? *Am J Orthod Dentofacial Orthop* 126:13A–15A, 2004.

34

Cleft–Orthognathic Surgery: The Isolated Cleft Palate Deformity

JEFFREY C. POSNICK, DMD, MD

- Facial Growth Implications of Cleft Palate Repair in the Infant with Isolated Cleft Palate
- Coordinated Team Approach
- Treatment Protocol
- Timing of Orthognathic Surgery
- Residual Deformities in the Adolescent with Isolated Cleft Palate
- Orthodontic Considerations in the Patient with Isolated Cleft Palate with a Jaw Deformity
- Immediate Presurgical Assessment
- Orthognathic Surgical Approach for Isolated Cleft Palate
- Clinical Management after Initial Surgical Healing
- Orthognathic Surgery for Isolated Cleft Palate: Review of Study
- Controversies and Unresolved Issues
- Conclusions

Isolated cleft palate (ICP) is a separate entity from cleft lip and palate. ICP has a different genetic pattern and associated anomalies, and it involves unique intraoral and facial anatomy and its own potential for head and neck dysfunction. It is estimated that approximately 30% of individuals born with cleft palate only (i.e., no cleft lip or alveolus) will have other associated malformations or be part of a known syndrome. Patients with ICP have a variety of jaw, dental, upper airway, and speech-related needs during childhood and into adolescence. The surgical repair of cleft palate during infancy with revision later in childhood, when it is needed to ensure adequate velopharyngeal competence, may result in disturbed growth of the upper jaw.

Facial Growth Implications of Cleft Palate Repair in the Infant with Isolated Cleft Palate

Ross documented that at least 20% of Caucasian individuals with ICP who undergo repair during infancy will experience maxillary hypoplasia that results in malocclusion that is not responsive to either traditional or compensatory orthodontic maneuvers alone.[86] Chen and colleagues reported on horizontal maxillary growth in both children and adults with Eastern Chinese ethnic backgrounds and with unoperated and operated ICP.[14] There were two study groups: individuals in Group 1 had non-syndromal and unoperated ICP that was evaluated during the mixed dentition (n = 16); individuals in Group 2 had non-syndromal and unoperated ICP that was evaluated during the permanent dentition (n = 25). The control groups included patients with ICP who underwent repair during childhood as well as non-cleft individuals with Class I occlusion that was evaluated during both the mixed and permanent dentitions. Lateral cephalograms of all subjects were analyzed. The results of the mixed dentition groups showed almost normal sagittal growth in unoperated patients except for the reduced anterior-posterior maxilla length. In contrast, the operated patients showed reduced length of both the maxilla and mandible, as well as a clockwise rotation of the mandible. The analysis of the permanent dentition groups showed that in both the unoperated and operated ICP patients, there were reductions in maxilla length, mandibular protrusion, and maxillary retrusion. Interestingly, there appeared to be no correlation between maxillary growth restriction and the extent of the congenital clefting of the secondary palate (i.e., soft palate only versus hard and soft palate). The authors concluded that, for individuals born with ICP, the etiology of the maxillomandibular deficiency likely results from a combination of factors, including the intrinsic primary (cleft) defect; secondary hypoplasia as a result of the surgical repair during infancy (iatrogenic); and functional (environmental) factors (e.g., the effects of muscles of mastication, respiratory patterns, mandibular

rest posture; see Chapter 4). These results were similar to those of Yoshida and colleagues, who found that individuals with ICP frequently end up with maxillary horizontal deficiency and clockwise rotation of the maxillomandibular complex that tend to become worse as the child reaches adulthood.[99]

A significant percentage of adolescent patients who were born with ICP will present with a jaw deformity that requires, at minimum, a maxillary Le Fort I (down-fracture) osteotomy to align the jaws.[5,85] When indicated, this is combined with mandibular and chin osteotomies to achieve improved facial proportions and with intranasal procedures (i.e., septoplasty and inferior turbinate reduction) to improve nasal breathing.

Coordinated Team Approach

The facial reconstructive and dental rehabilitation of an individual with ICP is best delivered via collaborative care provided by appropriate medical and dental specialists.[6,62,71] It is no longer justified for individual practitioners—whether they are surgeons, orthodontists, restorative dentists, speech pathologists, or otolaryngologists—to carry out extensive treatment without informing the patient and his or her family of available options and considering clinical input from the other specialists who are caring for the patient.

Treatment Protocol

Patients who are referred for possible orthognathic surgery are seen by an orthodontist, an orthognathic surgeon, a speech pathologist, and an otolaryngologist. Consultation with other medical (e.g., sleep specialist, geneticist) and dental (e.g., periodontist, restorative dentist) specialists may also be indicated. Initial records and tests include, at minimum, medical-quality photographs, including views of the face and the occlusion; cephalometric and Panorex radiographs; dental models with centric bite registration; speech assessment with instrumentation (ex. nasoendoscopy); and a thorough evaluation of the upper airway. Discussions with the medical and dental consultants, the family, and the patient are needed to set the clinical objectives. Decisions about the timing and extent of the orthodontic, surgical, and dental treatments are made in an effort to reach the chosen objectives.

The primary cleft surgeon completes the patient's palate repair during infancy, and this is followed by pharyngeal procedures later during the patient's childhood if they are required to improve velopharyngeal function. The surgeon is also likely to play an important role in directing the patient's care throughout adolescence and into adulthood. If the cleft surgeon is not trained in skeletal procedures, then a timely and seamless transition to the maxillofacial surgeon who will continue on with the patient's reconstruction is necessary. The orthodontist identifies early abnormal facial growth patterns and may carry out interceptive treatment.[6,10,11,18,19,27,34,51,55,64,65,87,90,91] Definitive orthodontic treatment is coordinated with orthognathic surgery, when indicated.[33] Extensive compensatory orthodontic treatment is likely to jeopardize periodontal health and to lead to dental relapse with recurrent malocclusion. Camouflage orthodontics should be avoided for these reasons and only be entered into with full disclosure to the family and other treating clinicians.

Before orthognathic surgery is performed, the speech pathologist performs an evaluation to assess the patient's velopharyngeal function; this will ideally include nasoendoscopic instrumentation. Such evaluation is important, because velopharyngeal function may deteriorate after maxillary Le Fort I osteotomy with advancement (see Chapter 8). Velopharyngeal closure that was adequate before surgery may become borderline afterward, and closure that was borderline may become inadequate. In the past, investigators have speculated that, if the distraction (DO) technique is used to advance the Le Fort I osteotomy, then velopharyngeal function will not deteriorate. However, clinical studies have now documented that velopharyngeal function will deteriorate in a similar fashion after Le Fort I advancement when either DO or standard techniques are used.[12,13,35,37,50,54,58,61,72,89,94] Articulatory distortions that result from the jaw discrepancy and malocclusion also are identified by the speech pathologist, and cause-and-effect relationships are determined. It is known that the successful orthodontic and surgical correction of crossbites, open bite, residual palatal fistulas, and negative overjet will generally correct the presenting articulation errors (see Chapter 8).

The otolaryngologist plays a role in the assessment of upper airway and middle ear function. Chronic nasal obstruction and sinusitis are frequent in the patient with a cleft palate. Mucous trapping may also be a problem after a pharyngeal flap procedure. If sleep apnea is suspected, an attended polysomnogram should be carried out (see Chapter 26). If intranasal procedures (i.e., septoplasty and inferior turbinate reduction) are needed to improve breathing, they should be simultaneously carried out in conjunction with orthognathic surgery (see Chapter 10).

Timing of Orthognathic Surgery

The correction of the jaw deformity associated with ICP is best carried out when the skeleton is mature and before the patient completes high school, if feasible. Maxillofacial growth is generally complete between the ages of 14 and 16 years in girls and between the ages of 16 and 18 years in boys. However, skeletal growth is variable, and it may be further clarified via the analysis of sequential cephalometric radiographs (see Chapter 17). Input from the patient and the family regarding the timing of surgery and the patient's functional needs (e.g., breathing, speech, chewing, swallowing, body image, self-esteem) are also important considerations.

As early as 1986, clinicians clarified through preliminary studies that, if jaw surgery is undertaken in the growing patient with a cleft palate patient, revision orthognathic

surgery will likely be required after skeletal maturity is reached.[95-98] More recently, other investigators have tested this theory by proceeding with Le Fort I osteotomy involving DO methods during the mixed dentition. Studies to date indicate that a Le Fort I osteotomy carried out during the mixed dentition, whether with a standard or a DO technique, results in limited or no further horizontal maxillary growth.[17,36,46,73,74] As the mandible continues to grow, an Angle Class III malocclusion recurs with the need for either additional Le Fort I advancement or mandibular set-back.

Residual Deformities in the Adolescent with Isolated Cleft Palate

The adolescent or adult patient with ICP who is referred for orthognathic evaluation will have an intact alveolar ridge and generally will have a full complement of teeth. However, these individuals may present with one or more of the following residual cleft-related problems (Figs. 34-1 through 34-9):

1. *Maxillary dysplasia.* When maxillary dysplasia occurs in the patient with ICP, it generally follows one of two patterns. The first and most frequently seen is horizontal maxillary retrusion, generally with a degree of vertical deficiency and often with clockwise rotation of the maxillary plane. The second pattern is vertical maxillary excess with a more limited degree of horizontal retrusion. The latter tends to occur in the presence of nasal obstruction with forced mouth breathing and an open-mouth posture, especially if a pharyngeal flap was placed during childhood (see Chapter 4).
2. *Residual oronasal (palatal) fistula.* There may be a residual midline palatal fistula located in the region between the incisive foramen and the soft palate. This is a residual defect that occurs after initial palate repair earlier in life.
3. *Residual bony defects.* The alveolus is not clefted, but residual bony defects of the hard palate are to be expected. The hard palate bony defects do not generally require grafting or reconstruction.
4. *Chin dysplasia.* The patient with ICP will frequently be a mouth breather with resulting open-mouth posture. The end result is often a vertically long and flat chin (i.e., a limited projection of the pogonion). If the Robin sequence was present at birth, a degree of microgenia will likely be a component of the deficiency (see Chapter 4).
5. *Mandibular dysplasia.* True mandibular prognathism in the patient with ICP is not common, but it may be seen more frequently among patients with certain ethnic backgrounds (e.g., Asian). A degree of mandibular retrognathism as a residual aspect of the Robin sequence is likely. This often occurs in combination with a degree of maxillary deficiency and clockwise rotation of the maxillomandibular complex. The occlusion may be satisfactory with orthodontic compensation; however, the maxillomandibular horizontal deficiency with clockwise rotation is aesthetically obvious, and it may also negatively affect the upper airway (see Ch. 26).
6. *Nasal obstruction and sinus blockage.* Obstructed breathing through the nose and intermittent sinusitis are frequent in the individual who is born with ICP. This may result from a combination of septal deviation and enlarged inferior turbinates. An in-place pharyngoplasty (e.g., a superiorly based flap, a sphincteroplasty) may also be a cause of partial nasopharyngeal obstruction. These findings will also predispose these patients to obstructive sleep apnea (see Chapter 26).
7. *Velopharyngeal dysfunction.* It has been documented that approximately 20% of individuals with a repaired cleft palate will demonstrate velopharyngeal insufficiency by the time that they are 5 years old. The adolescent with ICP who is arriving for the evaluation of a cleft jaw deformity may have a pharyngoplasty in place. The Le Fort I osteotomy with advancement is likely to improve the upper airway, but it may negatively affect velopharyngeal function (see Chapter 8).

Orthodontic Considerations in the Patient with Isolated Cleft Palate with a Jaw Deformity

A primary goal of presurgical orthodontic treatment in the teenager with an ICP jaw deformity is to eliminate all existing dental compensations. Instituting camouflage treatment is likely to jeopardize periodontal health, lead to dental relapse, and it may also cause root resorption (see Chapter 5).

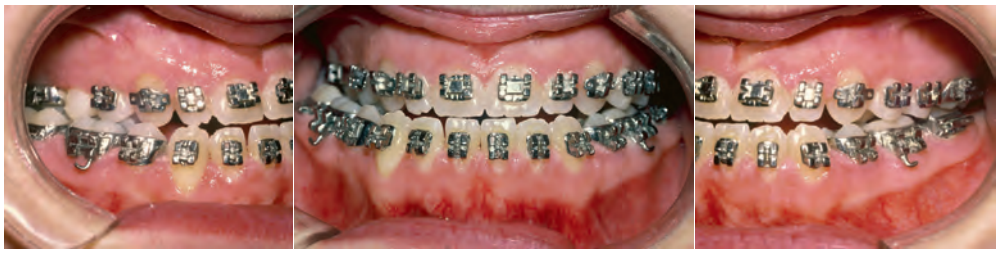
Correcting incisors inclination eliminating crowding, spacing, and rotations are all important orthodontic considerations. The arch form objectives are to achieve a satisfactory occlusion at operation that can be detailed afterward. To eliminate dental compensations, extractions may be required. When maxillary hypoplasia with negative overjet results in posterior crossbites as a result of "pseudo" constriction, it is important to not orthodontically overexpand the arch width. In the patient with ICP patient as compared with the patient with unilateral or bilateral cleft lip and palate, treatment is simplified because the alveolus is intact (i.e., there are no alveolar clefts) and there is generally a full complement of teeth.

Immediate Presurgical Assessment

When approaching the time of surgery, updated records are obtained, including alginate impressions of the maxillary

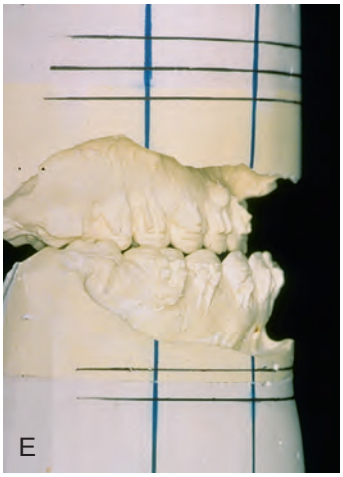
Text continued on p. 1465

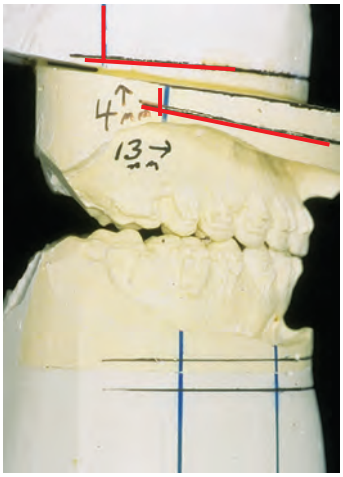
• **Figure 34-1** A 19-year-old woman who was born with isolated cleft palate. The maxillary deformity is characterized primarily by horizontal deficiency. The patient underwent mandibular first bicuspid extractions. She was referred to this surgeon and agreed to a comprehensive orthodontic and orthognathic surgical approach. The patient's procedures included Le Fort I osteotomy (horizontal advancement, vertical lengthening, and clockwise rotation) with interpositional grafting; osseous genioplasty (vertical reduction and horizontal advancement); and septoplasty and inferior turbinate reduction. **A,** Frontal views in repose before and after reconstruction. **B,** Frontal views with smile before and after reconstruction. **C,** Profile views before and after reconstruction.

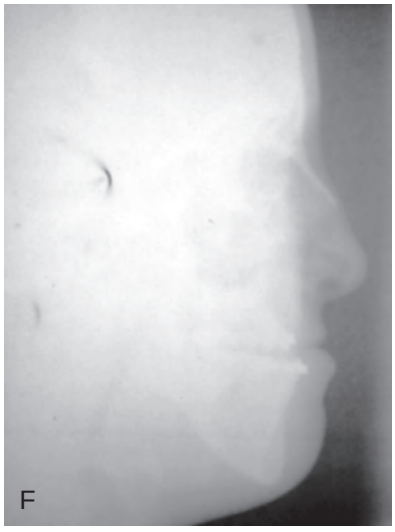


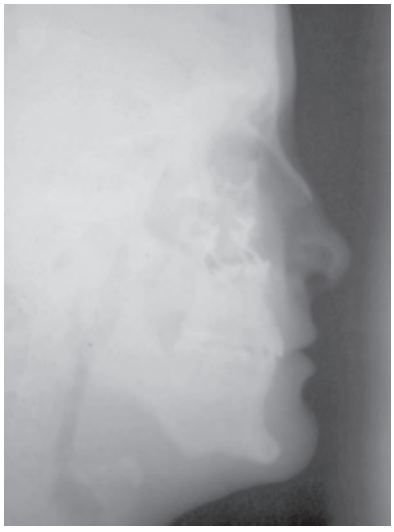
Ortho in progress after mandibular bicuspid extraction

After treatment







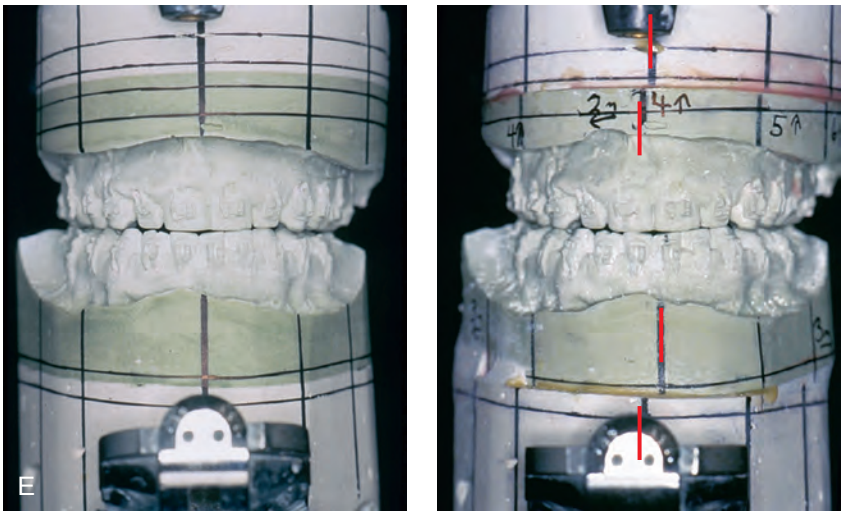


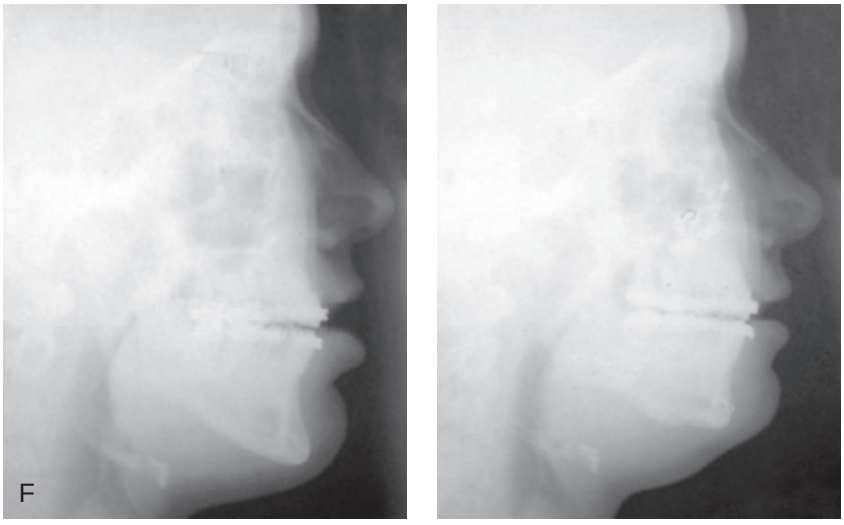
• **Figure 34-1, cont'd D,** Occlusal views during orthodontics and after reconstruction. **E,** Articulated dental casts indicate analytic model planning. **F,** Lateral cephalometric radiographs before and after reconstruction. *A, C, D (top center and bottom center), E (right), from Posnick JC: Skeletal stability and relapse patterns after Le Fort I osteotomy using miniplate fixation in patients with isolated cleft palate.* Plast Reconstr Surg *94:51, 1994.*

• **Figure 34-2** A 19-year-old woman who was born with isolated cleft palate. The deformity is characterized by vertical maxillary excess and jaw asymmetry. The patient underwent four-bicuspid extractions. She was referred to this surgeon and agreed to a comprehensive orthodontic and orthognathic approach. The patient's procedures included Le Fort I osteotomy (minimal horizontal advancement, clockwise rotation, vertical intrusion, and asymmetry correction); bilateral sagittal split ramus osteotomies (correction of asymmetry); osseous genioplasty (vertical reduction); and septoplasty, inferior turbinate reduction, and nasal recontouring. **A,** Frontal views in repose before and after reconstruction. **B,** Frontal views with smile before and after reconstruction. **C,** Profile views before and after reconstruction.

Prior to treatment after four-bicuspid extractions

After treatment



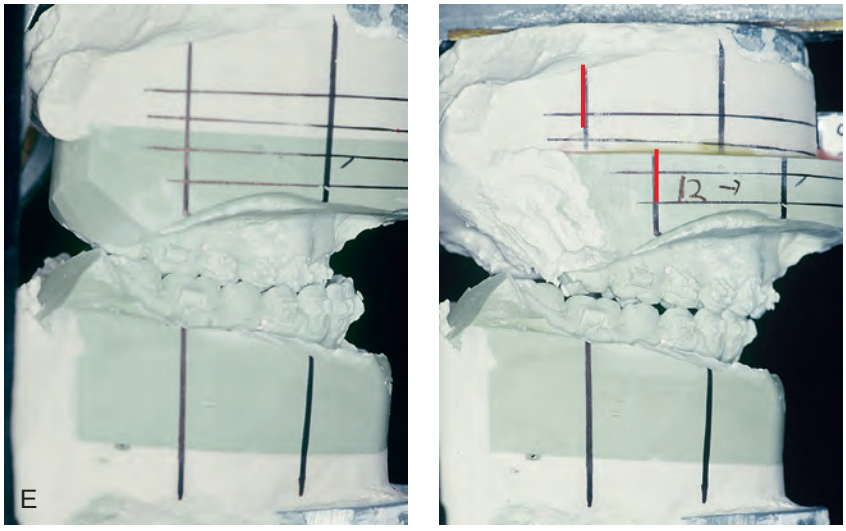


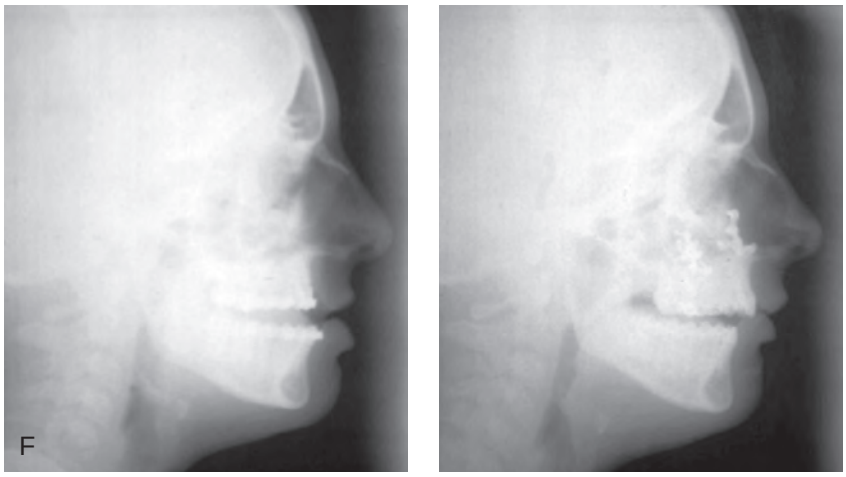
• **Figure 34-2, cont'd** **D,** Occlusal views with orthodontics in progress and after reconstruction. **E,** Articulated dental casts indicate analytic model planning. **F,** Lateral cephalometric radiographs before and after reconstruction.

• **Figure 34-3** A 17-year-old girl who was born with isolated cleft palate. The deformity is characterized primarily by horizontal maxillary deficiency and a palate fistula. The patient was referred to this surgeon and agreed to a comprehensive orthodontic and orthognathic approach. The maxillary lateral incisors were congenitally absent. The patient's procedures included a Le Fort I osteotomy (horizontal advancement and vertical lengthening) with interpositional grafting. One year after the initial procedure, she underwent an anterior-based dorsal tongue flap procedure for palatal fistula closure. **A,** Frontal views in repose before and after reconstruction. **B,** Frontal views with smile before and after reconstruction. **C,** Profile views before and after reconstruction.

Prior to surgery with absent maxillary lateral incisors

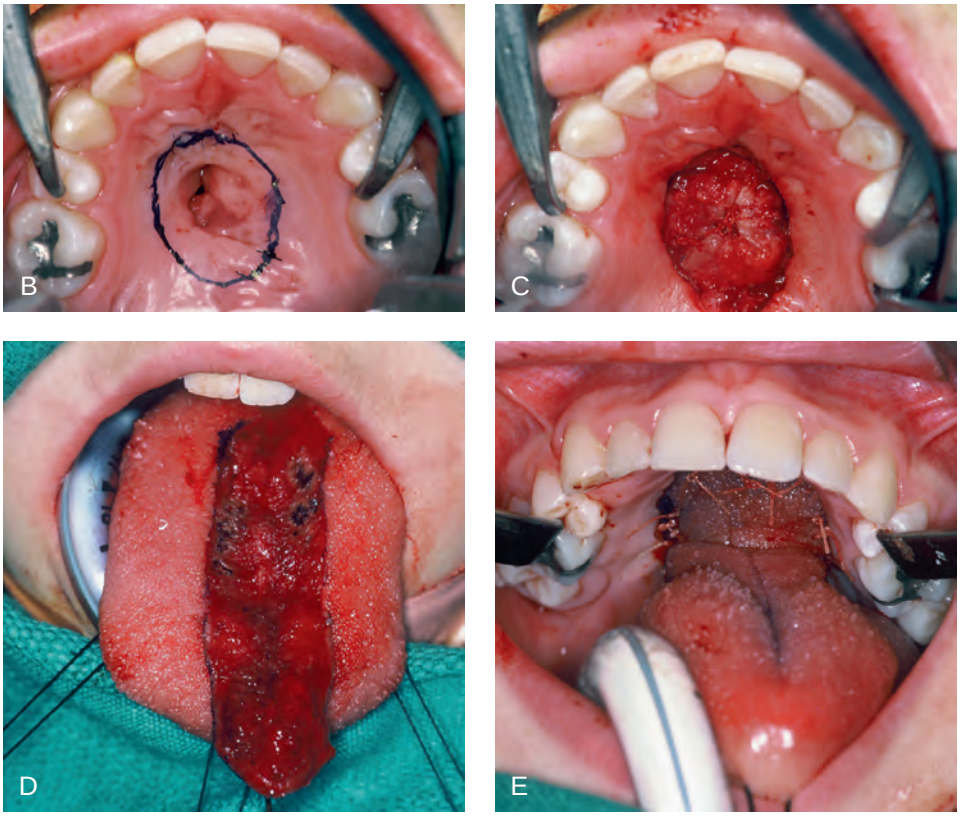
After treatment





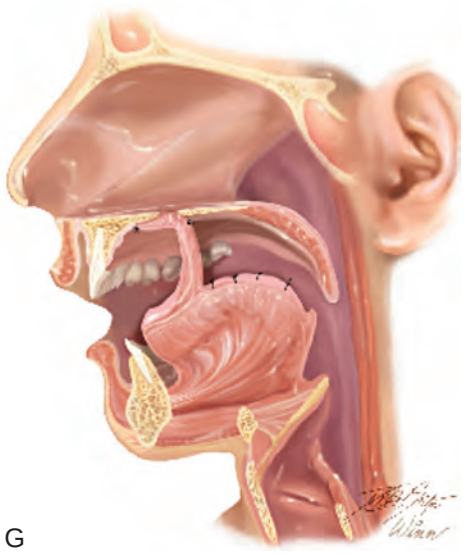
• **Figure 34-3, cont'd** **D,** Occlusal views with orthodontics in progress and after reconstruction. **E,** Articulated dental casts indicate analytic model planning. **F,** Lateral cephalometric radiographs before and after reconstruction. *A, C, D, F, from Posnick JC, Tompson B: Cleft–orthognathic surgery: complications and long-term results.* Plast Reconstr Surg *96:255, 1995.*

• **Figure 34-4** The patient from Figure 34-3 is shown after the successful placement of an anterior-based dorsal tongue flap for palatal fistula closure. **A,** Palatal views before and after the tongue-flap closure of a recalcitrant fistula. **B,** Facial views of the patient demonstrating a full range of tongue motion after the harvesting of tissue for palatal reconstruction.



• **Figure 34-5** A 17-year-old girl with a recalcitrant fistula at the incisal foramen region. The fistula had been present since the time of her initial cleft palate repair, despite several attempts at closure at another institution during the patient's childhood. She was referred to this surgeon for management. The fistula at the incisive foramen was successfully closed with an anteriorly based dorsal tongue flap. **A,** Facial views before fistula closure. **B,** Intraoperative view of the palatal fistula, with ink used to mark the extent of the bony defect. **C,** Local palatal flaps were developed and turned over for nasal-side closure. **D,** The elevated anteriorly based dorsal tongue flap is shown. It measured two thirds of the width of the tongue and was approximately 5 cm in length. **E,** The flap is inset to cover the oral side of the palatal defect. The tongue flap is then sutured to the adjacent palatal tissue three quarters of the way around. The posterior aspect of the flap is not sutured, because this would compromise flap circulation. The orotracheal tube can be seen on the side of the tongue.

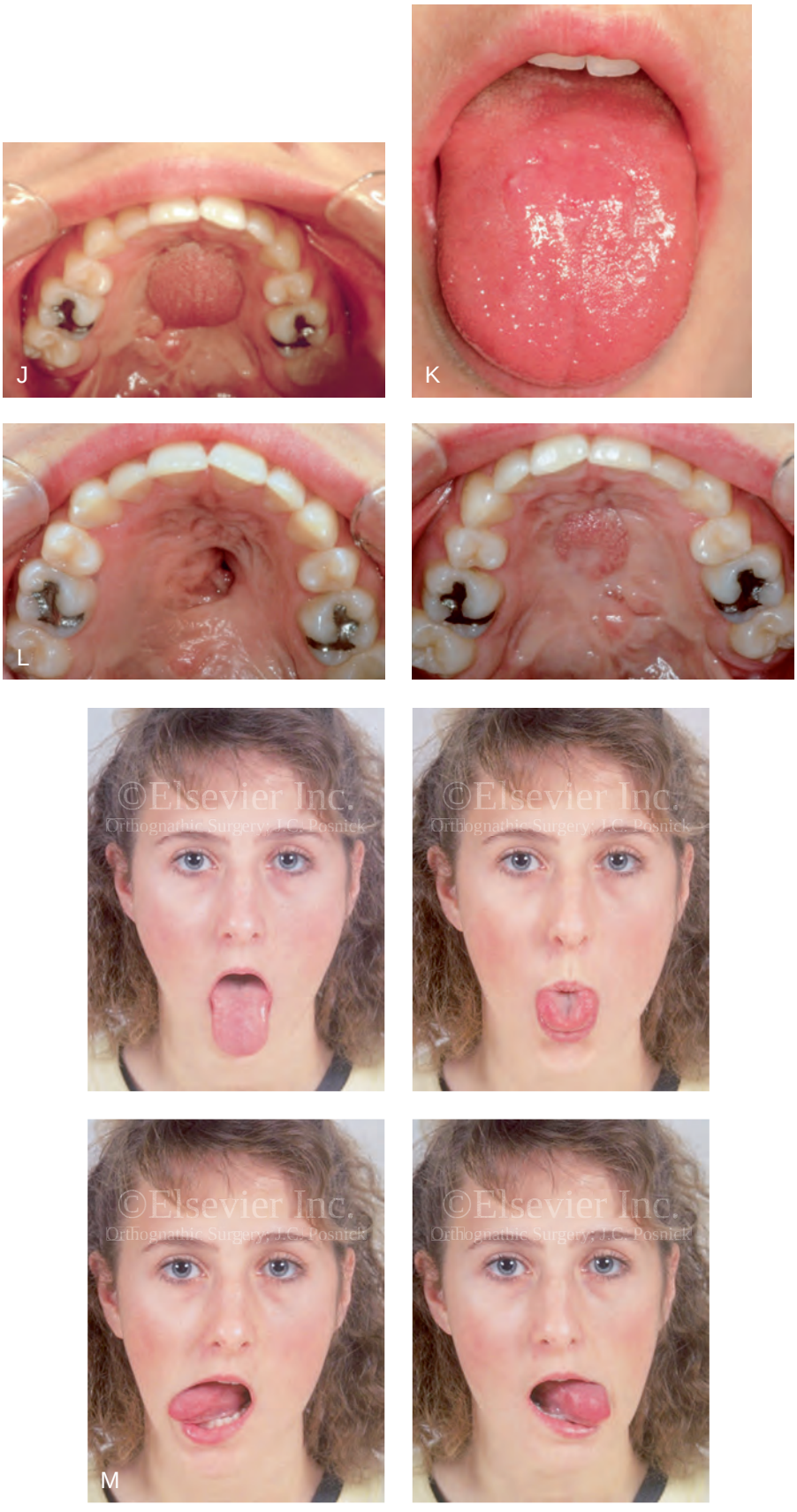
Continued


G

H

I

• **Figure 34-5, cont'd F,** Range of motion of the tongue is demonstrated at 10 days after flap inset and before flap release. **G,** Illustration of a cross-sectional sagittal view of face. The tongue flap is shown inset into the palate. Intra-operative view just 10 days after the procedure is also shown. She is returned to the operating room for flap release and inset. **H,** Intraoperative view of the dorsum of the tongue just after sectioning of the flap and before recontouring. **I,** Palatal view of bulky tongue tissue on the roof of the mouth before recontouring.



• **Figure 34-5, cont'd J,** Palatal view 3 months after flap inset and before debulking. **K,** Dorsum of the tongue 3 months after flap inset. **L,** Palatal views before and 8 months after the tongue flap was placed and debulking procedure completed. **M,** Demonstration of the range of tongue motion 3 months after flap release.

• **Figure 34-6** A 16-year-old girl who was born with isolated cleft palate. The deformity is characterized by horizontal and vertical deficiency of the maxilla and obstructed nasal breathing. The patient agreed to a comprehensive orthodontic and orthognathic surgical approach. Her maxillary first bicuspids were removed. The patient's procedures included Le Fort I osteotomy (horizontal advancement and vertical lengthening) with interpositional grafting; osseous genioplasty (vertical reduction and horizontal advancement); and septoplasty and inferior turbinate reduction. **A,** Frontal views in repose before and after reconstruction. **B,** Frontal views with smile before and after reconstruction.

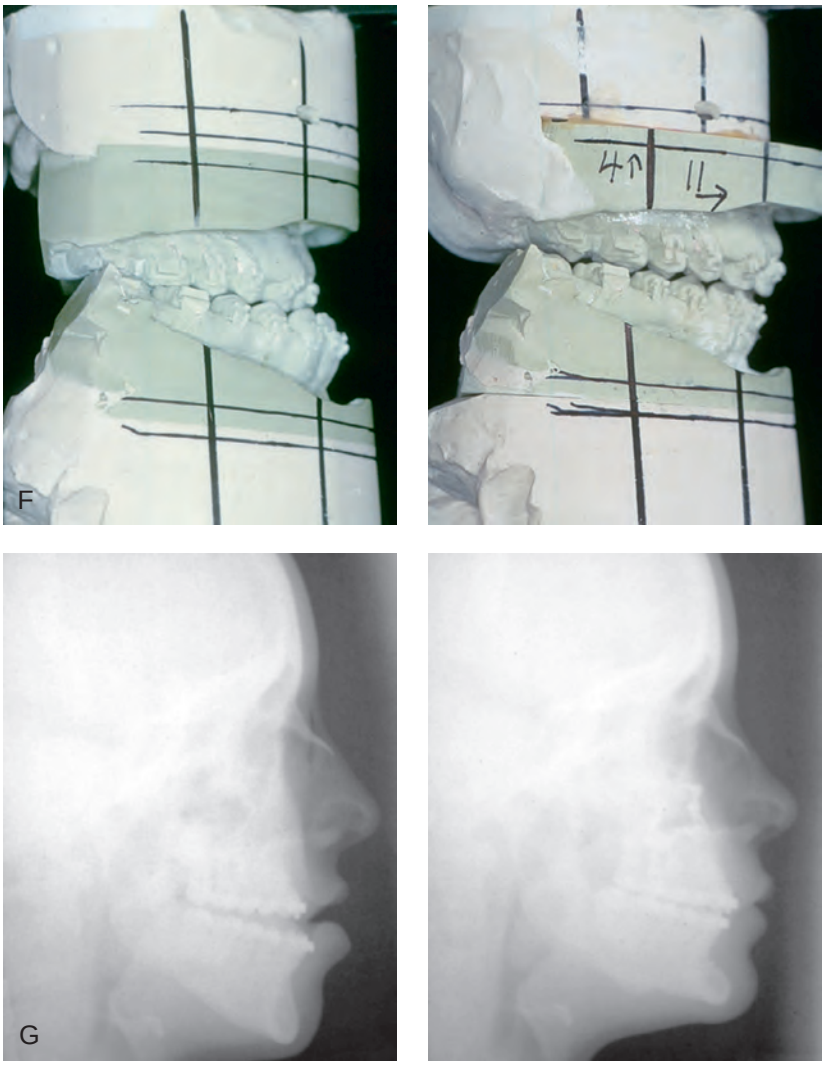
©Elsevier Inc.
Orthognathic Surgery; J.C. Posnick

• **Figure 34-6, cont'd** **C,** Oblique facial views before and after reconstruction. **D,** Profile views before and after reconstruction.

Continued

Prior to surgery after maxillary bicuspid extractions

After treatment



• **Figure 34-6, cont'd** **E,** Occlusal views with orthodontics in progress and after reconstruction. **F,** Articulated dental casts that indicate analytic model planning. **G,** Lateral cephalometric radiographs before and after reconstruction.

©Elsevier Inc.
Orthognathic Surgery; J.C. Posnick

• **Figure 34-7** A woman in her early 20s who was born with isolated cleft palate. The deformity is characterized primarily by the horizontal and vertical deficiency of the maxilla. She was referred to this surgeon and agreed to a comprehensive orthodontic and orthognathic surgical approach. She had a congenital absence of some of her teeth, and also required extractions. All total four bicuspids in the maxilla and two bicuspids in the mandible are missing. The patient's procedures included Le Fort I osteotomy (horizontal advancement and vertical lengthening) with interpositional grafting; osseous genioplasty (vertical reduction and horizontal advancement); and septoplasty and inferior turbinate reduction. **A,** Frontal views in repose before and after reconstruction. **B,** Frontal views with smile before and after reconstruction.

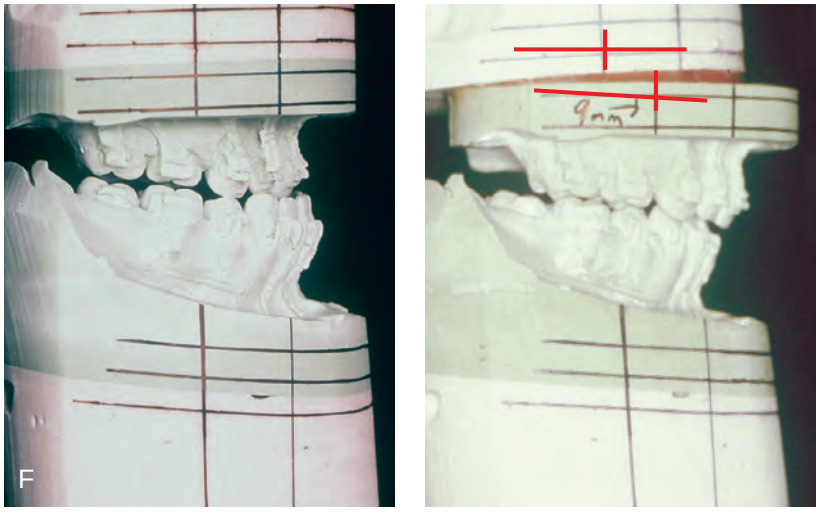
Continued

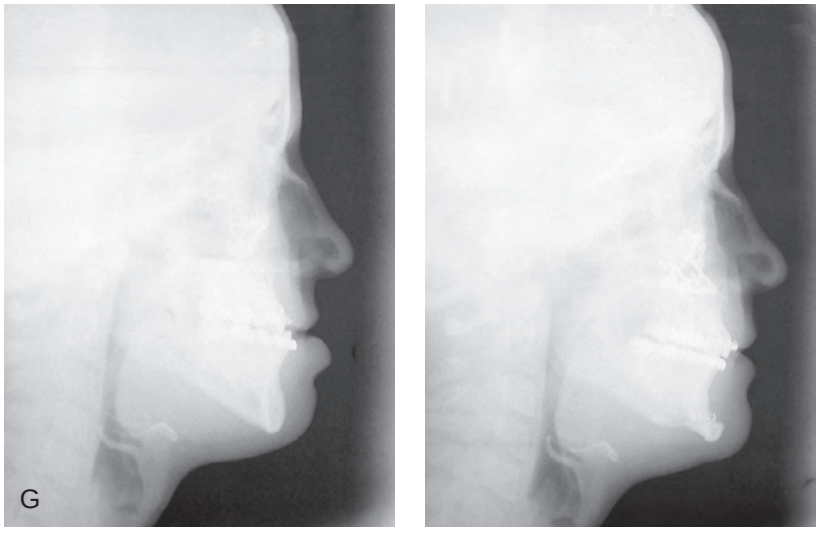
©Elsevier Inc.
Orthognathic Surgery, J.C. Posnick

• **Figure 34-7, cont'd C,** Profile views before and after reconstruction. **D,** Worm's-eye views before and after reconstruction.

Presurgery after maxillary four-bicuspid and mandibular two-bicuspid extraction / congenital absence

After treatment



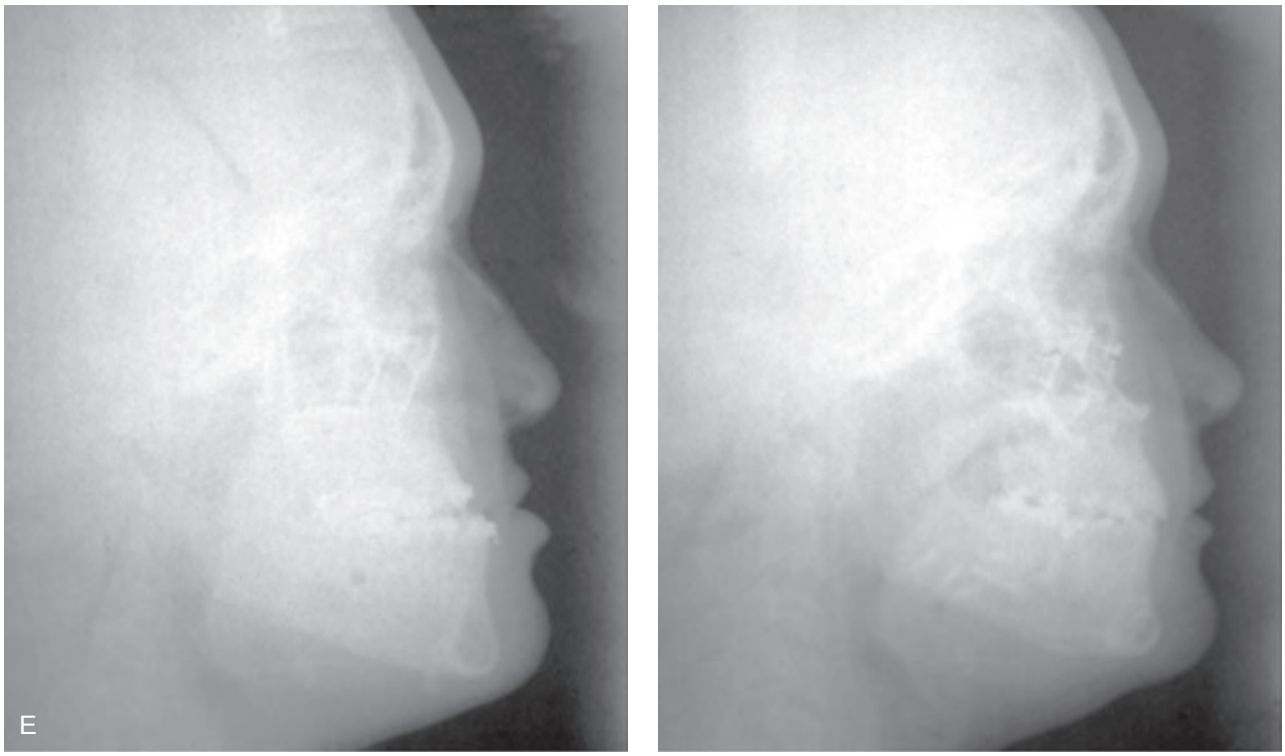


• **Figure 34-7, cont'd** **E,** Occlusal views with orthodontics in progress and after reconstruction. **F,** Articulated dental casts that indicate analytic model planning. **G,** Lateral cephalometric radiographs before and after reconstruction. *A, B, C (left), E (top middle, bottom middle), F, G, from Posnick JC, Ewing MP: The role of plate and screw fixation in the treatment of cleft lip and palate jaw deformities. In Yaremchuk MJ, Gruss JS, Manson PM, eds:* Rigid fixation of the craniomaxillofacial skeleton. *Stoneham, Mass, 1992, Butterworth, pp 466–485.*

• **Figure 34-8** A 16-year-old girl who was born with isolated cleft palate. The deformity is characterized primarily by the horizontal and vertical deficiency of the maxilla and chronic obstructed nasal breathing. The patient was referred to this surgeon and underwent a combined orthodontic and orthognathic surgical approach. Four bicuspids were removed. The patient's procedures included Le Fort I osteotomy (vertical lengthening and horizontal advancement) with interpositional grafting; sagittal split ramus osteotomy of the mandible (clockwise rotation); osseous genioplasty (vertical reduction and horizontal advancement); and septoplasty and inferior turbinate reduction. **A,** Frontal views with smile before and after reconstruction. **B,** Oblique views before and after reconstruction. **C,** Profile views before and after reconstruction.

Presurgery after four-bicuspid extractions

After treatment



• **Figure 34-8, cont'd D,** Occlusal views with orthodontics in progress and after reconstruction. **E,** Lateral cephalometric radiographs before and after reconstruction.

• **Figure 34-9** A woman in her late 20s who was born with isolated cleft palate. She developed a jaw deformity with malocclusion. Attempts to neutralize the occlusion included four bicuspid extractions with 6 years of orthodontic growth modification and orthodontic mechanics when the patient was between 11 and 17 years old. She was left with generalized labial bone loss and gingival recession, especially of the lower anterior teeth. She presented to this surgeon as an adult with a lifelong history of obstructed nasal breathing and a long face growth pattern that involved the maxilla, the mandible, and the chin; this included excess anterior facial height and horizontal retrusion. She was evaluated by clinicians in many specialties, including periodontics, prosthodontics, orthodontics, surgery, speech pathology, and otolaryngology. She underwent periodontal treatment and then orthodontic decompensation. The patient's procedures included Le Fort I osteotomy (horizontal advancement, vertical intrusion and clockwise rotation); bilateral sagittal split ramus osteotomies (horizontal advancement and counterclockwise rotation); osseous genioplasty (vertical reduction and horizontal advancement); and septoplasty, inferior turbinate reduction, and nasal floor recontouring. **A,** Frontal views in repose before and after reconstruction. **B,** Frontal views with smile before and after reconstruction.

• **Figure 34-9, cont'd** **C,** Oblique facial views before and after reconstruction. **D,** Profile views before and after reconstruction.

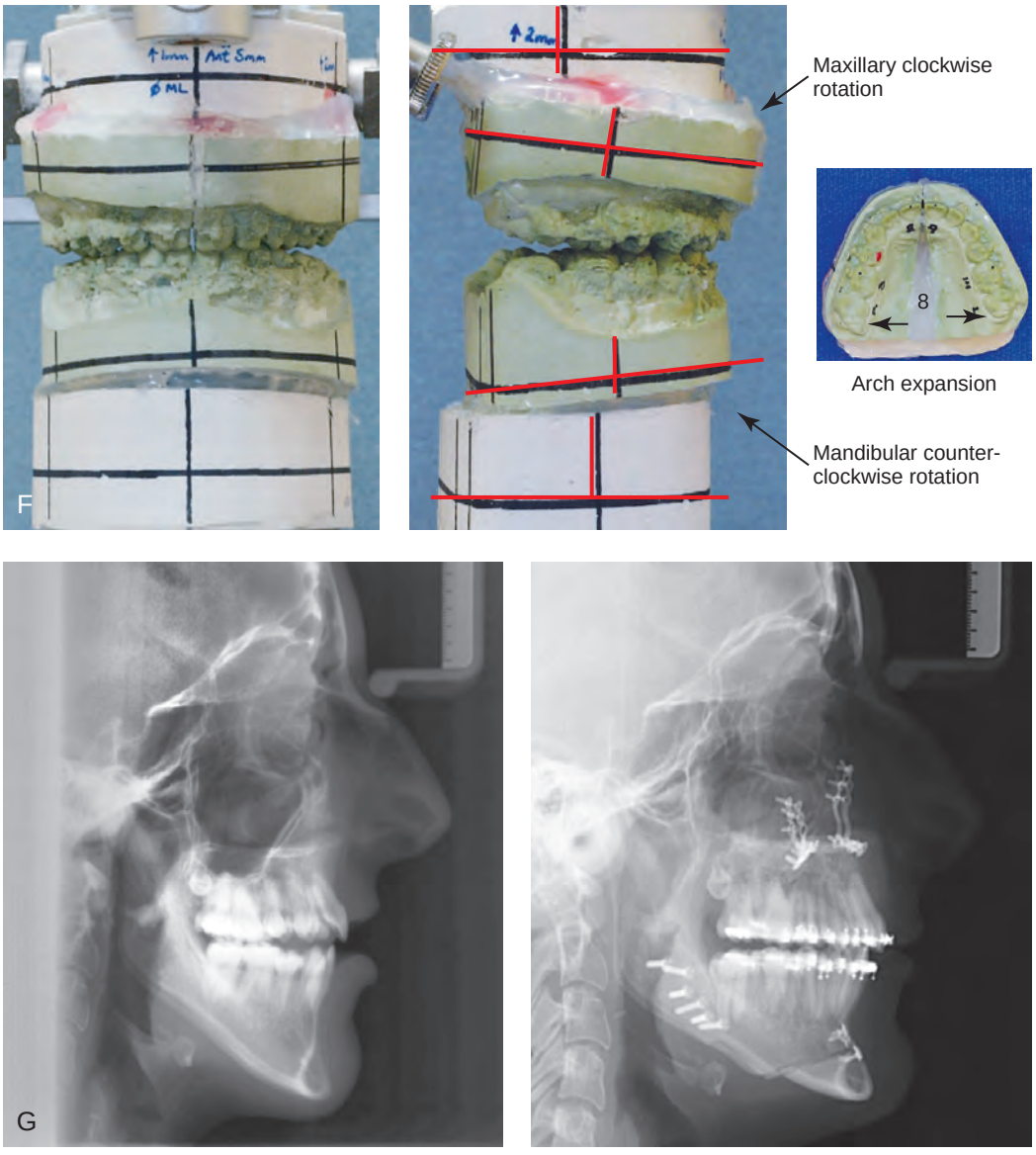
Continued

Prior to retreatment after four-bicuspid extractions

Pre surgery

After treatment

• **Figure 34-9, cont'd E,** Occlusal views before retreatment, after orthodontic decompensation, and after reconstruction.



• **Figure 34-9, cont'd** **F,** Articulated dental casts that indicate analytic model planning. **G,** Lateral cephalometric views before and after reconstruction.

and mandibular arches, a centric relation bite registration, a face-bow registration, and facial measurements. The patient's medical and dental records are reviewed, including radiographs; dental models; photographs of the face and the occlusion; and special studies. Decisions are made with regard to the preferred changes (i.e., repositioning) of the jaws and the precise millimeter distances and angles required in each jaw to achieve the desired result (see Chapter 12).[59,60] Analytic model planning is carried out on the articulated dental casts, and splints are constructed. The splints assist with the achievement of the precise occlusion and the preferred facial aesthetics that were determined preoperatively (see Chapter 13).

Orthognathic Surgical Approach for Isolated Cleft Palate

Evolution of Surgical Techniques

In general, the primary jaw deformity observed in the adolescent with ICP is maxillary hypoplasia or dysplasia as a result of the original cleft deformity and the subsequent surgical interventions.[31,32] The usual reconstructive procedure to consider is a Le Fort I maxillary osteotomy.[20,28,30,47,53,56,92] Obwegeser showed that a circumvestibular incision with the separation of the pterygoid maxillary sutures followed by down-fracture and the disimpaction of

the maxilla was not only possible but clinically relevant.[66-70] This allowed the maxilla to be moved in any direction, either as one unit or in segments. Obwegeser clarified that full mobilization of the down-fractured maxilla was essential to the achievement of an orthognathic correction on the operating room table and to the limiting of skeletal relapse over time. Bell and others validated the use of the Obwegeser Le Fort I technique to allow for adequate blood supply for routine satisfactory bone healing without aseptic necrosis or dental injury (see Chapter 2).[3,4,7,21-24]

Current Operative Technique

The surgical technique for management of the maxillary deformity involves a standard circumvestibular incision and Le Fort I osteotomy (see Chapter 15).[75-81,83,84] In the patient with ICP, the soft-tissue dissection may be difficult just before down-fracture as a result of the scarring of the nasal mucosa to the palatal tissue. There will be a need to sharply separate the oral and nasal layers completely during the down-fracture procedure. Note that this is not a contraindication to maxillary surgery but rather just a technical point. Care is taken to prevent the subperiosteal dissection (i.e., separation) of the palatal mucosa from the underlying hard palate, because this would compromise circulation to the down-fractured maxilla. Although it is of interest, the presence of a pharyngeal flap generally does not alter the surgical approach. This author has not found it necessary to section an in-place flap to accomplish the planned surgical objectives.

Residual palatal oronasal fistulas in the patient with ICP will be difficult to close simultaneously with the Le Fort I procedure; the elevation and transposition of palatal flaps are generally required to do so, and this would compromise the blood supply to the down-fractured maxilla. Interestingly, if a water-tight nasal side closure can be achieved through the down-fractures before the maxilla is fixed in its new location, then the residual palatal side mucosa separation will frequently heal by secondary intention. However, if a palatal fistula remains, it can be closed 6 months to 1 year after the orthognathic procedure, either with local palatal flaps or, if necessary, by using an anteriorly based dorsal tongue flap.[82]

The simultaneous management of secondary deformities frequently requires bilateral sagittal split osteotomies of the mandible and an oblique osteotomy of the chin. If removal of impacted wisdom teeth is recommended for long-term dental health this is accomplished at the same time. If septoplasty or inferior turbinate reduction or both are required to improve nasal airflow, then the procedures are also carried out simultaneously (see Chapter 10).

Clinical Management after Initial Surgical Healing

After surgery, cephalometric and Panorex radiographs and facial photographs are obtained at standard intervals. The orthodontic maintenance of the surgical result and the detailing of the occlusion are generally resumed after initial healing (i.e., 5 weeks after surgery). A seamless transition from the surgeon to the orthodontist for ongoing care is essential. Speech and velopharyngeal function maybe reassessed 3 months after surgery. If a pharyngeal flap, a revision pharyngoplasty, or final palatal fistula closure is required, this can be carried out 6 months after the orthognathic procedure.

Orthognathic Surgery for Isolated Cleft Palate: Review of Study

In a previously published study, we assessed initial and long-term skeletal stability after Le Fort I osteotomy in 14 skeletally mature patients with ICP who underwent palate repair during infancy and who then presented as adolescents with maxillary dysplasia and malocclusion.[83]

Patient and Methods

In a study by Posnick and colleagues, the medical records, longitudinal cephalometric radiographs, and current clinical examinations of all patients with ICP who underwent Le Fort I osteotomy over a 3-year period by a single surgeon (Posnick) were reviewed.[83] The following information was noted: all previous cleft and maxillofacial surgical procedures; stabilization techniques; the presence of a pharyngoplasty; the use of a bone graft; perioperative orthodontics; age at surgery; perioperative morbidity; and, at final follow up, the amount of overjet and overbite of the incisors. All patients were skeletally mature at the time of the operation. In all patients, lateral cephalograms were taken before surgery and at 3 to 7 days, 6 to 8 weeks, and 1 year postoperatively.

On each preoperative radiograph, the facial plane was constructed for each patient. At the level of the palatal plane, a perpendicular plane was constructed to form a Cartesian coordinate system that was referenced for skeletal measurements. The serial radiographs taken after surgery were superimposed on the anterior cranial base structures with the Cartesian coordinate axes as planes of reference. Vertical and horizontal directional changes were then measured at each postoperative interval. With the radiographs superimposed, the amount of horizontal advancement was measured by the method of anatomic best fit. Vertical change over time was analyzed anteriorly at the incisor area and posteriorly at the first molar area. Actual measurements could be determined reliably within 0.5 mm. The 1-year postoperative lateral cephalograms also were assessed for the measurement of incisor overjet and overbite.

Differences between groups were assessed by Student's *t*-test. Associations between the magnitude of surgical advancement and the degree of relapse were examined by the Pearson correlation coefficient (r) and by least-squares linear regression analysis. Correlations were considered significant when the P value was less than .05.

All patients underwent a standard one-piece Le Fort I osteotomy extended through the zygomatic buttress (i.e., below the malar eminence) and anteriorly into the pyriform apertures. After titanium mini-plates were used to rigidly fix the osteotomy in place, crafted corticocancellous iliac bone grafts were wedged between the zygomatic buttress and the pyriform aperture on each side and secured with an additional micro-plate and screws. Simultaneous bilateral sagittal split ramus osteotomies were used in 4 of 14 patients to manage secondary deformities. All subjects underwent a simultaneous osseous genioplasty. In all patients, the prefabricated splint secured to the maxillary orthodontic brackets confirmation that the occlusion remained unchanged for 5 weeks.

The age at operation ranged from 17 to 25 years (mean, 19 years). During the study period, the ICP patients (N = 14) represented just 12% of the surgeon's patients with cleft lips and palates who underwent orthognathic surgery. All study patients had a complete set of longitudinal records (clinical and cephalometric) and were available for late postoperative clinical reassessment. Clinical follow up ranged from 1.5 to 5.5 years (mean, 2.5 years) after surgery at the close of the study.

Results

Perioperative morbidity was unremarkable when patients were reviewed for cardiopulmonary compromise, maxillofacial infections, hemorrhage, aseptic necrosis, loss of teeth, or the need for root canal therapy. All patients maintained a positive overjet and overbite at the incisor level, which was documented on both the 1-year clinical and cephalometric examinations. No significant difference was seen in vertical or horizontal surgical change or in postoperative relapse between those who had Le Fort I osteotomy only (n = 10) and those who underwent simultaneous Le Fort I and bilateral sagittal split ramus osteotomies of the mandible (n = 4) or between those who underwent iliac bone grafts (n = 10) and those who did not (n = 4). With reference to these variables, the patients were therefore considered a homogeneous group for the purposes of further analysis.

As measured directly from the serial cephalometric radiographs for each patient, the mean horizontal advancement achieved was 6.4 mm, and 5.4 mm of advancement was maintained at 1 year. Three of the 14 patients showed no more than 0.5 mm of relapse, and none had more than 1.5 mm of relapse. In the eight patients without a pharyngoplasty in place at the time of Le Fort I osteotomy, the horizontal advancement was 6.1 mm immediately after the operation and 5.1 mm 1 year later. The remaining six patients achieved a similar horizontal advancement both immediately after the operation (6.9 mm) and 1 year later (5.9 mm).

The vertical change that was achieved initially and then maintained over the long term was analyzed in both the anterior and posterior regions of the upper jaw to assess regional differences in skeletal ability patterns more accurately. The mean anterior vertical change of the maxilla was 2.0 mm immediately after the operation and 1.4 mm after 1 year. The mean posterior vertical change of the maxilla was 2.7 mm immediately after the operation and 1.9 mm after 1 year (mean relapse, 0.8 mm).

Controversies and Unresolved Issues

Skeletal Stability after Le Fort I Osteotomy

The study by Posnick and Taylor assessed the skeletal stability and relapse patterns in mature adolescents and young adults with ICP who underwent palate repair during infancy and who later developed maxillary dysplasia that required orthognathic surgery for the improvement of facial balance, the airway and the occlusion.[83] Interestingly, the skeletal stability in the patients with ICP after Le Fort I osteotomy was somewhat better than that documented in patients with unilateral cleft lip and palate. Posnick and Taylor's study confirm that an in-place pharyngoplasty does not contribute to relapse or increase perioperative morbidity. Roughly half the measured horizontal and vertical relapse occurred during the first 6 weeks after the operation, and the remainder occurred during the rest of the first year. A long-term positive overjet and overbite was maintained in all patients.

The Standard Approach versus the Distraction Approach to Le Fort I Advancement

The use of a DO technique to reposition the maxilla gradually over several weeks into the preferred horizontal position and to then hold the maxilla in place with the DO appliance for several months during the consolidation phase is relatively inconvenient and costly to the patient, the family, and the health care system. It remains a distant second choice as compared with the standard approach when the surgeon is able to down-fracture the maxilla, adequately mobilize it, and then place it into the preferred position in the operating room. The standard approach originally described by Obwegeser during the 1960s and then continually refined offers a safe and effective way to manage most jaw deformities that occur in the patient with ICP.[66-70] Despite two decades of use, the DO technique for maxillary advancement has not been shown to offer less morbidity or improved stability for the patient with a cleft jaw deformity.* It does not allow for the correction of the deformity during childhood, as many clinicians had initially hoped it would. No horizontal growth can be expected after a Le Fort I osteotomy is carried out, whether with a DO approach or a standard appro ach.[17,36,46,73,74,95-98] The DO approach has not diminished the occasional occurrence of velopharyngeal insufficiency after Le Fort I osteotomy, and it has not eliminated the problem of skeletal relapse.[12,13,35,37,50,54,58,61,72,89,94] Alternatively, the DO approach is useful in unusual circumstances, when the surgeon is not able to adequately mobilize and fully advance the maxilla as planned in the operating room. For patients

*References 1, 2, 8, 9, 15, 16, 25, 26, 29, 38, 39, 40-45, 48, 49, 52, 57, 63, 78, 83, 84, 88, 93

of special concern, the surgeon may wish to discuss the DO option before the Le Fort I advancement occurs. If the surgeon is unable to sufficiently mobilize and reposition the maxilla with the use of standard techniques, then a DO appliance can be placed (see Chapter 32).

Closure of Large Residual Palatal Fistula

When a recalcitrant large oronasal fistula at the incisal foramen region remains in a patient with ICP, palatal flaps alone may not be adequate for closure. In these cases, options are limited. This author often suggests achieving nasal side closure with the use of local full-thickness palatal (turnover) flaps and then obtaining oral side closure by elevating and placing an anteriorly based dorsal tongue flap. The tongue flap will provide needed vascularized soft tissue for effective fistula closure on the oral side.[82] This approach does require the sacrifice of a small portion of the tongue donor site, a second general anesthetic, and downtime for the patient during the initial convalescence period (see Figure 34-4 and 34-5).

Conclusions

Patients with ICP present varied dental, occlusal, upper airway, middle ear, speech-related, and aesthetic needs throughout their childhood, adolescent, and adult years. At least 20% of these patients who undergo palate repair during infancy will develop maxillary dysplasia that is not responsive to orthodontic treatment alone. An integrated comprehensive surgical–orthodontic approach is required to resolve these problems at the time of skeletal maturity. When indicated, a Le Fort I osteotomy in combination with mandibular, chin, and intranasal procedures represents the standard of care for the establishment of improved facial balance, occlusion and dental rehabilitation, and breathing.

References

1. Aksu M, Saglam-Aydinatay B, Akcan CA, et al: Skeletal and dental stability after maxillary distraction with a rigid external device in adult cleft lip and palate patients. *J Oral Maxillofac Surg* 68:254–259, 2010.
2. Araujo A, Schendel SA, Wolford IM, et al: Total maxillary advancement with and without bone grafting. *J Oral Surg* 36:849, 1978.
3. Bell WH, Fonseca RJ, Kennedy JW III, et al: Bone healing and revascularization after total maxillary osteotomy. *J Oral Surg* 33:253, 1975.
4. Bell WH: Le Fort I osteotomy for correction of maxillary deformities. *J Oral Surg* 33:412, 1975.
5. Berkowitz S: State of the art in cleft palate or facial growth and dentistry: A historical perspective. *Am J Orthod* 74:564–576, 1978.
6. Bishara SE: Cephalometric evaluation of facial growth in operated and nonoperated individuals with isolated clefts of the palate. *Cleft Palate J* 3:239–245, 1973.
7. Bloomquist DS: Intraoperative assessment of maxillary perfusion during Le Fort I osteotomy [discussion]. *J Oral Maxillofac Surg* 52:831, 1994.
8. Braun TW, Sotereanos GC: Orthognathic and secondary cleft reconstruction of adolescent patients with cleft palate. *J Oral Surg* 38:425, 1980.
9. Braun TW, Sotereanos GC: Orthognathic surgical reconstruction of cleft palate deformities in adolescents. *J Oral Surg* 39:255, 1981.
10. Canady JW, Thompson SA, Colburn A: Craniofacial growth after iatrogenic cleft palate repair in a fetal bovine model. *Cleft Palate Craniofac J* 34:69, 1997.
11. Capelozza Filho L, Normando AD, da Silva Filho OG: Isolated influences of lip and palate surgery on facial growth: Comparison of operated and unoperated male adults with UCL/P. *Cleft Palate Craniofac J* 33:51–56, 1996.
12. Chanchareonsook N, Samman N, Whitehill TL: The effect of cranio-maxillofacial osteotomies and distraction osteogenesis on speech and velopharyngeal status: A critical review. *Cleft Palate Craniofac J* 43:477–487, 2006.
13. Chanchareonsook N, Whitehill TL, Samman N: Speech outcome and velopharyngeal function in cleft palate: Comparison of Le Fort I maxillary osteotomy and distraction osteogenesis—early results. *Cleft Palate Craniofac J* 44:23–32, 2007.
14. Chen ZQ, Qian YF, Wang GM, Shen G: Sagittal maxillary growth in patients with unoperated isolated cleft palate. *Cleft Palate Craniofac J* 46:664–667, 2009.
15. Cheung LK, Chua HD: A meta-analysis of cleft maxillary osteotomy and distraction osteogenesis. *Int J Oral Maxillofac Surg* 35:14, 2006.
16. Cho BC, Kyung HM: Distraction osteogenesis of the hypoplastic midface using a rigid external distraction system: The results of a one- to six-year follow-up. *Plast Reconstr Surg* 118:1201, 2006.
17. Correa Normando AD, da Silva Filho OG, Capelozza Filho L: Influence of surgery on maxillary growth in cleft lip and/or palate patients. *J Craniomaxillofac Surg* 20:111, 1992.
18. David DJ, Anderson PJ, Schnitt DE, et al: From birth to maturity: A group of patients who have completed their protocol management. Part II. Isolated cleft palate. *Plast Reconstr Surg* 117:515–526, 2006.
19. DeLuke DM, Marchand A, Robles EC, Fox P: Facial growth and the need for orthognathic surgery after cleft palate repair: Literature review and report of 28 cases. *J Oral Maxillofac Surg* 55:694–698, 1997.
20. Des Prez JD, Kiehn CL: Surgical positioning of the maxilla. Symposium on management of cleft lip and palate and associated deformities. *Am Plast Reconstr Surg* 8:222, 1974.
21. Dodson TB, Neuenschwander MC, Bays RA: Intraoperative assessment of maxillary perfusion during Le Fort I osteotomy. *J Oral Maxillofac Surg* 52:827, 1994.
22. Dodson TB, Neuenschwander MC: Maxillary perfusion during Le Fort I osteotomy after ligation of the descending palatine artery. *J Oral Maxillofac Surg* 55:51, 1997.
23. Drommer R: The history of the "Le Fort I-Osteotomy." *J Maxillofac Surg* 14:119–122, 1986.
24. Drommer R, Luhr HG: The stabilization of osteotomized maxillary segments with Luhr miniplates in secondary cleft surgery. *J Maxillofac Surg* 9:166–169, 1981.
25. Figueroa AA, Polley JW, Friede H, et al: Long-term skeletal stability after maxillary advancement with distraction osteogenesis using a rigid external distraction device in cleft maxillary deformities. *Plast Reconstr Surg* 114:1382, 2004.
26. Figueroa AA, Polley JW, Ko EW: Maxillary distraction for the management of cleft maxillary hypoplasia with a rigid external distraction system. *Semin Orthod* 5:46, 1999.
27. Filho LC: Isolated influences of lip and palate surgery on facial growth: Comparison of operated and unoperated male adults. *Cleft Palate Craniofac J* 33:51, 1996.
28. Fitzpatrick B: Mid-face osteotomy in the adolescent cleft patient. *Aust Dent J* 22:338, 1977.

29. Freihofer HPM Jr: Results of osteotomies of the facial skeleton in adolescence. *J Maxillofac Surg* 5:267, 1977.
30. Georgiade NG: Mandibular osteotomy for the correction of facial disproportion in the cleft lip and palate patient. Symposium on management of cleft lip and palate and associated deformities. *Am Plast Reconstr Surg* 8:238, 1974.
31. Gillies HD, Millard DR Jr: *The principles and art of plastic surgery*, Boston, 1957, Little, Brown.
32. Gillies HD, Rowe NL: L'ostéotomie du maxillaire supérieur enoisagée essentiellement dans le cas de bec-de-lièvre total. *Rev Stomatol* 55:545–552, 1954.
33. Gnoinski W: Early identification of candidates for corrective maxillary osteotomy in cleft lip and palate group. *Scand J Plast Reconstr Surg Hand Surg* 21:39, 1987.
34. Good PM, Mulliken JB, Padwa BL: Frequency of Le Fort I osteotomy after repaired cleft lip and palate or cleft palate. *Cleft Palate Craniofac J* 44:396–401, 2007.
35. Guyette TW, Polley JW, Figueroa A, Smith BE: Changes in speech following maxillary distraction osteogenesis. *Cleft Palate Craniofac J* 38:199–205, 2001.
36. Harada K, Baba Y, Ohyama K, et al: Maxillary distraction osteogenesis for cleft lip and palate children using an external, adjustable, rigid distraction device: A report of 2 cases. *J Oral Maxillofac Surg* 59:1492, 2001.
37. Harada K, Ishii Y, Ishii M, et al: Effect of maxillary distraction osteogenesis on velopharyngeal function: A pilot study. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 93:538, 2002.
38. Hedemark A, Freihofer HP Jr: The behavior of the maxilla in vertical movements after Le Fort I osteotomy. *J Maxillofac Surg* 6:244, 1978.
39. He D, Genecov DG, Barcelo R: Nonunion of the external maxillary distraction in cleft lip and palate: Analysis of possible reasons. *J Oral Maxillofac Surg* 68:2402–2411, 2010.
40. Hierl T, Hemprich A: Callus distraction of the midface in the severely atrophied maxilla: A case report. *Cleft Palate Craniofac J* 36:457, 1999.
41. Hirano A, Suzuki H: Factors related to relapse after Le Fort I maxillary advancement osteotomy in patients with cleft lip and palate. *Cleft Palate Craniofac J* 38:1, 2001.
42. Hochban W, Ganss C, Austermann KH: Long-term results after maxillary advancement in patients with clefts. *Cleft Palate Craniofac J* 30:237–243, 1993.
43. Höltje WJ, Scheuer H: Skeletal stability and relapse patterns after Le Fort I osteotomy using miniplate fixation in patients with isolated cleft palate [discussion]. *Plast Reconstr Surg* 94:59–60, 1994.
44. Horster W: Experience with functionally stable plate osteosynthesis after forward displacement of the upper jaw. *J Maxillofac Surg* 8:176, 1980.
45. Houston WJB, James DR, Jones E, Kavvadia S: Le Fort I maxillary osteotomies in cleft palate cases: Surgical changes and stability. *J Craniomaxillofac Surg* 17:9–15, 1989.
46. Huang CS, Harikrishnan P, Liao YF, et al: Long-term follow-up after maxillary distraction osteogenesis in growing children with cleft lip and palate. *Cleft Palate Craniofac J* 44:274, 2007.
47. Hui E, Hägg EU, Tideman H: Soft tissue changes following maxillary osteotomies in cleft lip and palate and non-cleft patients. *J Craniomaxillofac Surg* 22:182–186, 1994.
48. Jackson IT: Cleft and jaw deformities. *Symposium on Reconstruction of Jaw Deformities* 113, 1978.
49. James D, Brook K: Maxillary hypoplasia in patients with cleft lip and palate deformity: The alternative surgical approach. *Eur J Orthod* 7:231, 1985.
50. Janulewicz J, Costello BJ, Buckley MJ, et al: The effects of Le Fort I osteotomies on velopharyngeal and speech functions in cleft patients. *J Oral Maxillofac Surg* 62:308–314, 2004.
51. Jorgenson RJ, Shapiro SD, Odiner KL: Studies on facial growth and arch size in cleft lip and palate. *J Craniofac Genet Dev Biol* 4:33, 1984.
52. Kanno T, Mitsugi M, Hosoe M, et al: Long-term skeletal stability after maxillary advancement with distraction osteogenesis in nongrowing patients. *J Oral Maxillofac Surg* 66:1833–1846, 2008.
53. Kiehn CL, DesPrez JD, Brown F: Maxillary osteotomy for late correction of occlusion and appearance in cleft lip and palate patients. *Plast Reconstr Surg* 42:203, 1968.
54. Ko EW, Figueroa AA, Guyette TW, et al: Velopharyngeal changes after maxillary advancement in cleft patients with distraction osteogenesis using a rigid external distraction device: 1-year cephalometric follow-up. *J Craniofac Surg* 10:312–320, 1999.
55. Lu D-W, Shi B, Wang H-J, Zheng Q: The comparative study of craniofacial structural characteristic of individuals with different types of cleft palate. *Ann Plast Surg* 59:382–387, 2007.
56. Luhr HG: Zur stabilen osteosynthese bei unterkiefer-frakturen. *Dtsch Zahnarztl Z* 23:754, 1968.
57. Luyk NH, Ward-Booth RP: The stability of the Le Fort I advancement osteotomies using bone plates without bone grafts. *J Maxillofac Surg* 13:250, 1985.
58. Marrinan EM, LaBrie RA, Mulliken JB: Velopharyngeal function in nonsyndromic cleft palate: Relevance of surgical technique, age at repair, and cleft type. *Cleft Palate Craniofac J* 35:95–100, 1998.
59. McCance AM, Moss JP, Fright WR, et al: Three-dimensional analysis techniques. Part 1: Three-dimensional soft-tissue analysis of 24 adult cleft palate patients following Le Fort I maxillary advancement: A preliminary report. *Cleft Palate Craniofac J* 34:36, 1997.
60. McCance AM, Orth M, Moss JP, et al: Three-dimensional analysis techniques. Part 4: Three-dimensional analysis of bone, and soft tissue to bone ratio of movements in 24 cleft patients following Le Fort I osteotomy: A preliminary report. *Cleft Palate Craniofac J* 43:58–62, 1997.
61. McComb R, Marrinan E, Nuss RC, et al: Predictors of velopharyngeal insufficiency after Le Fort I maxillary advancement in patients with cleft palate. *J Oral Maxillofac Surg* 69:2226–2232, 2011.
62. McKinstry RE: *Cleft palate dental care: A historical perspective*, Arlington, Va, 2000, ABI Publications.
63. Molina F, Ortiz Monasterio F, de la Paz Aguilar M, Barrera J: Maxillary distraction: Aesthetic and functional benefits in cleft lip-palate and prognathic patients during mixed dentition. *Plast Reconstr Surg* 101:951, 1998.
64. Motohashi N, Kuroda T, Filho LC, et al: P-A cephalometric analysis of nonoperated adult cleft lip and palate. *Cleft Palate Craniofac J* 31:193, 1994.
65. Nanda SK: Patterns of vertical growth in the face. *Am J Orthod Dentofacial Orthop* 93:103–116, 1988.
66. Obwegeser HL: Surgery as an adjunct to orthodontics in normal and cleft palate patients. *Rep Congr Eur Orthod Soc* 42:343–353, 1966.
67. Obwegeser HL: Surgical correction of deformities of the jaws in adult cleft cases. Paper read at the First International Conference on Cleft Lip and Palate, Houston, Tex, April 14-17, 1969.
68. Obwegeser HL: Surgical correction of small or retrodisplaced maxillae: The "dish-face" deformity. *Plast Reconstr Surg* 43:351, 1969.
69. Obwegeser HL: Surgical correction of maxillary deformities. In Grabb WC, Rosenstein SW, Bzoch KR, editors: *Cleft lip and palate*, Boston, 1971, Little and Crown, pp 515–556.
70. Obwegeser HL, Lello GE, Farmand M: Correction of secondary cleft deformities. In Bell WH, editor: *Surgical correction of dentofacial deformities. New concepts,* Vol III, Philadelphia, 1985, Saunders, 13, pp 592–638.
71. Perko M: The history of treatment of cleft lip and palate. *Prog Pediatr Surg* 20:238–251, 1986.
72. Phillips JH, Klaiman P, Delorey R, MacDonald DB: Predictors of velopharyngeal insufficiency in cleft palate orthognathic surgery. *Plast Reconstr Surg* 115:681–686, 2005.
73. Polley JW, Figueroa AA: Management of severe maxillary deficiency in childhood and adolescence through distraction osteogenesis with an external, adjustable, rigid distraction device. *J Craniofac Surg* 8:181, 1997.

74. Polley JW, Figueroa AA: Rigid external distraction: Its application in cleft maxillary deformities. *Plast Reconstr Surg* 102:1360, 1998.
75. Posnick JC: Orthognathic surgery in the cleft patient. In Russel RC, editor: *Instructional courses, Plastic Surgery Education Foundation,* St. Louis, 1991, CV Mosby Co, 4:129–157.
76. Posnick JC: Orthognathic surgery for the cleft lip and palate patient. *Semin Orthod* 2(3):205–214, 1996.
77. Posnick JC: The treatment of secondary and residual dentofacial deformities in the cleft patient: Surgical and orthodontic therapy. *Clin Plast Surg* 24(3):583–597, 1997.
78. Posnick JC: Cleft-orthognathic surgery: The isolated cleft palate deformity. In Posnick JC, editor: *Craniofacial and maxillofacial surgery in children and young adults*, Philadelphia, 2000, WB Saunders Co, 36, pp 951–978.
79. Posnick JC: The staging of cleft lip and palate reconstruction: Infancy through adolescence. In Posnick JC, editor: *Craniofacial and maxillofacial surgery in children and young adults*, Philadelphia, 2000, WB Saunders Co, 32, pp 785–826.
80. Posnick JC, Agnihotri N: Managing chronic nasal airway obstruction at the time of orthognathic surgery: A twofer. *J Oral Maxillofac Surg* 69:695–701, 2011.
81. Posnick JC, Ricalde P: Cleft-orthognathic surgery. *Clin Plast Surg* 31(2):315–330, 2004.
82. Posnick JC, Ruiz R: Repair of large anterior palatal fistulas using thin tongue flaps [discussion]. *Ann Plast Surg* 45:114–117, 2000.
83. Posnick JC, Taylor M: Skeletal stability and relapse patterns after Le Fort I osteotomy using miniplate fixation in patients with isolated cleft palate. *Plast Reconstr Surg* 94:51–58, 1994.
84. Posnick JC, Tompson B: Cleft-orthognathic surgery: Complications and long-term results. *Plast Reconstr Surg* 96(2):255–266, 1995.
85. Rosenstein SW: Facial growth and the need for orthognathic surgery after cleft palate repair: Literature review and report of 28 cases [discussion]. *J Oral Maxillofac Surg* 55:698, 1997.
86. Ross RB: The clinical implications of facial growth in the cleft lip and palate. *Cleft Palate J* 7:37–47, 1970.
87. Shaw WC, Asher-McDade C, Brattstrom V, et al: A six-center international study of treatment outcomes in patients with clefts of the lip and palate. *Cleft Palate Craniofac J* 29:393, 1992.
88. Stoelinga PJ, vd Vijver HR, Leenen RJ, et al: The prevention of relapse after maxillary osteotomies in cleft palate patients. *J Craniomaxillofac Surg* 15:325, 1987.
89. Trindale IE, Yamashita RP, Suguimoto RM, et al: Effects of orthognathic surgery on speech and breathing of subjects with cleft lip and palate: Acoustic and aerodynamic assessment. *Cleft Palate Craniofac J* 40:54–64, 2003.
90. Will LA: Growth and development in patients with untreated clefts. *Cleft Palate Craniofac J* 37:523–526, 2000.
91. Williams AC, Bearn D, Mildinhall S, et al: Cleft lip and palate care in the United Kingdom—the Clinical Standards Advisory Group (CSAG) study. Part 2: Dentofacial outcomes and patient satisfaction. *Cleft Palate Craniofac J* 38:24–29, 2001.
92. Willmar K: On Le Fort I osteotomy: A follow-up study of 106 operated patients with maxillofacial deformity. *Scand J Plast Reconstr Surg* 12(Suppl 1):1–68, 1974.
93. Wiltfang J, Hirschfelder U, Neukam FW, et al: Long-term results of distraction osteogenesis of the maxilla and midface. *Br J Oral Maxillofac Surg* 40:473, 2002.
94. Witzel MA, Munro IR: Velopharyngeal insufficiency after maxillary advancement. *Cleft Palate J* 14:176, 1977.
95. Wolford LM: Effects of orthognathic surgery on nasal form and function in the cleft patient. *Cleft Palate Craniofac J* 29:546–555, 1992.
96. Wolford LM, Cassano DS, Cottrell DA, et al: Orthognathic surgery in the young cleft patient: Preliminary study on subsequent facial growth. *J Oral Maxillofac Surg* 66:2524–2536, 2008.
97. Wolford LM, Karras SC, Mehra P: Considerations for orthognathic surgery during growth: Part 1. Mandibular deformities. *Am J Orthod Dentofacial Orthop* 119:95, 2001.
98. Wolford LM, Karras SC, Mehra P: Considerations for orthognathic surgery during growth: Part 2. Maxillary deformities. *Am J Orthod Dentofacial Orthop* 119:102, 2001.
99. Yoshida H, Nakamura A, Michi K, et al: Cephalometric analysis of maxillofacial morphology in unoperated cleft palate patients. *Cleft Palate Craniofac J* 29:419–424, 1992.

35

Management of Secondary Jaw Deformities after Maxillofacial Trauma

JEFFREY C. POSNICK, DMD, MD

- Posttraumatic Temporomandibular Joint Ankylosis in the Pediatric Population
- Posttraumatic Saddle-Nose Deformity
- Posttraumatic Orthognathic Deformities
- Posttraumatic Segmental Dentoalveolar Defects
- Conclusions

Despite the best of intentions by the primary trauma team, a subgroup of individuals who sustain facial injuries later present with deformities that require secondary reconstruction. The initial evaluation and resuscitation of the individual who has sustained craniomaxillofacial trauma is similar to that of patients with other organ system injuries. At times, the patient's general health and other body region injuries are likely to take precedence over the management of any facial fracture. In addition, there are treatment options to consider for each craniomaxillofacial fracture, and judgment is required to select the approach that is most likely to result in a favorable outcome: open versus closed reduction; method of fixation; grafting requirements; immediate or delayed dental treatment; and the management of associated soft-tissue injuries. Treatment is individualized to accommodate the healing of each fracture and to simultaneously consider cerebral function, vision, breathing, chewing, swallowing, speech, the cervical spine, and temporomandibular joint (TMJ) injuries and to maintain cardiovascular health. Another factor is the residual facial growth requirements in a child who sustains an injury and who has not yet reached skeletal maturity. If the injured and repaired craniomaxillofacial bones do not grow normally, secondary deformities result over time. For all of these reasons, secondary deformities after facial skeletal injury (with or without effective primary repair) may occur and require later reconstruction. Specific patterns of secondary maxillofacial deformities are seen with enough frequency that each is reviewed separately.

Posttraumatic Temporomandibular Joint Ankylosis in the Pediatric Population

Background

The treatment of TMJ ankylosis poses challenges to the maxillofacial surgeon as a result of technical difficulties associated with access to the joint, the currently available autogenous and prosthetic TMJ replacement options, and the high incidence of ankylosis recurrence. Failure to restore adequate mandibular opening is likely to result in speech and swallowing impairment; difficulty with mastication; poor oral hygiene and dental neglect; continued facial growth disturbances; and the potential for airway compromise. The surgical management of bony ankylosis of the TMJ requires the complete excision of the involved osseous mass with intraoperative achievement of satisfactory passive mouth opening. The immediate reconstruction with a costochondral rib graft is often carried out, and this is followed by a postoperative physiotherapy regimen to maintain mouth opening. A number of authors have critically evaluated the treatment of TMJ ankylosis in adults, with less emphasis placed on addressing this problem exclusively in the pediatric population.*

In a study by Posnick and colleagues, a consecutive series of nine pediatric patients (mean age, 7.7 years) who underwent a standardized treatment protocol for 13 affected ankylosed temporomandibular joints was reviewed.[88] Four patients had unilateral TMJ ankylosis, and five had bilateral ankylosis. One child required bilateral release but only unilateral reconstruction. Radiographic evidence demonstrated bony ankylosis in all 13 operated joints. Two patients had

*References 1, 3-6, 8, 12, 13, 16-19, 34, 36, 41, 42, 44-47, 56, 57, 59, 61, 62, 67, 68, 71, 73, 76, 78-81, 87, 88, 96, 98-100, 102-104, 116, 119, 120, 122, 123

previously undergone surgical intervention of the TMJ. The cause of ankylosis within the study group was primarily traumatic or congenital. The protocol that was followed included complete excision of the involved ankylotic structures with mobilization through coronal scalp and Risdon neck incisions as well as the achievement of wide mouth opening. This was followed by immediate costochondral grafting. Fixation with mini-plates and screws allowed for early mobilization with the rapid institution of a physiotherapy program (Fig. 35-1).

For the study patients, despite the achievement of full passive mouth opening in the operating room, less long-term success was realized. Those with unilateral TMJ ankylosis maintained the best long-term vertical opening. On average, they went from 5 mm of maximum vertical opening (separation of the incisors) before surgery to 25 mm of active range of motion maintained for the long term. For patients with bilateral TMJ ankylosis, the maximum long-term incisal opening was only 18 mm. Mean follow-up duration at the close of the study was 2 years. Perioperative complications were minimal, with no evidence of infection, facial nerve injuries, or need for transfusion. In general, patients with unilateral or bilateral ankylosis of traumatic cause achieved satisfactory functional results after surgery, whereas those in the bilateral congenital TMJ ankylosis group attained results that were far more limited. This likely represents limitations in the intrinsic neuromotor function of the congenital cases.

Controversies and Unresolved Issues

Knowing the cause of TMJ ankylosis helps with the understanding of its pathophysiology. In developed countries, the most common etiology is trauma, followed by infection.[33,48,121,125,126] Topazian found an association with trauma in 39% and with infection in 43% of patients with TMJ ankylosis (N = 229).[119] Since the late 1970s, authors have found trauma to be a more frequent cause than infection, probably because of the use of antibiotics to combat the latter. In a report of a series of patients with TMJ ankylosis, Rajgopal and colleagues concluded that 80% of cases were traumatic in origin.[96] Unfortunately, these reviews lumped both pediatric and adult patients together.

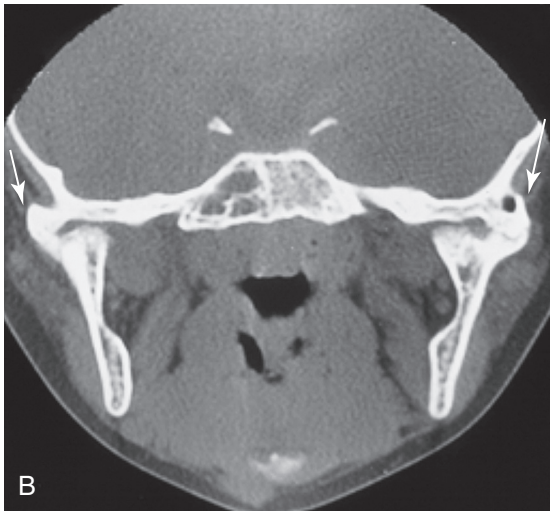
Surgical attempts to release TMJ ankylosis have been described dating back to 1850.[5] Since then, ankylosis release followed by reconstruction using a variety of alloplastic and autogenous materials has been repoted.[9,15,34,46,50,53,62,68,72,79,81,101,114,124,129] Although Gillies first described costochondral grafting for TMJ reconstruction in 1920,[30] it was Poswillo[93,94] and MacIntosh and Henny[53] who popularized its use during the 1970s. Poswillo attempted to demonstrate the histologic and physiologic similarities between the mandibular condyle and rib cartilage in humans.[93] MacIntosh and Henny described 26 cases of costochondral grafting for mandibular condyle replacement, with 6 of them being for ankylosis.[53] Their approach included graft stabilization with interosseous wires and intermaxillary fixation (IMF) for 6 to 8 weeks. They claimed subsequent growth of the grafts in their pediatric patients, but they failed to provide any objective data.

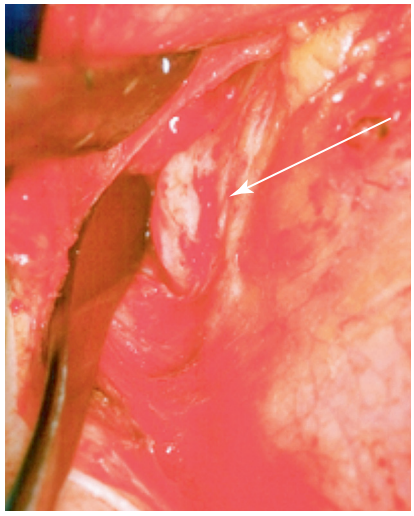
In 1973, Kennett reported on two cases of unilateral TMJ ankylosis in young patients who underwent the combination of condylectomy, coronoidectomy, and reconstruction with a wired rib graft.[45] He concluded that ankylosis in children should be treated as soon as possible and that recurrence is the most frequent complication. In 1986, Munro and colleagues reviewed their series of 18 adult and pediatric patients with TMJ ankylosis and facial deformity.[67] Their reconstruction included interosseous wiring of the rib graft and 8 weeks of IMF followed by physiotherapy. They demonstrated better results in unilateral ankylosis cases than in bilateral cases. The next year, Lindqvist and associates described 27 patients of varying ages with TMJ ankylosis; 25 of them underwent costochondral graft reconstruction. Fixation was also by direct interosseous wires, but IMF was reduced to 3.5 weeks to encourage early mobilization and to limit recurrence. The maximum incisor opening improved from 16 mm to 31 mm. In 1987, Politis and others reported satisfactory results in six patients (five adults) with ankylosis.[81] They employed a preauricular approach that involved the use of costochondral grafts and either wire or plate and screw fixation followed by 2 or 6 weeks of IMF, respectively. However, 50% of their patients developed seventh cranial nerve palsies. In 1990, Kaban and colleagues described their experience in 14 adult and pediatric patients with TMJ ankylosis and indicated satisfactory results.[41,42] The protocol reported by Posnick and colleagues was similar to that of Kaban and colleagues except that the former found that the use of a mini-plate with screw fixation improved stability (as compared with interosseous wire fixation), thereby allowing for the minimal use of IMF and the early initiation of mandibular range of motion with active physiotherapy.[88]

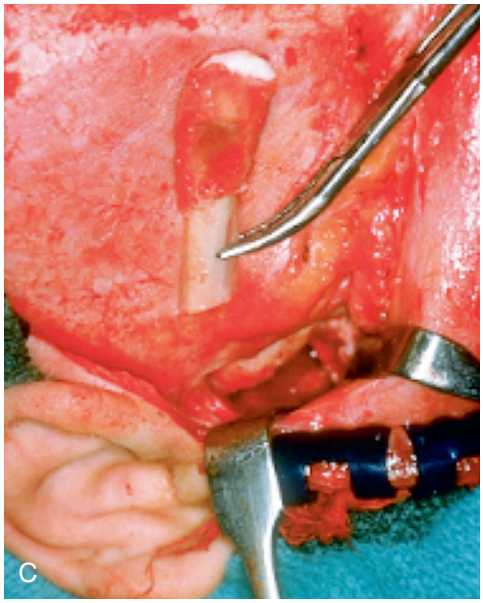
In 2009, Kaban and colleagues described an updated protocol for the management of TMJ ankylosis exclusively in children.[78,79,122,123] They restated their observation that the most common cause of treatment failure was inadequate resection of the ankylotic mass followed by the failure to achieve adequate passive maximum opening in the operating room. Clearly, unless adequate passive mandibular vertical opening is achieved in the operating room, failure will inevitably occur. Unfortunately, this is no guarantee that mouth opening will be maintained. Their seven-step protocol consisted of the following:

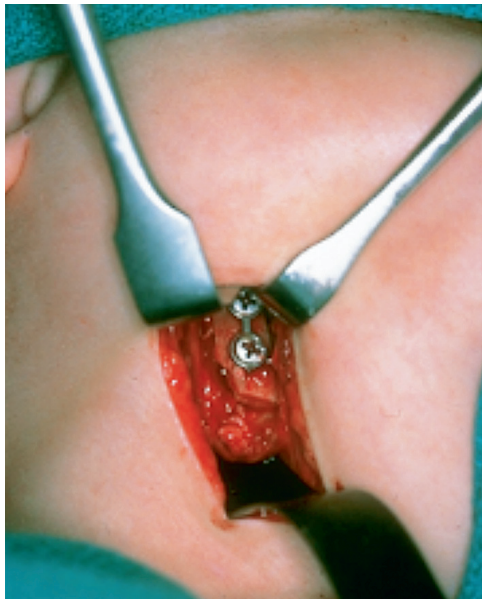
1. Complete excision of the fibrous and/or bony ankylotic mass
2. Coronoidectomy of the affected side
3. Coronoidectomy of the contralateral side if steps 1 and 2 do not result in a maximum incisal opening (>35 mm) or opening to the point of dislocation of the unaffected TMJ
4. Lining of the TMJ with either a temporalis myofascial flap or the native disc, if it can be salvaged

Bony ankylosis



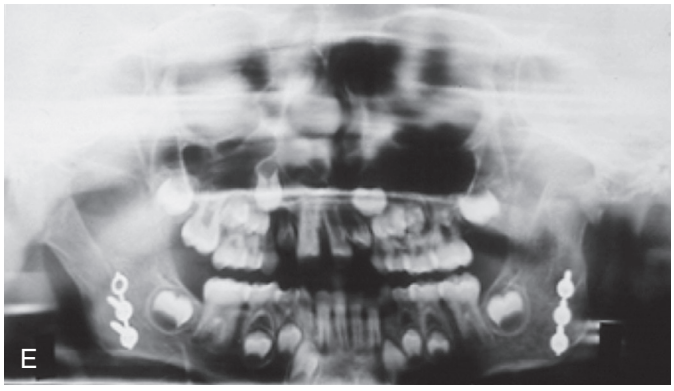


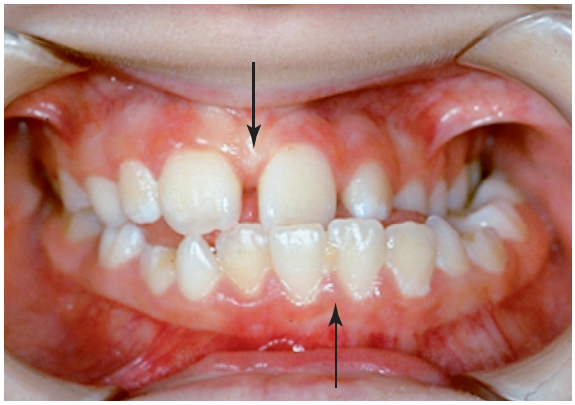




• **Figure 35-1** A 5-year-old girl was referred to this surgeon after intracapsular condyle fractures of the mandible resulted in bilateral temporomandibular joint ankylosis. These fractures were unrecognized and went untreated at the time of injury 1 year earlier. **A,** Preoperative frontal views showing maximal opening limited to 4 mm. **B,** Preoperative computed tomography scan that demonstrates the bony ankylosis of each condyle to its zygomatic arch. Intraoperative view demonstrates ankylosis to the zygomatic arch to the condyle as viewed through the coronal scalp incision. **C,** Intraoperative view of costochondral graft placement through the coronal scalp incision. Stabilization of the rib graft through Risdon neck incision with the use of a mini-plate and screws.

Continued





• **Figure 35-1, cont'd** **D,** Facial views 1 year after reconstruction showing good cranial nerve VII function and satisfactory vertical mouth opening being maintained. **E,** Postoperative Panorex and anteroposterior facial radiographs. **F,** Facial view with maximum mouth opening maintained 3 years after reconstruction but with asymmetrical overgrowth of the costochondral grafts. Occlusal view 3 years after reconstruction that shows an asymmetrical Angle class III malocclusion as a result of the overgrowth.

5. Reconstruction of the ramus condyle unit with either proximal segment osteotomy and distraction osteogenesis or costochondral graft and plate and screw fixation
6. Early mobilization of the jaws: If distraction osteogenesis is used to reconstruct the ramus condyle unit, mobilization begins the day of operation. In patients who are undergoing costochondral graft reconstruction, mobilization begins after 10 days of IMF.
7. All patients receive rigorous physiotherapy after surgery to maintain mouth opening, generally for at least 3 to 6 months

Observations and Recommendations

Several generalizations can be made about pediatric patients with TMJ ankylosis. *First,* in first-world countries as in the adult population, trauma remains a prevalent cause, and this is followed closely by congenital issues. In underdeveloped countries, infection continues to be an important causative factor.[92] *Second,* although patients with unilateral ankylosis present with a severely limited preoperative opening, they are more likely to achieve satisfactory long-term functional results. *Third,* children with bilateral congenital TMJ ankylosis generally attain poor long-term results, despite adequate intraoperative release. This is likely the result of associated masticatory muscle anomalies, neuromuscular discoordination, or longstanding muscular disuse atrophy. *Fourth,* the use of mini-plate and screw fixation of the graft diminishes the need for immobilization, thereby allowing for the early institution of physiotherapy. Unfortunately, this does not guarantee the maintenance of a satisfactory long-term opening. *Fifth,* when a costochondral graft is used in children, only several millimeters of cartilage should remain. This is to limit the frequent complication of overgrowth and its complex secondary deformities. In clinical practice, overgrowth after costochondral grafting remains a frequent occurrence (see Chapter 28 and Fig. 35-1). *Sixth,* for most patients, reconstruction to achieve long-term maxillomandibular harmony must wait until they have achieved skeletal maturity (i.e., 14 to 18 years of age).

Posttraumatic Saddle-Nose Deformity

Background

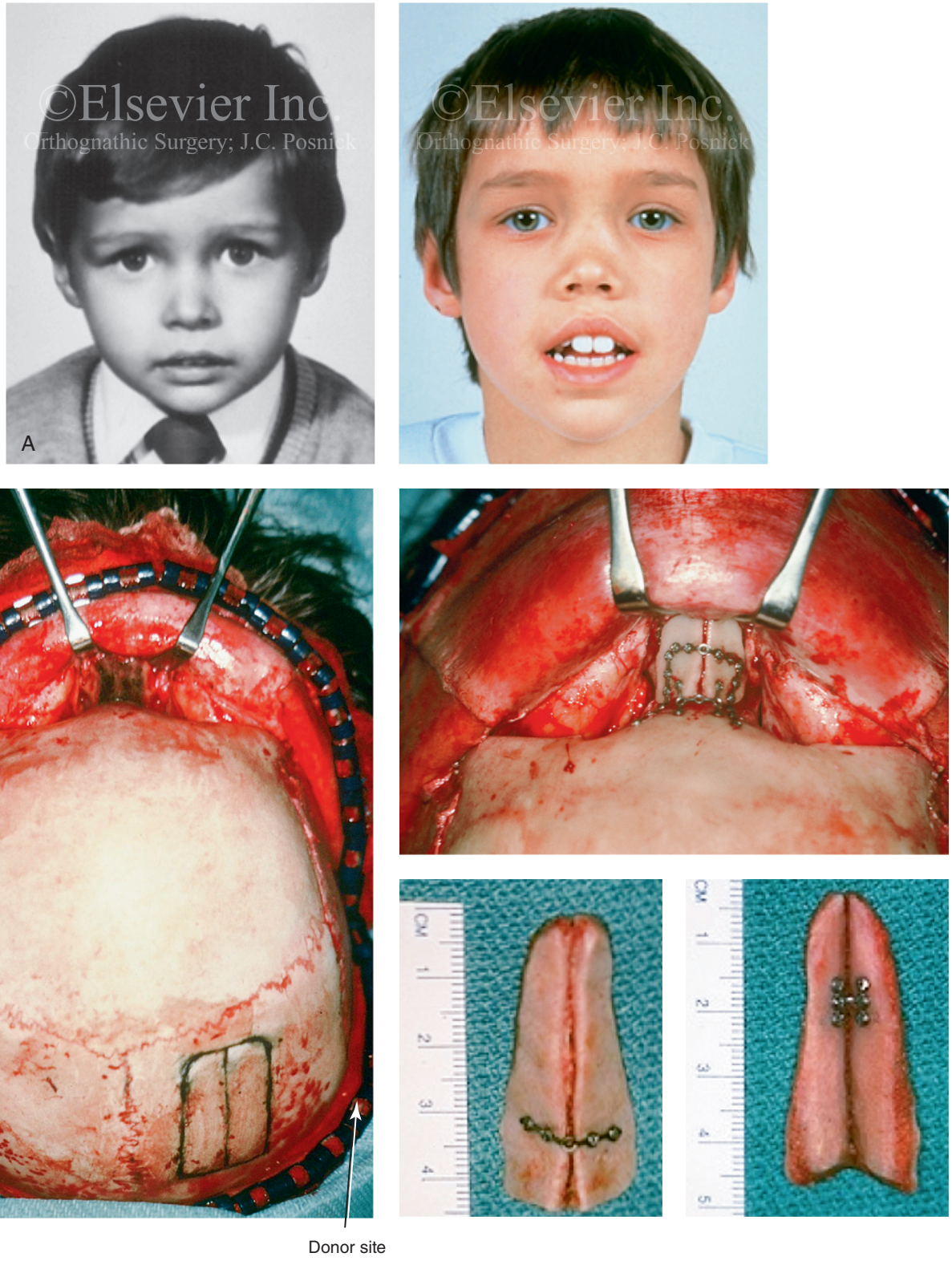
Nasal injuries are often recognized but then ignored as being unimportant.[11,82,86] When a depressed nasal fracture occurs in a child, injury to the growth center leaves the nose prone to a so-called "saddle deformity" with flattening of the osseocartilaginous vault.[63] In general, the overlying soft tissues, the upper and lower lateral cartilages, and the nasal lining are distorted but remain intact. Reconstruction of a saddle deformity that involves the bony and cartilaginous dorsum is generally carried out with an autogenous graft (e.g., chondral, costochondral, iliac, split- or full-thickness cranial) (Fig. 35-2).[27,28,32,38,60,74,91,117,118,131] The use of an allogenic graft (e.g., porous polypropylene) is always a second choice as a result of a higher incidence of associated infection and extrusion.[43] The type of graft material selected (e.g., bone, cartilage, allograft), the method of fixation, the incisions required for access, the extent of dorsum that requires reconstruction, and the timing of treatment are all important details and will vary according to the age of the patient, the extent of the deformity, and the surgeon's personal preferences (see Chapter 38).

Involvement of Both the Bone and the Cartilage Vault

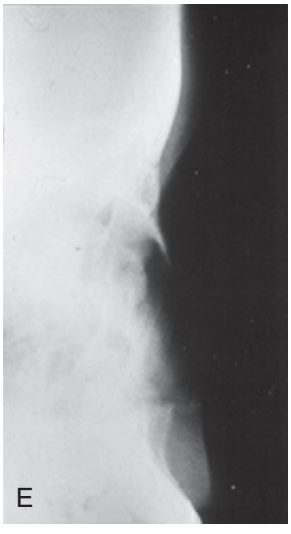
The autogenous graft used (e.g., chondral, costochondral, rib, iliac, split- or full-thickness cranial) is contoured to provide reasonable nasal dorsum morphology. When the whole dorsum requires reconstruction (i.e., the radix to the nasal tip), the lower lateral cartilages are sutured over the top of the graft to provide a more natural tip contour and feel (see Fig. 35-2). When a bone graft extends from the radix to the tip, minor degrees of graft resorption at the tip should be anticipated. When correcting the saddle deformity with bone graft, freshening the base of the nasal bones with a rotary drill before graft placement is carried out; the onlay graft will then rest on a bleeding base. The accurately shaped graft is either dovetailed into a bony groove at the nasofrontal process or abutted to the frontal bones while resting evenly on the contoured and freshened) dorsal base to establish the correct nasofrontal angle. Stabilization of the bone graft is either with microplates and screws or transcutaneous Kirschner wires, depending on the access provided by the incisions that are used for the reconstruction. If a coronal scalp incision or a direct vertical nasal incision is used, plate and screw fixation is easily accommodated. An open approach (i.e., a columella splitting incision) should be combined when a coronal scalp incision is used to best manage the lower lateral cartilages and the nasal tip (see Chapter 38 and Fig. 35-2).

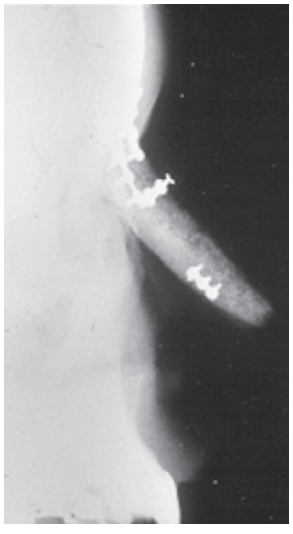
Involvement of the Cartilage Vault Only

When the saddle deformity causes collapse of the septal cartilage without injury to the nasal bones, then a cartilage graft reconstruction is preferred. A rib cartilage dorsal strut graft is crafted and placed flush with the nasal bones. The graft then extends caudal to the tip. The tip of the dorsal strut graft is joined to a second rib cartilage graft (caudal strut) that extends from the base of the maxilla to the nasal tip. The caudal strut is stabilized with a short buried Kirschner wire (no. 35 threaded) that is secured to the base of the maxilla/anterior nasal spine region and then pierced into the graft (Fig. 35-3). There is no advantage to harvesting the L-shaped rib cartilage graft as one unit. The separately crafted dorsal strut and caudal strut grafts are then joined together at the tip with non-resorbable suture. The lower lateral cartilages are sutured together and over the



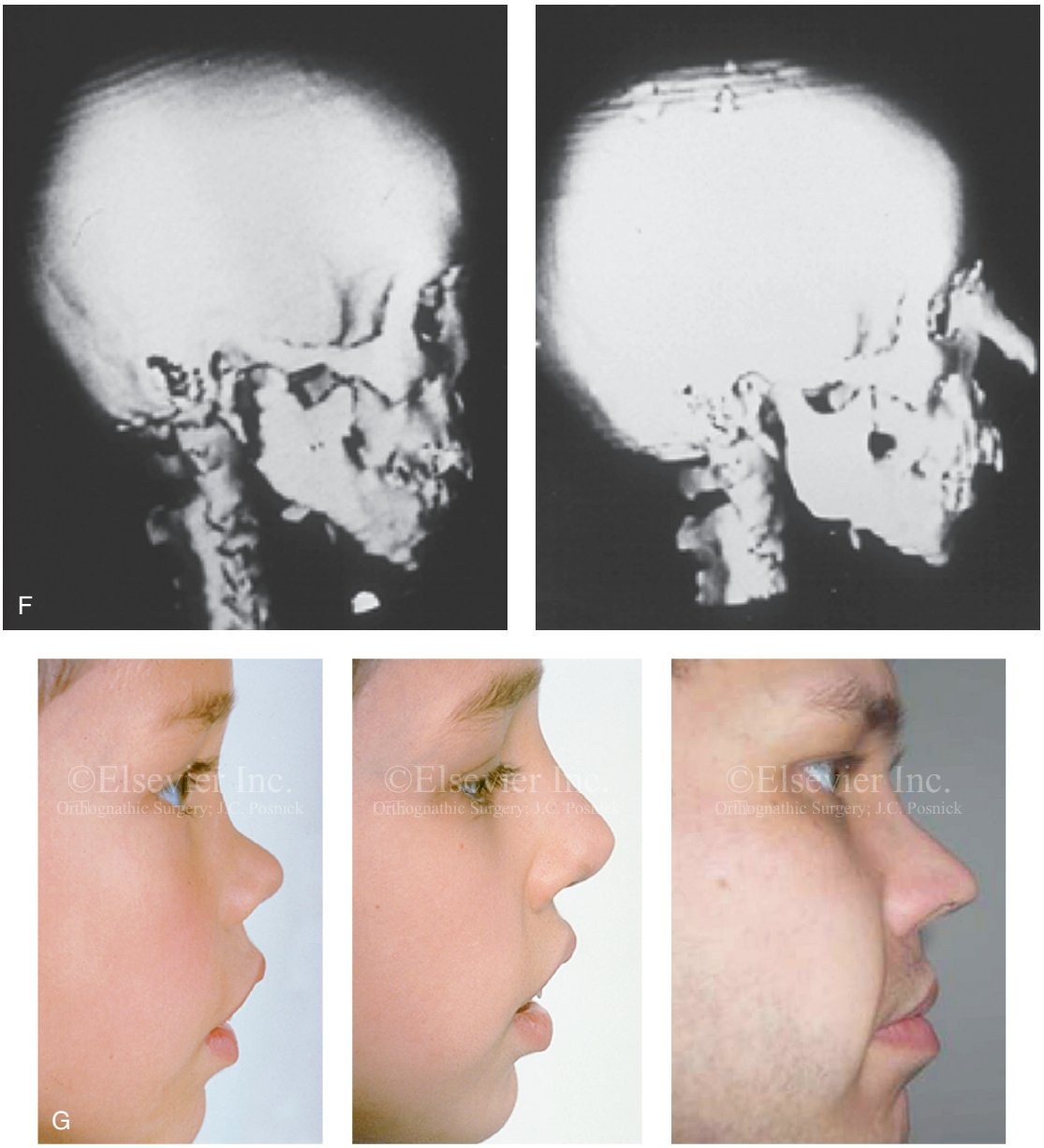
• **Figure 35-2** A 4-year-old boy fell and hit the bridge of his nose on a hard tabletop, which resulted in a saddle-nose deformity (i.e. osseous and cartilagenous vault). He arrived for evaluation when he was 8 years old and then underwent reconstruction through a coronal scalp incision with nasal osteotomies (in-fracture) and the placement of fixed crafted full-thickness cranial grafts. **A,** Frontal view at 3 years of age just before injury and at 8 years of age just before surgery. **B,** Intraoperative views through the coronal scalp incision that show the frontonasal region and the proposed right calvarial donor site. Crafted full-thickness cranial bone grafts are shown before placement for nasal reconstruction. Stabilization is achieved with microplates and screws. A close-up view of the frontonasal region shows the full-thickness cranial bone grafts in place and stabilized with microplates and screws.





• **Figure 35-2, cont'd C,** Frontal views before and 6 months after reconstruction. **D,** Oblique views before and 6 months after reconstruction. **E,** Lateral radiographs of the nose before and 6 months after reconstruction.

Continued



• **Figure 35-2, cont'd F,** Computed tomography scan views before and 6 months after reconstruction. **G,** Close-up lateral view just before surgery, 6 months after treatment, and then 16 years after reconstruction. *A, B, C, E, F, from Posnick JC: The role of plate and screw fixation in the management of pediatric facial fractures. In Gruss JS, Manson PM, Yaremchuk MJ, eds:* Rigid fixation of the craniomaxillofacial skeleton, *Stoneham, Mass, 1992, Butterworth-Heinemann, p 1412.*

grafts to form the new nasal tip. This technique is efficient and results in a "soft" nasal tip that looks and feels natural (see Chapter 38).

Posttraumatic Orthognathic Deformities

Background

At times, the resuscitation of the trauma patient may lead to incomplete preoperative imaging and evaluation and the rushed treatment of the associated maxillofacial injuries. At other times, the primary facial fracture management is delayed by injury of multiple systems. If so, the incidence of infection, malunion, or non-union that results in secondary deformities and malocclusion is increased. In severely traumatized patients, neurologic injuries are often the main concern, especially during the first days of observation. Maxillofacial injuries may be neglected and the status of the occlusion not properly evaluated as a result of the need for orotracheal intubation, masticatory muscle spasm, difficulty assessing a comatose or uncooperative patient, or the lack of availability of a maxillofacial specialist. There may also be associated dental trauma (e.g., fracture or avulsion of

• **Figure 35-3** A middle-aged woman of Arabic origin with a postsurgical (iatrogenic) saddle-nose deformity. Two years earlier, she underwent an aggressive septoplasty procedure at another institution. This resulted in a large septal perforation with collapse of the cartilaginous vault. She arrived for evaluation and then underwent nasal reconstruction that included rib cartilage grafting (caudal and dorsal struts). **A,** Frontal views before and after reconstruction. **B,** Left oblique view before and after reconstruction. **C,** Profile view before and after reconstruction. **D,** Computed tomography scan views (coronal cut) of nasal septum confirming the extent of the septal deficits before reconstruction.

the teeth) that was not recognized, neglected, or managed unfavorably in a hospital setting without available dental expertise.

Posttraumatic secondary maxillomandibular deformities may occur as a result of inadequate initial anatomic reduction or stabilization and fixation of a jaw fracture or the late resorption and remodeling of the bones resulting in malunion.[23-25,35,101,105,111,115,132] The segmental loss of maxillary or mandibular dentoalveolar components may also have occurred, as discussed later in this chapter. In the child, limited growth of the bones after the initial injury (e.g., condylar fracture) or as a result of the surgical intervention carried out to reduce and stabilize the fracture may also cause secondary deformities.[58,108,113]

There are patterns of posttraumatic orthognathic deformities that occur with enough regularity that they are reviewed separately in the following sections of this chapter.

Malunion after Midface (Le Fort I) Fracture

An elongated face with an anterior open-bite malocclusion (counterclockwise rotated maxilla and clockwise rotated mandible) can occur when a Le Fort fracture is allowed to heal with inadequate reduction or stabilization at the time of initial management.[30,40,87,132] The displaced maxilla often heals inferiorly with counterclockwise rotation. The mandible is forced into a clockwise-rotated position that involves a retrusive pogonion seen in profile. Another common reason for malocclusion after midface fracture is an unrecognized palatal split with secondary arch-form deformity.[25,132] Reconstruction requires osteotomies to recreate the midface fractures followed by the surgical repositioning of the maxilla into the correct (preinjury) anatomic location. The uninjured mandible will then counterclockwise rotate to close the anterior open bite, to improve the horizontal projection at the pogonion, and to reduce the vertical height of the lower face.[43,87] Another frequent scenario is when a midface fracture occurs during childhood and then results in growth disturbance. At the time of facial growth maturity (i.e., 14 to 18 years of age), maxillary hypoplasia with a skeletal class III malocclusion will be evident (Fig. 35-4).

Late Secondary Consequences of a Condylar Fracture

There are many published opposing treatment protocols for the primary management of mandibular condyle fractures.[2,10,21,22,31,59,63-66,112,127,133] Despite much discussion, controversy remains regarding which type of condyle fracture should be treated open rather than closed and, if operated, what approach (i.e., incisions for access, extent of condylar fragment degloving, method of fixation, postoperative immobilization and remobilization protocol) is optimal for each specific fracture. In addition, when a condyle injury or fracture occurs in a child, it can easily be overlooked or misdiagnosed, especially if the individual has no other apparent injuries. The child may not even arrive for evaluation; if evaluation does occur, the patient may be seen by a general practitioner rather than a specialist. Radiographs may be limited as a result of the need for sedation or concern by the parents about ionized radiation exposure.[49,83,84]

Regardless of the primary treatment rendered, a frequently seen pattern of late secondary deformity results when the condylar injury occurs prior the completion of mandibular growth (Figs. 35-5 through 35-10).[26,29,69,83-85,87,89,90,95,109,110] Often there is asymmetrical growth of the mandible from that point forward, with ipsilateral mandibular hypoplasia (see Chapter 4). Canting of the mandible (up on the ipsilateral side), shift of the dental midline (toward the fracture), and malocclusion (ipsilateral Class II) are frequently seen. The upper jaw is secondarily affected, growing with vertical asymmetry (canting) and with a shift of the maxillary dental midline toward the side of the fracture.

When facial growth is complete, orthodontic alignment to eliminate dental compensations in combination with orthognathic surgery (Le Fort I osteotomy, sagittal ramus osteotomies of the mandible, and osseous genioplasty) will allow three-dimensional repositioning of the jaws to improve facial symmetry, to restore Euclidian proportions, and to correct the malocclusion (see Figs. 35-5 and 35-7). This classic posttraumatic dentofacial deformity and orthognathic approach to reconstruction assumes a stable posterior stop when seating the ipsilateral condyle, adequate mouth opening, and minimal TMD.

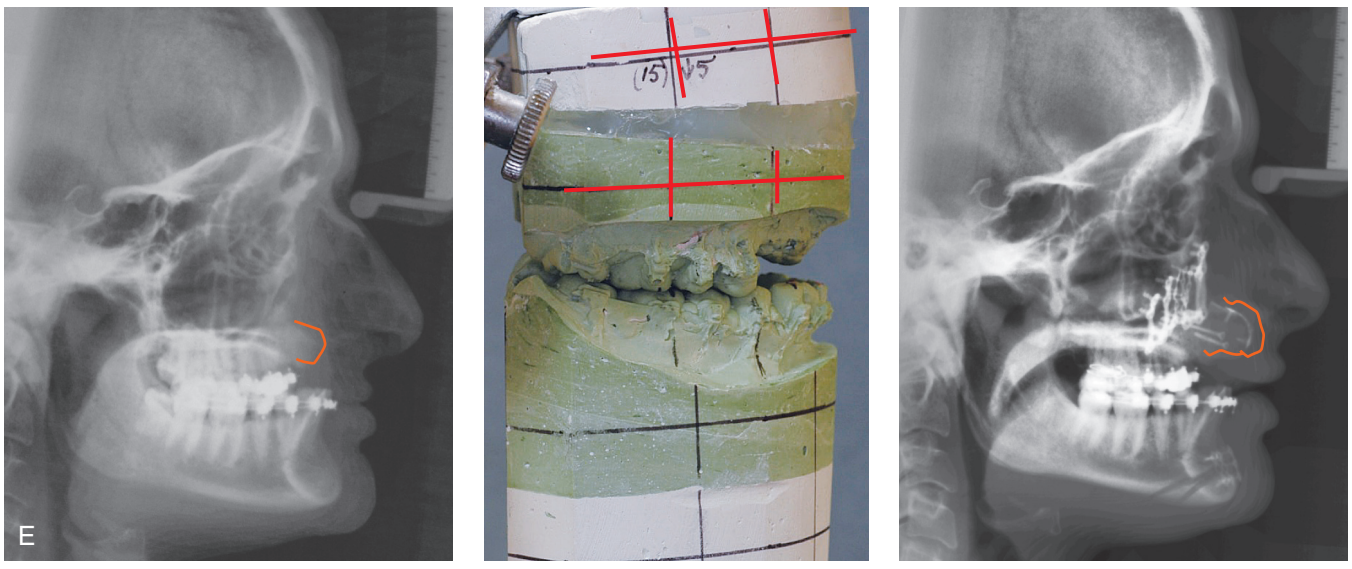
Orthognathic surgery is not preferred for patients with significant mandibular hypomobility. Limited mouth opening makes intraoral surgery difficult; in some cases, it may worsen when the cicatricial effects of a surgical wound are added.[14,20,70,77,106] If feasible, the hypomobility is resolved before the jaw deformity is definitively addressed (see Fig. 35-9).

When considering the timing of orthognathic surgery for a patient after a condyle fracture, another essential aspect is to wait until the temporomandibular articulation has stabilized. Becking and colleagues reported stable orthognathic results in their patients who had sustained displaced condylar fracture with a loss of posterior facial height.[3] They postponed definitive orthognathic treatment until at least 9 months after the initial injury. In clinical practice, each of these patients should be considered individually with regard to the stability of the temporomandibular articulation. In general, I like to allow 6 months of healing before proceeding with definitive reconstruction (see Fig. 35-10). Unfortunately, no one test or radiographic study can reliably answer the question of condylar stability (see Chapter 4).

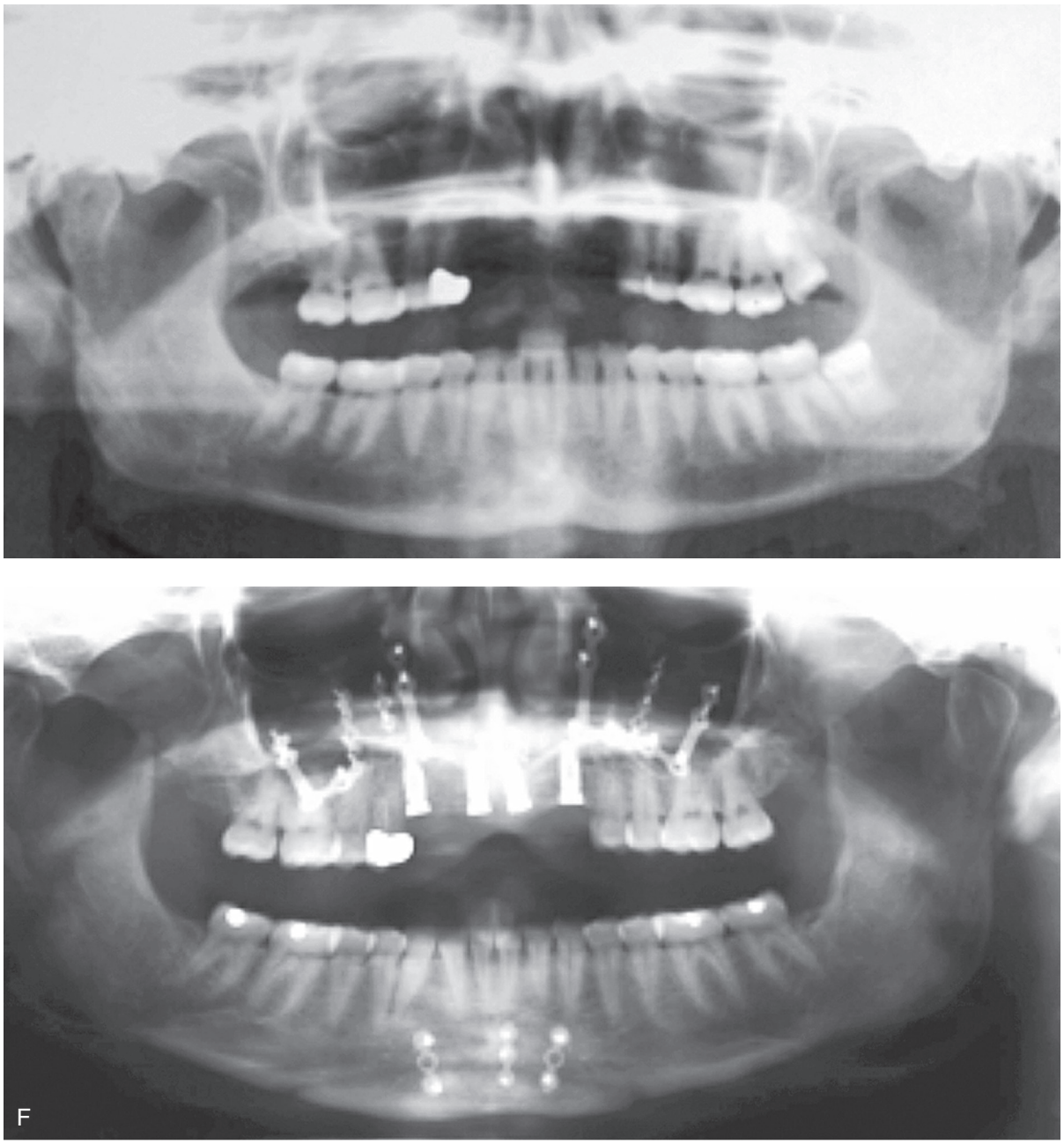
When a condylar neck fracture heals with resorption of the entire proximal condylar fragment, not only is there significant loss of ipsilateral posterior facial height but there is also an unstable posterior stop. Correction will require the construction of a neocondyle.[15,37,114,124,128,129] In this case, as long as there is satisfactory mouth opening, use a costochondral graft for reconstruction is preferred (see

Text continued on p. 1507

• **Figure 35-4** In this patient, midface trauma occurred during childhood and included the loss of the anterior teeth and the alveolar bone component. She presented during adulthood with maxillary hypoplasia, Class III malocclusion, and an anterior segmental dentoalveolar defect. Comprehensive reconstruction and dental rehabilitation involved a surgeon, a periodontist, an orthodontist, and a restorative dentist. The patient underwent Le Fort I osteotomy (horizontal advancement and vertical lengthening) with interpositional corticocancellous iliac bone grafting. A corticocancellous (iliac) bloc graft was simultaneously crafted to match the anterior maxillary defect; it was inset and secured with microplates and screws. This was followed 4 months later by the placement of four implants and then final restorations 6 months later. **A,** Frontal views with smile before and after reconstruction and rehabilitation. **B,** Oblique views before and after reconstruction and rehabilitation. **C,** Profile views before and after reconstruction and rehabilitation. *Continued*

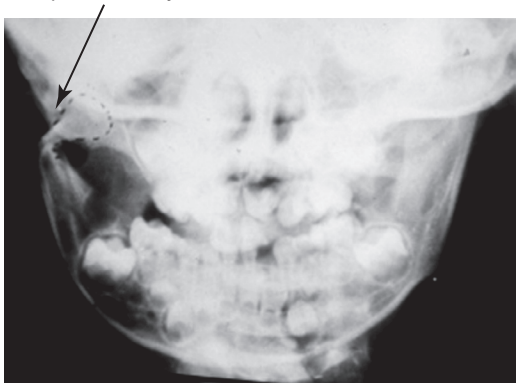


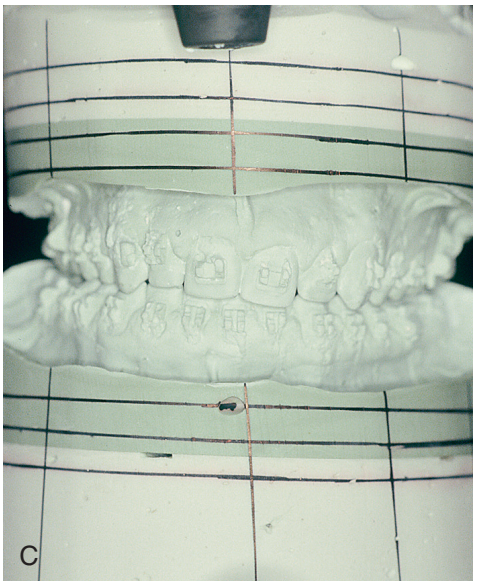
• **Figure 35-4, cont'd** **D,** Occlusal views before and after reconstruction and rehabilitation. **E,** Lateral cephalometric radiographs and analytic model planning before and after reconstruction.

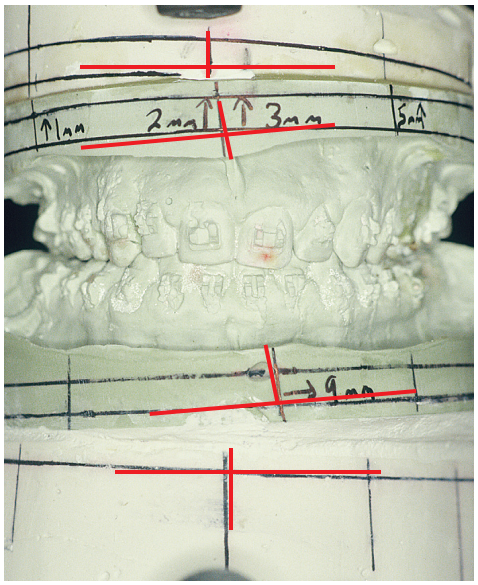


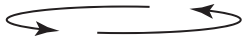
• **Figure 35-4, cont'd F,** Panorex radiographs before and after reconstruction and rehabilitation.

Continued





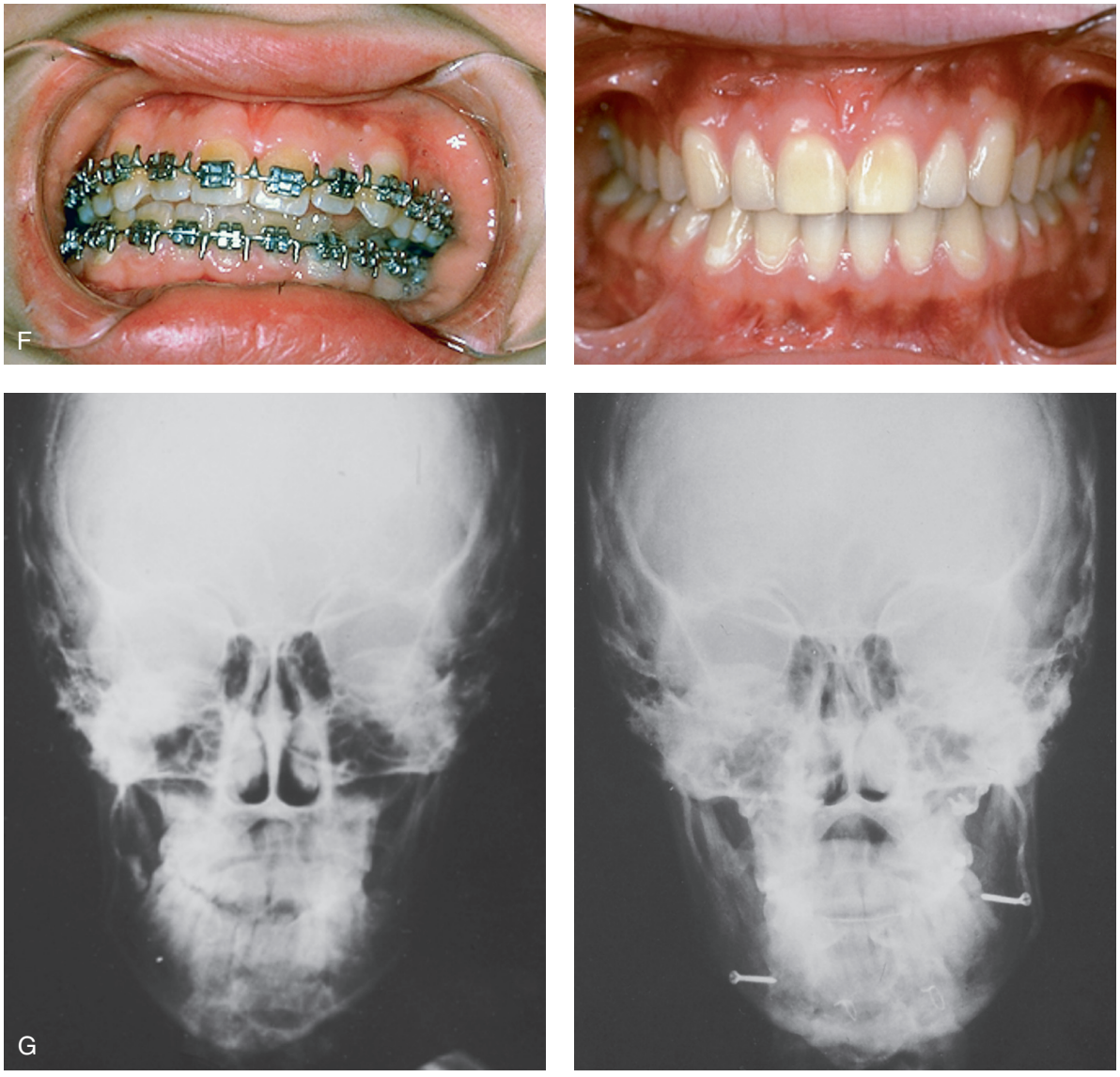




• **Figure 35-5** A 5-year-old girl sustained a right condyle fracture of the mandible and then presented as a teenager with resulting facial asymmetry that involved the maxilla and the mandible. She then underwent a comprehensive orthodontic and surgical approach that included a Le Fort I osteotomy; bilateral sagittal split ramus osteotomies; and an osseous genioplasty. **A,** Frontal view at 6 years of age, 1 year after right condyle fracture. The anteroposterior facial radiograph indicates a medially displaced right condyle fracture. **B,** Frontal view at 15 years of age. An anteroposterior cephalometric radiograph confirms facial asymmetry. **C,** Articulated dental casts that indicate analytic model planning.

• **Figure 35-5, cont'd** **D,** Frontal views before and 2 years after reconstruction. **E,** Profile views before and 2 years after reconstruction.

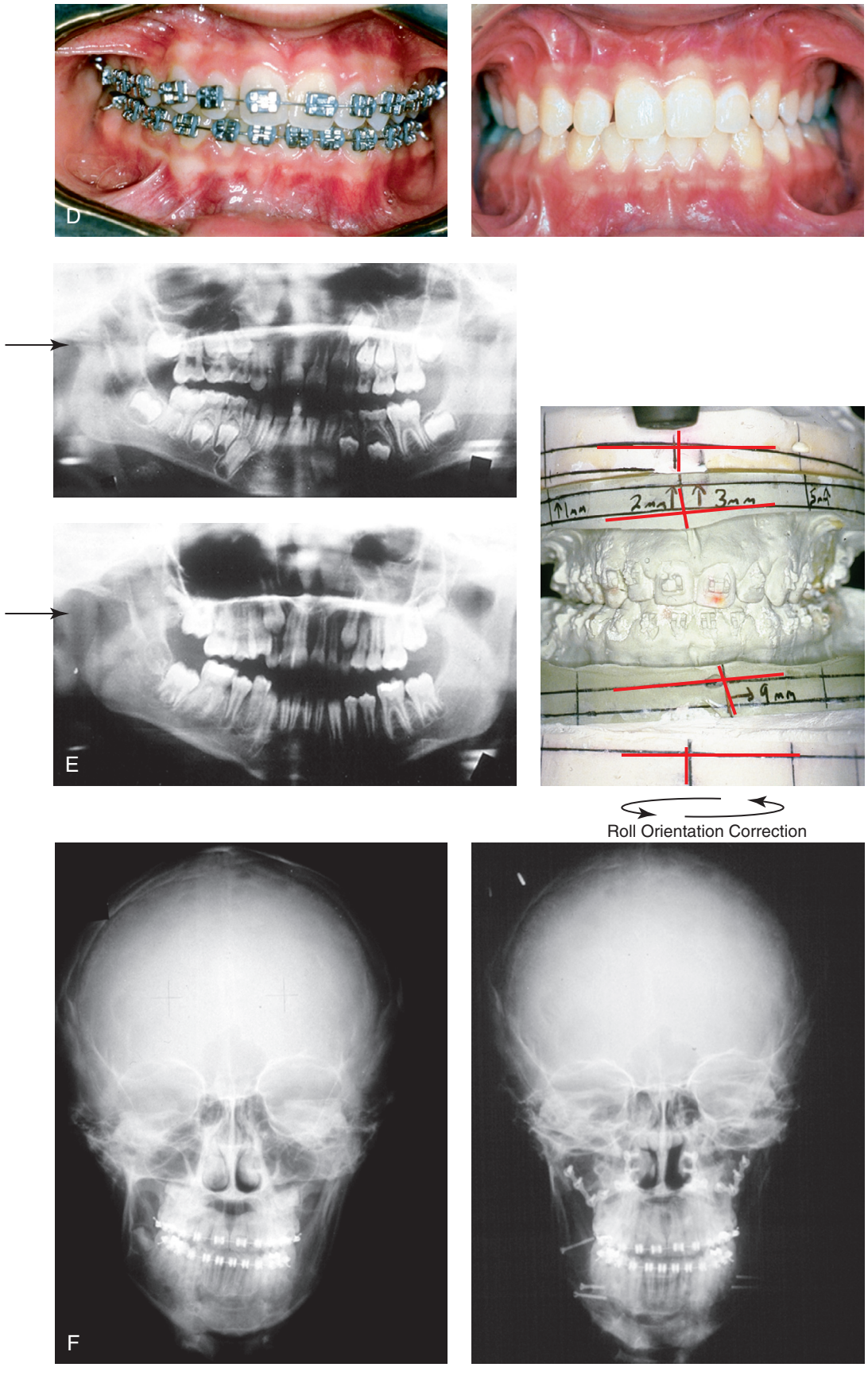
Continued



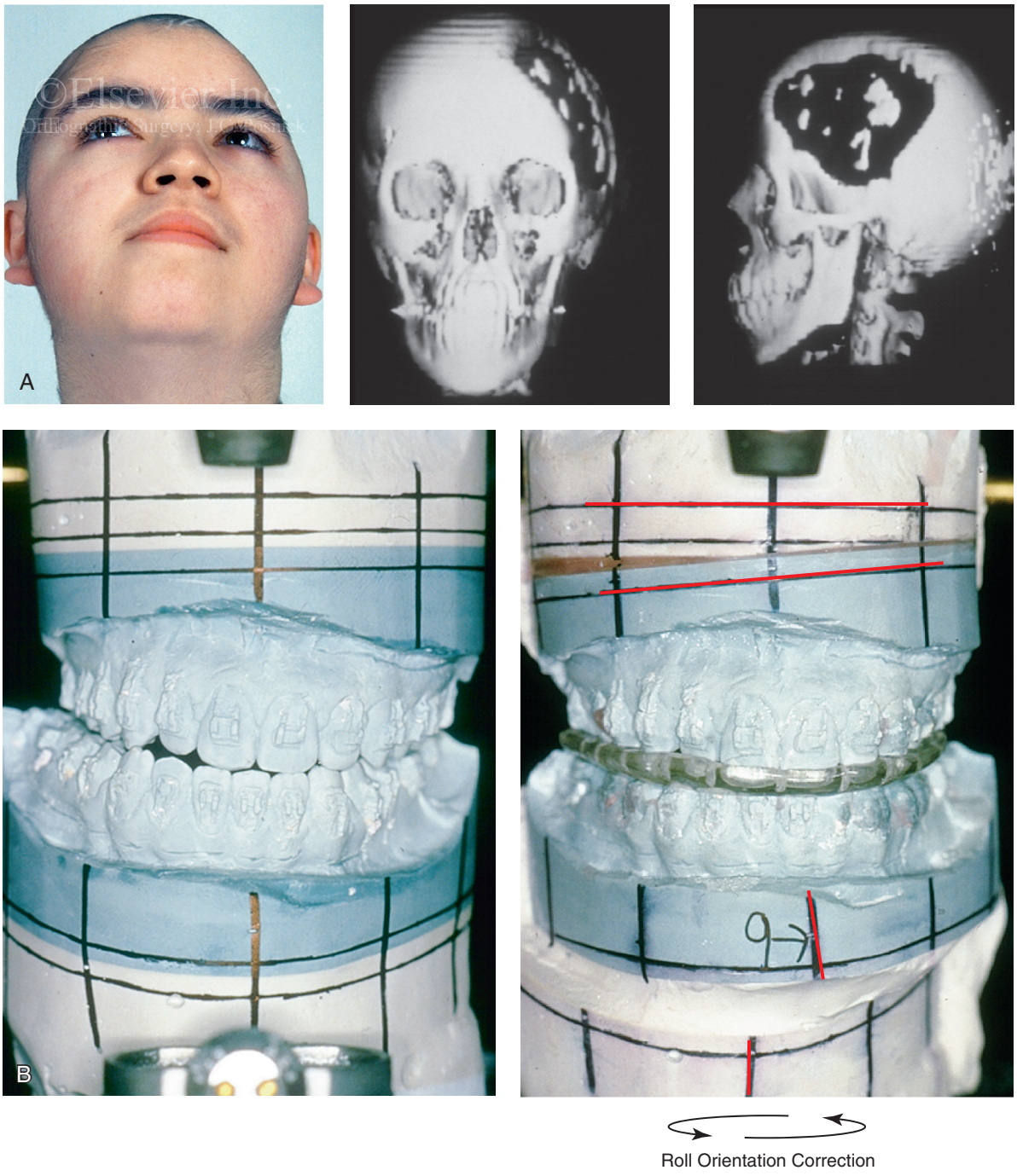
• **Figure 35-5, cont'd** **F,** Occlusal views before and 2 years after reconstruction. **G,** Panorex radiographs before and after reconstruction. **H,** Anteroposterior cephalometric radiographs before and after reconstruction.

• **Figure 35-6** A 15-year-old boy who had sustained a fracture of the right condyle of the mandible during early childhood presented with facial asymmetry that involved the maxilla and the mandible. He underwent a comprehensive orthodontic and surgical approach that included a Le Fort I osteotomy; bilateral sagittal split ramus osteotomies; and an osseous genioplasty. **A,** Frontal view in repose before and 2 years after reconstruction. **B,** Frontal view with smile before and 2 years after reconstruction. **C,** Profile view before and 2 years after reconstruction.

Continued



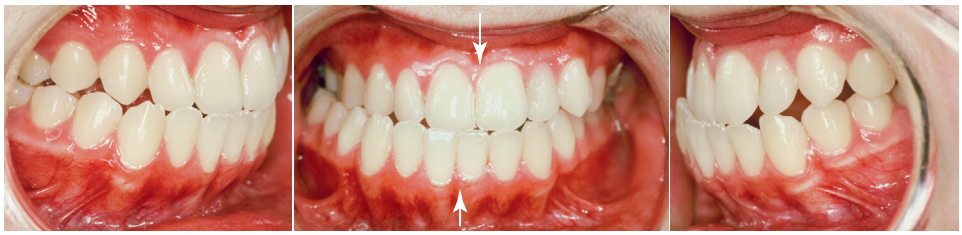
• **Figure 35-6, cont'd** **D,** Occlusal view before and 3 years after reconstruction. **E,** Longitudinal Panorex radiographs before surgery. Articulated dental casts that indicate analytic model planning. **F,** Anteroposterior cephalometric radiographs before and after reconstruction.



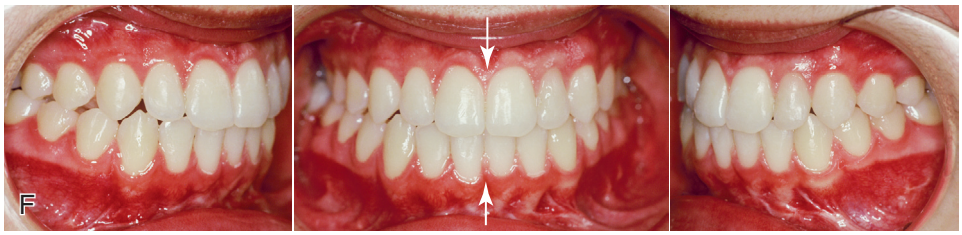
• **Figure 35-7** A 14-year-old girl sustained left cranial vault and left facial trauma. She required cranial debridement that resulted in a large, full-thickness frontotemporal skull defect. She developed a lower-face deformity that was characterized as an asymmetrical mandibular excess, likely as a result of the traumatic stimulation of the left condyle. A secondary maxillary deformity also occurred (i.e., canting). She was then referred for reconstruction. **A,** Worm's-eye facial view before reconstruction. Three-dimensional computed tomography scan views that indicate the extent of skull defect and jaw asymmetry. **B,** Articulated dental casts that indicate analytic model planning.

Continued

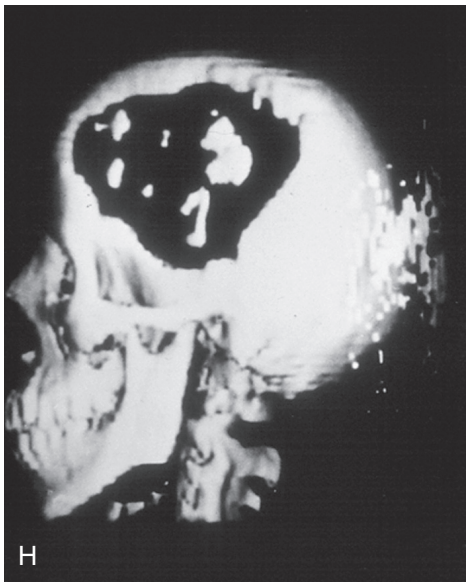
• **Figure 35-7, cont'd C,** Frontal view before and after the reconstruction of the skull defect and the orthognathic surgery that included Le Fort I osteotomy, bilateral sagittal split ramus osteotomies, and an osseous genioplasty. **D,** Right oblique views before and after jaw reconstruction. **E,** Left oblique views before and after reconstruction.

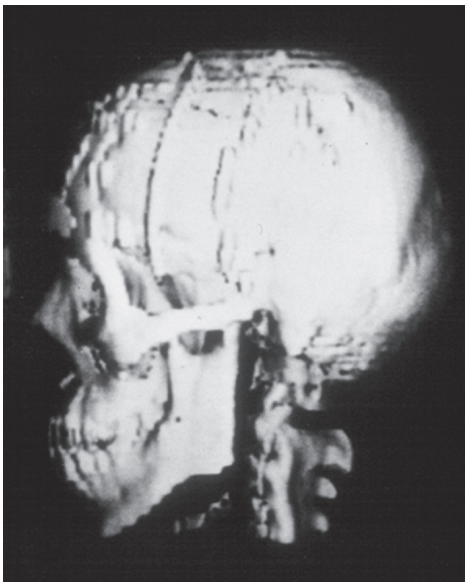


Prior to treatment

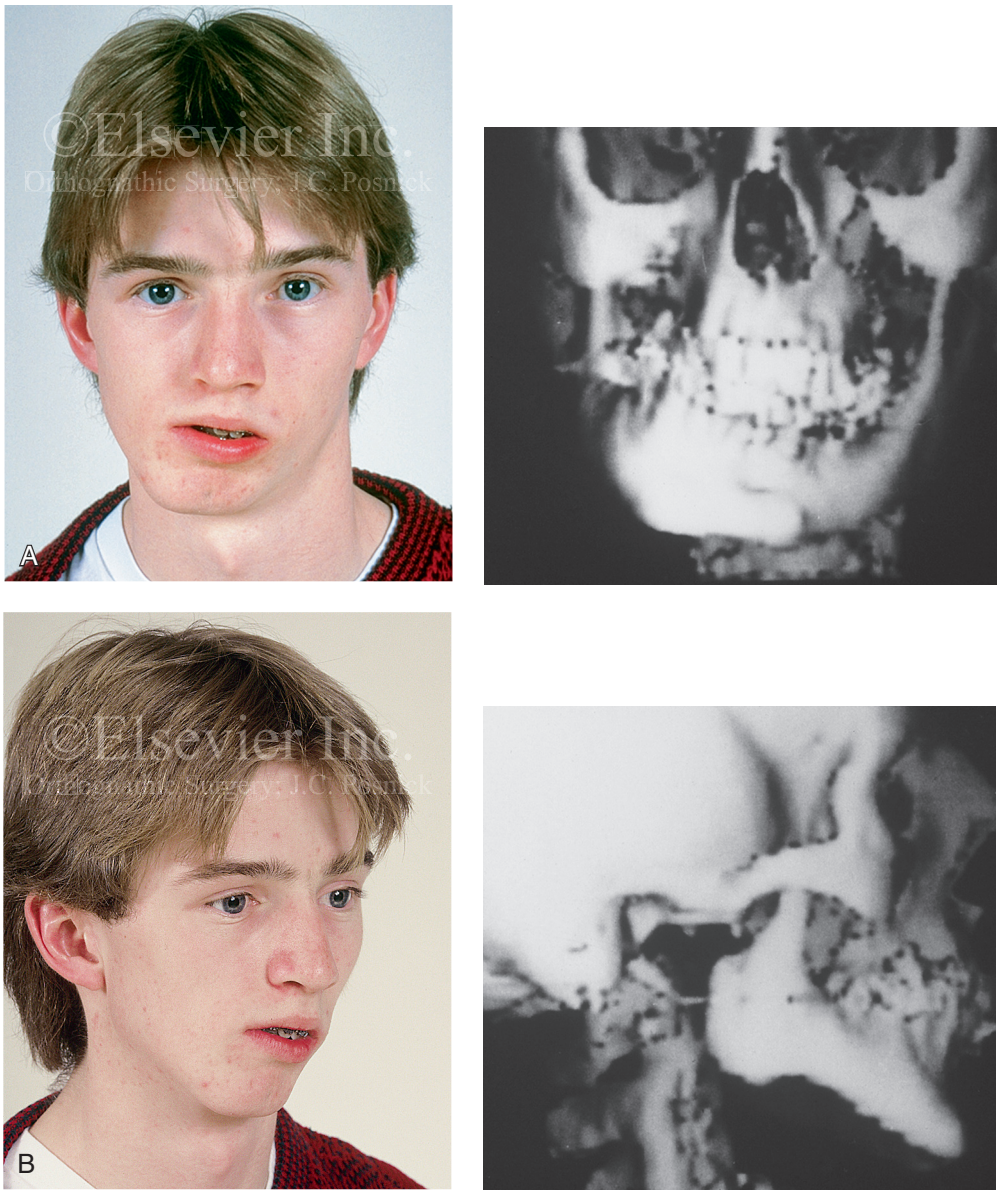


After treatment



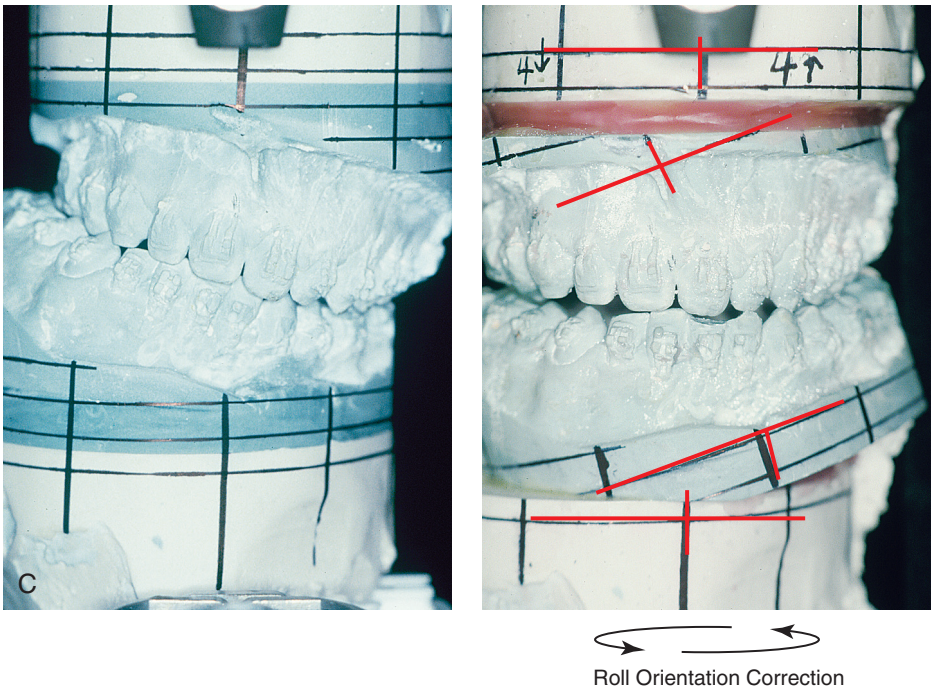


• **Figure 35-7, cont'd F,** Occlusal views before and after reconstruction. **G,** Lateral cephalometric radiographs before and after reconstruction. **H,** Three-dimensional computed tomography scan views before and after cranial vault reconstruction. *A (right), C (left), H, from Posnick JC: The role of plate and screw fixation in the management of pediatric facial fractures. In Gruss JS, Manson PM, Yaremchuk MJ, eds:* Rigid fixation of the craniomaxillofacial skeleton, *Stoneham, Mass, 1992, Butterworth-Heinemann, p 416.*



©Elsevier Inc.
Orthognathic Surgery: J.C. Posnick

• **Figure 35-8** A 16-year-old boy who had sustained a fracture of the right condyle of the mandible during early childhood presented with resorption of the condylar fragment and resulting facial asymmetry. He previously underwent several years of compensating orthodontics to neutralize the occlusion, which resulted in the loss of labial bone along the anterior mandibular teeth. He then underwent a comprehensive orthodontic and orthognathic surgical approach that included Le Fort I osteotomy, left sagittal split ramus osteotomy, reconstruction of the right condyle–ascending ramus with a costochondral graft, and osseous genioplasty. **A,** Frontal facial view in repose before surgery. The computed tomography scan demonstrates facial asymmetry that affects the maxilla and the mandible. **B,** Oblique facial view before surgery. The computed tomography scan demonstrates an absent right condyle.



• **Figure 35-8, cont'd C,** Articulated dental casts that indicate analytic model planning. **D,** Costochondral graft is inserted through coronal scalp incision. The stabilization of the graft involves an extended mini-plate and screws placed through a Risdon neck incision.

Continued

©Elsevier Inc.
Orthognathic Surgery; J.C. Posnick

• **Figure 35-8, cont'd** **E,** Frontal views in repose before and after reconstruction. **F,** Frontal views with smile before and after reconstruction.

©Elsevier Inc.
Orthognathic Surgery; J.C. Posnick

• **Figure 35-8, cont'd** **G,** Maximum vertical mouth opening before and after reconstruction. **H,** Oblique views before and after reconstruction.

Continued

• **Figure 35-8, cont'd I,** Worm's-eye views before and after reconstruction. **J,** Occlusal views before and after reconstruction. Note the recession along the labial aspects of the mandibular incisors both before and after surgery. Gingival grafting is indicated. *A (right), B (right), C, F, G, I, from Posnick JC: Management of facial fractures in children and adolescents,* Ann Plast Surg *33:442-457, 1994.*

• **Figure 35-9** When she was 16 years of age, this patient fell from a height of 6 meters and sustained maxillofacial and lower extremity trauma. Her maxillofacial injuries included a displaced left condyle fracture, a displaced right condyle fracture, a comminuted left anterior mandibular fracture with the loss of teeth, and left anterior maxillary dentoalveolar fractures. She was treated at another institution with tracheostomy, open reduction and internal fixation of the comminuted anterior mandibular fractures, debridement of the anterior maxilla and the associated teeth, and closed reduction of the bilateral condyle fractures. The lower extremity fractures required the placement of left and right femoral rods and open reduction and internal fixation of the right ankle and foot fractures. The patient underwent several months of intermaxillary fixation. With the release of the fixation, radiographs confirmed bilateral temporomandibular joint (TMJ) ankylosis. One year later, the patient was taken to the operating room for the removal of the internal fixation of the left anterior mandible.

She was referred to this surgeon when she was 20 years old for secondary reconstruction. She was found to have bilateral TMJ ankylosis, malunion and displacement of the mandible, anterior maxillary dentoalveolar defects, the loss of multiple teeth in both arches, and impacted wisdom teeth. She underwent a comprehensive assessment that also included the evaluation of her dental rehabilitative needs. An orthodontist and a prosthodontist were consulted, and perioperative orthodontic treatment was instituted. **A,** Frontal views in repose before and after reconstruction and dental rehabilitation. **B,** Frontal views with smile before and after reconstruction and dental rehabilitation.

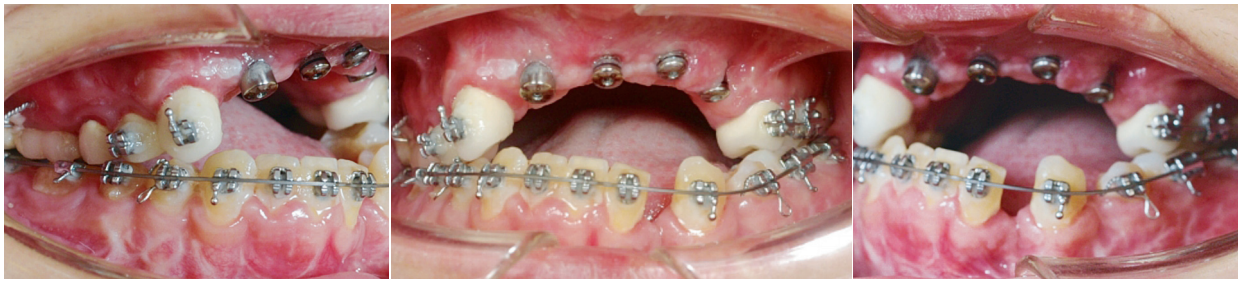
Continued

• **Figure 35-9, cont'd** Staged maxillofacial reconstruction included the following:

1. Left and right TMJ ankylosis release, including left and right coronoidectomies with the immediate improved mandibular range of motion
2. Six months later, maxillomandibular reconstruction that included:
   - Bilateral sagittal split ramus osteotomies
   - Harvesting of anterior iliac crest graft
   - Oblique osteotomy of the chin
   - Reconstruction of the anterior maxillary defect with autogenous corticocancellous bloc graft
   - Revision of the tracheostomy scar
3. Six months later, a left parasymphyseal osteotomy and a right sagittal split ramus osteotomy to further improve the mandibular arch form

Six months after successful bloc grafting to the anterior maxilla, four dental implants were placed. Six months after successful implant placement, restorative dentistry was carried out that included crown and bridge work in both arches. **C,** Profile facial views before and after reconstruction and dental rehabilitation. **D,** Oblique facial views before and after reconstruction and dental rehabilitation.

Prior to secondary reconstruction



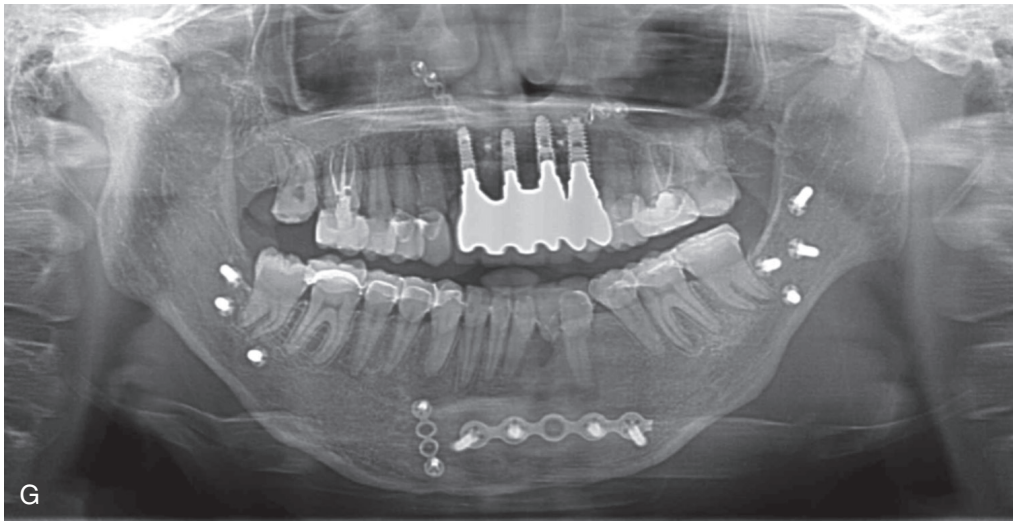
After first stage reconstruction

After reconstruction and dental rehabilitation

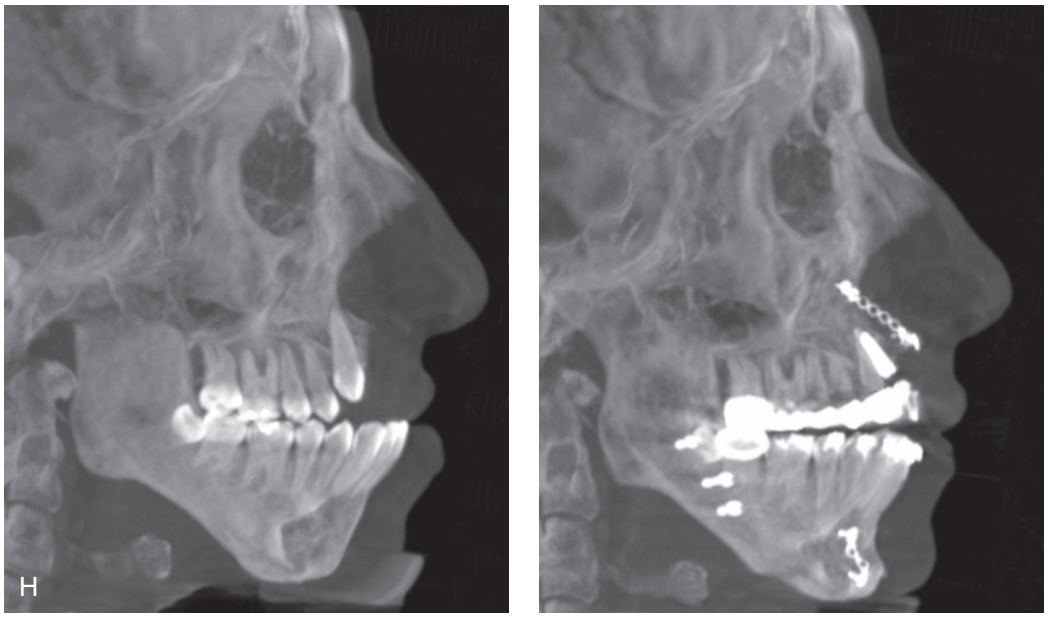
• **Figure 35-9, cont'd E,** Occlusal views before secondary reconstruction; after TMJ ankylosis release, bone grafting of the anterior maxilla, and the placement of implants; and then at the completion of dental rehabilitation. **F,** Craniofacial computed tomography scan views before reconstruction with demonstrated bilateral TMJ ankylosis.

Continued

After TMJ ankylosis release



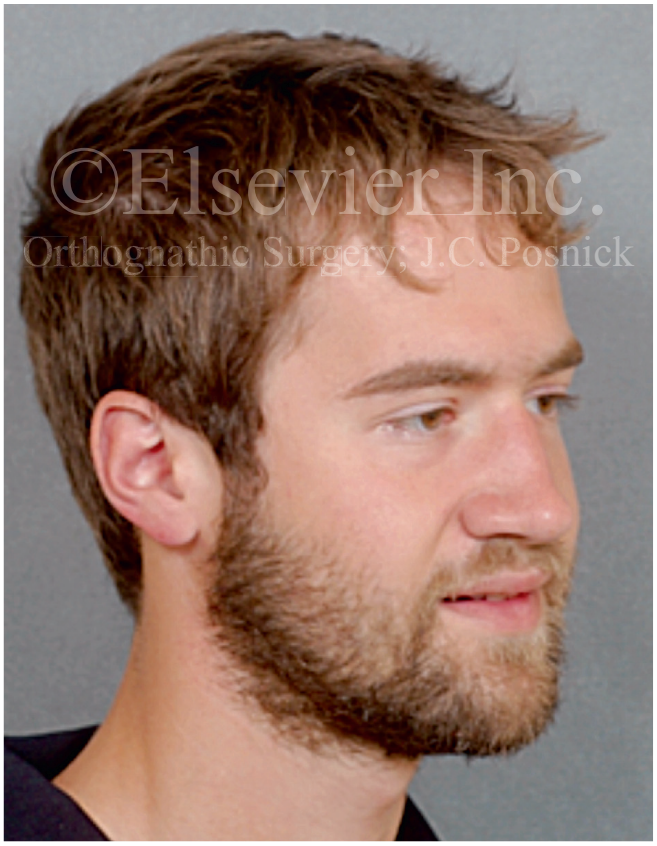
After reconstruction/dental rehabilitation

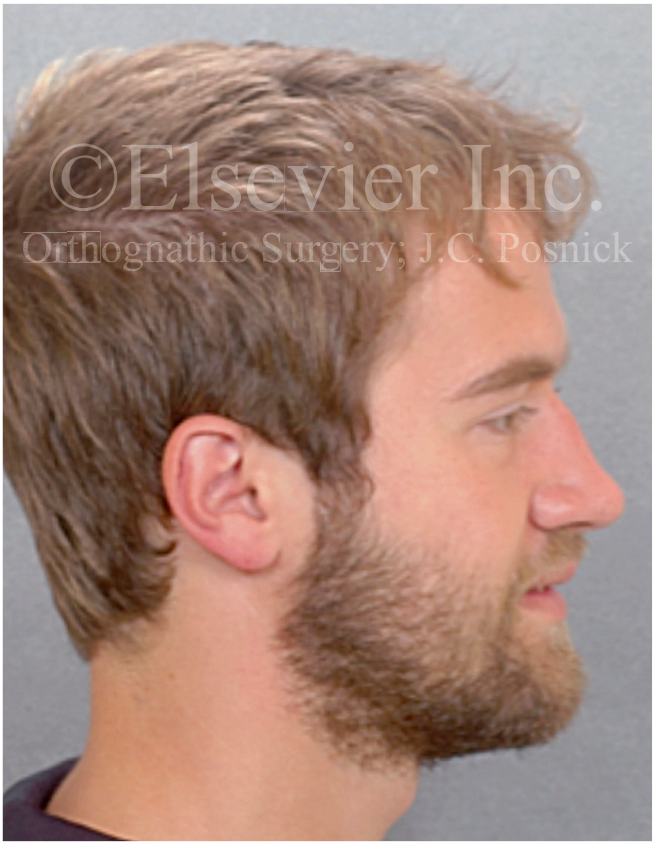


• **Figure 35-9, cont'd G,** Panorex radiographs after ankylosis release and then after the completion of reconstruction and dental rehabilitation. **H,** Lateral cephalometric radiographs before and after ankylosis relapse, bone grafting, and mandibular osteotomies.

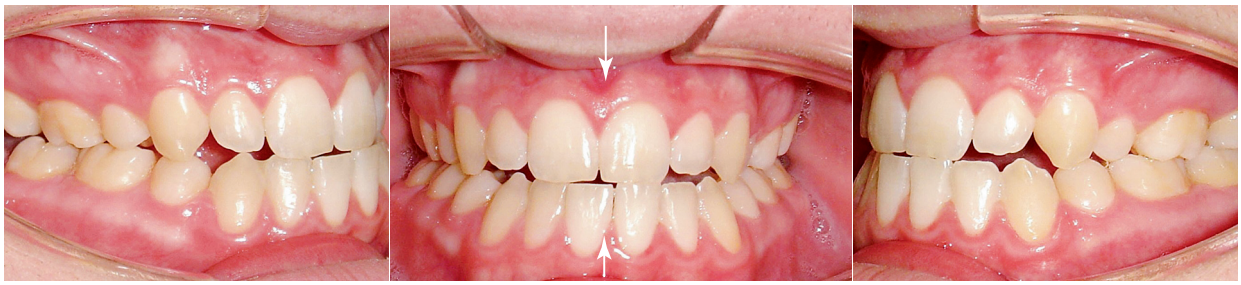
• **Figure 35-10** A recent college graduate was referred by his orthodontist for a surgical consult. He was known to have a developmental jaw deformity that was characterized as an asymmetric mandibular excess growth pattern in combination with maxillary deficiency. He had undergone orthodontic mechanics earlier during his life in an attempt to modify growth and then to straighten the teeth; this included four bicuspid extractions. An asymmetric Angle Class III negative overjet malocclusion remained. The patient had a lifelong history of chronic obstructive nasal breathing that was unresponsive to nasal sprays. At this time, he made a personal decision to proceed with orthognathic surgical reconstruction. Just 1 week after the surgical consult, he was assaulted while walking on a city street. Maxillofacial injuries included a displaced right parasymphyseal fracture and a displaced left condyle fracture. **A,** Facial views in repose before and after reconstruction. **B,** Facial views with smile before and after reconstruction.

Continued

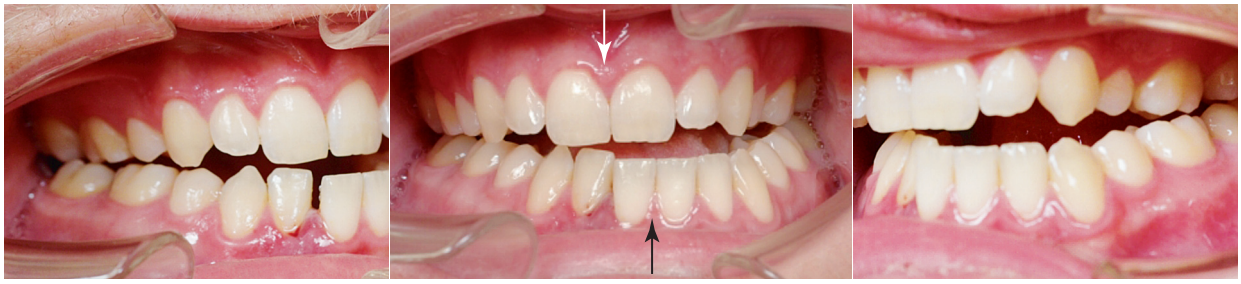




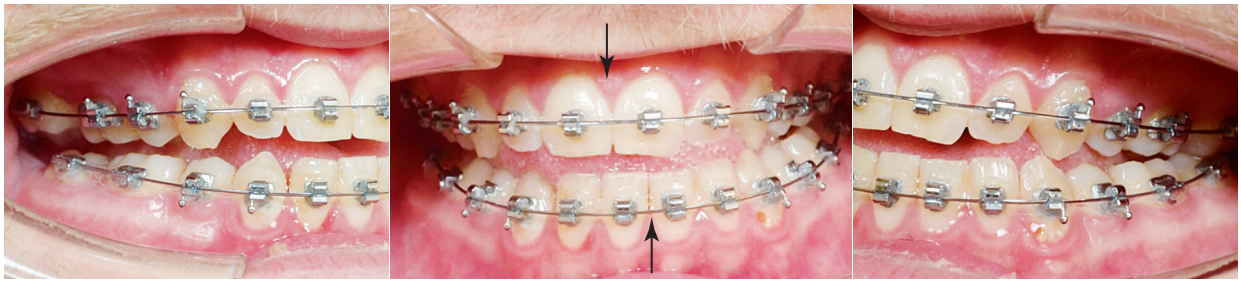
• **Figure 35-10, cont'd** This further altered the occlusion and involved a shift of the mandible to the left and a step-off between the right central and lateral incisors. The fractures were treated with the placement of surgical arch wires; open reduction and internal fixation of the right parasymphyseal fracture; and closed treatment of the left condyle fracture. Four weeks later, the patient was converted to orthodontic appliances, and orthodontic treatment continued in preparation for jaw reconstruction. Six months after the maxillofacial injuries and with orthodontic preparation complete, he underwent definitive jaw and intranasal surgery to improve the airway, occlusion, and facial morphology. The patient's procedures included maxillary Le Fort I osteotomy (vertical lengthening and horizontal advancement); bilateral sagittal split osteotomies of the mandible (lengthening of the left posterior facial height and horizontal advancement); septoplasty; and the reduction of the inferior turbinates. Orthodontic maintenance and detailing continued until the appliances were removed 6 months after surgery. **C,** Oblique facial views before and after reconstruction. **D,** Profile views before and after reconstruction.



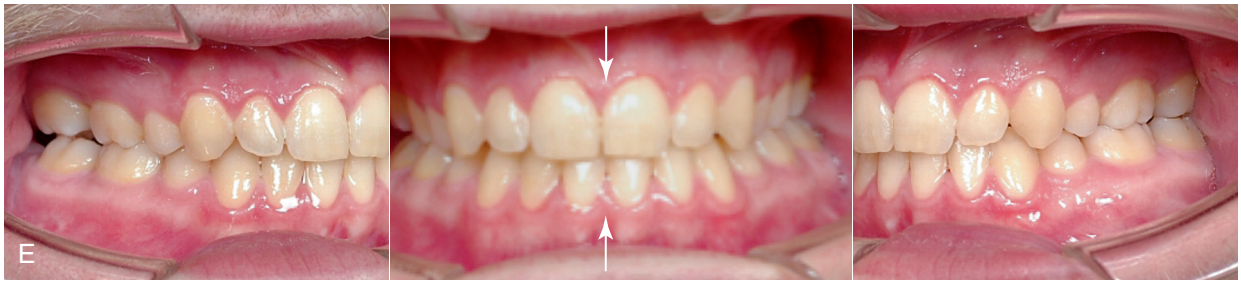
After compensating orthodontic treatment as a teenager including four-bicuspid extractions



Prior to treatment of mandibular fractures



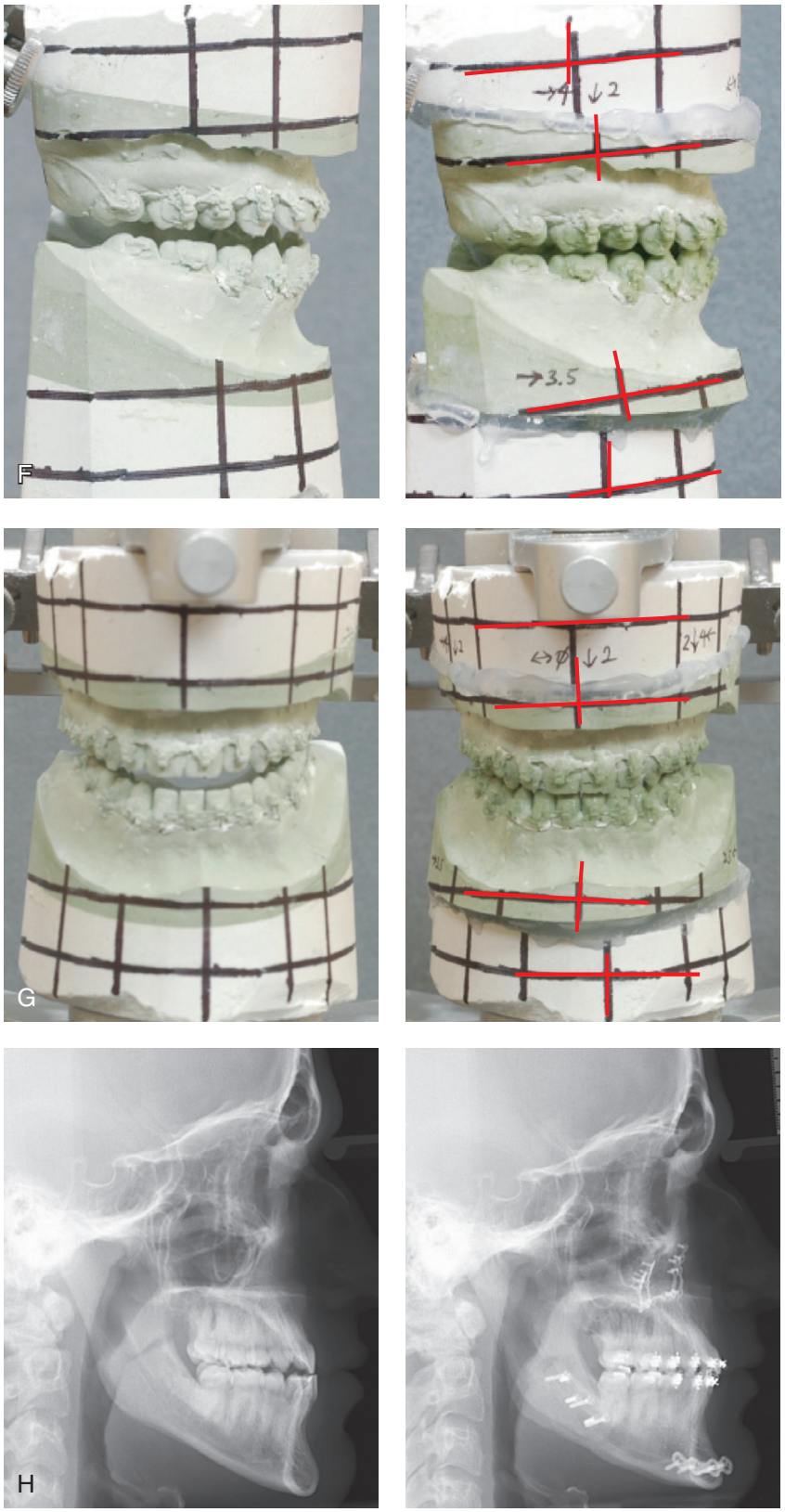
After primary fracture treatment with orthodontics in progress, prior to reconstruction



After reconstruction

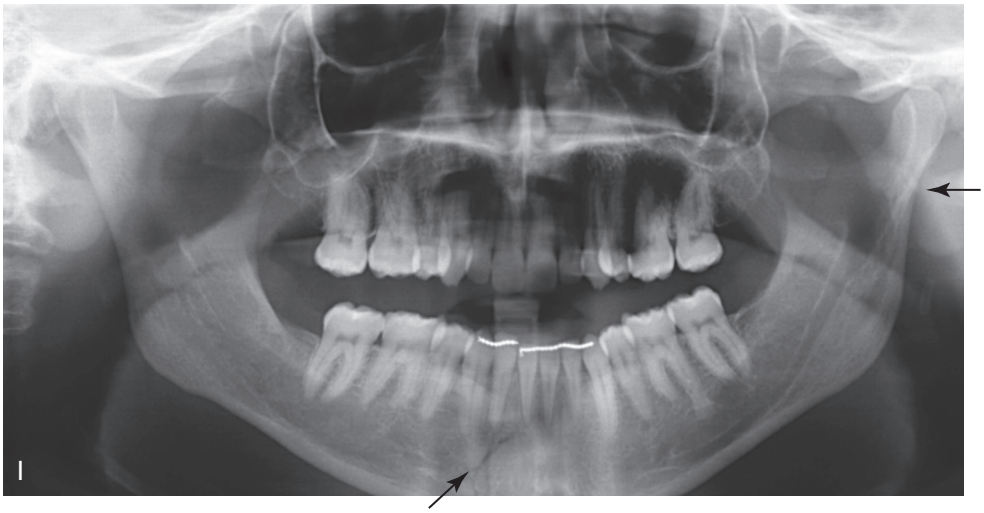
• **Figure 35-10, cont'd E,** Occlusal views before retreatment, after the mandibular fractures, after primary fracture treatment with orthodontics in progress, and after the completion of the reconstruction.

Continued



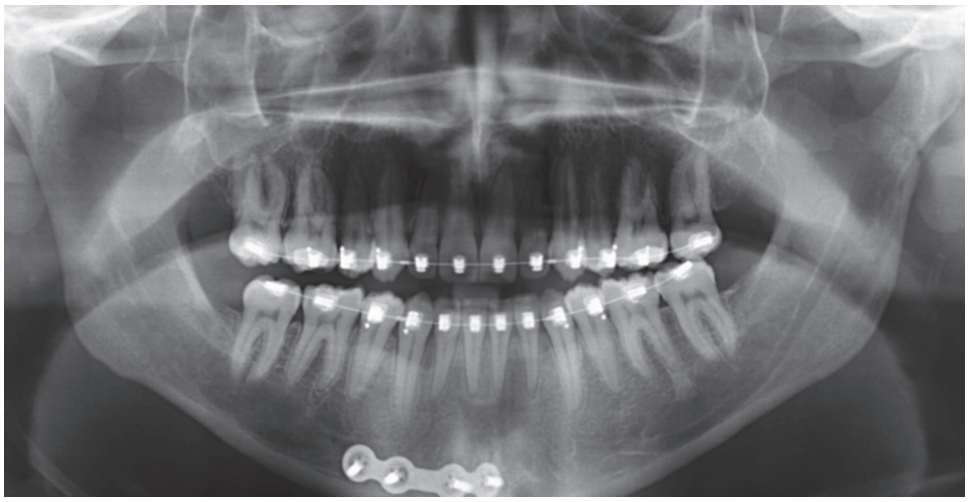
• **Figure 35-10, cont'd F** and **G,** Articulated dental casts that indicate analytic model planning. **H,** Lateral cephalometric radiographs before and after reconstruction.

Prior to retreatment (after four-bicuspid extractions). Prior to mandibular fractures

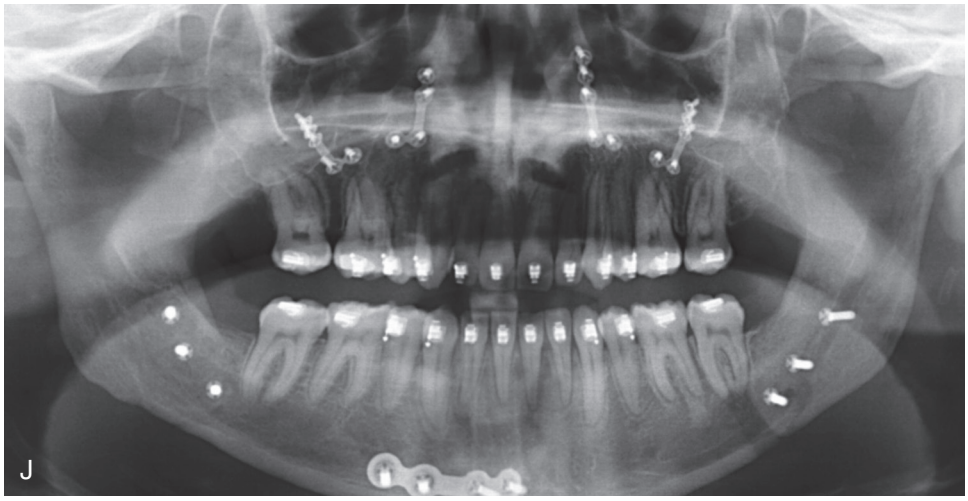


• **Figure 35-10, cont'd I** and **J,** Panorex radiographs taken before any surgery; after the mandibular fractures were sustained; after treatment of the mandibular fractures but before orthognathic surgery; and after orthognathic surgery.

Continued



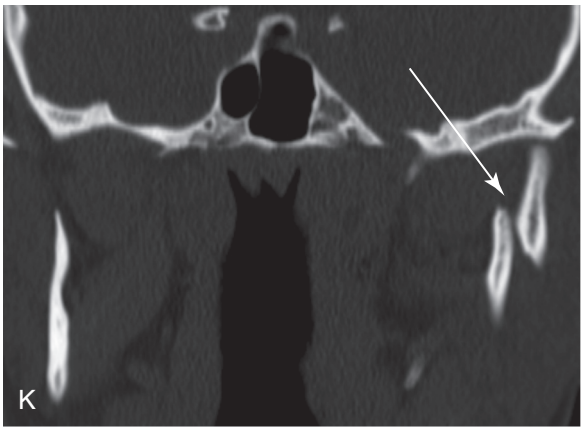
After ORIF right parasymphyseal fracture and closed treatment left condyle fracture

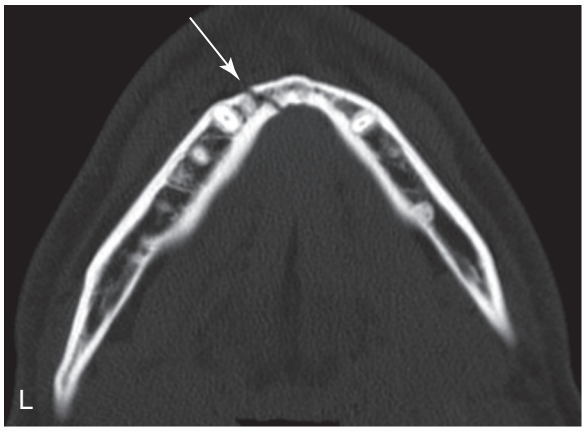


After definitive reconstruction

Displaced condyle fracture

Parasymphyseal fracture





• **Figure 35-10, cont'd K** and **L,** Computed tomography scans that indicate the locations of the mandibular fractures (left condyle and right parasymphysis) before treatment.

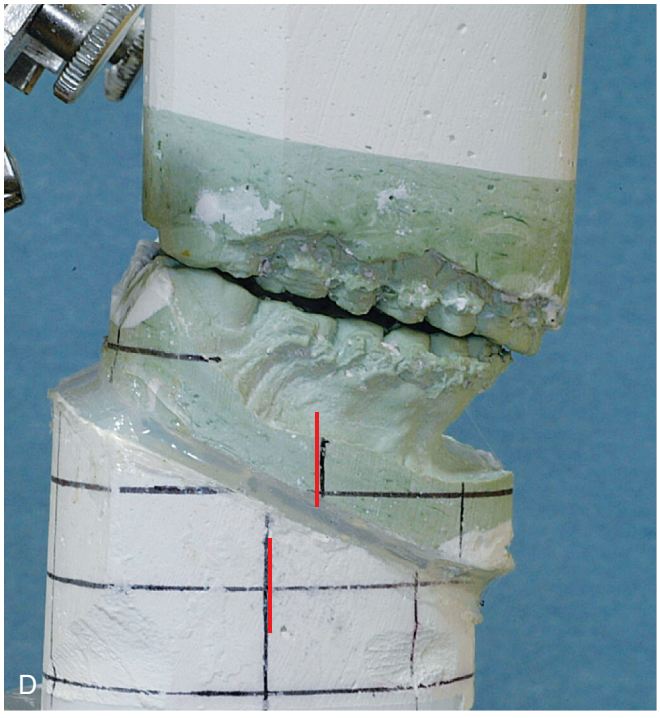
Chapter 28).[4,9,51,54,55,72,75,97,107] If the initial fracture occurred during childhood and was followed by condylar resorption, then additional secondary deformities of the maxilla and chin will have also occurred during growth. In addition to reconstruction of the ipsilateral mandible with a costochondral graft, surgical correction of the maxilla above and the chin below is often required. This includes use of Le Fort I osteotomy (primarily for cant and dental midline correction); contralateral sagittal split ramus osteotomy (asymmetry correction); and osseous genioplasty[7] (see Fig. 35-8).

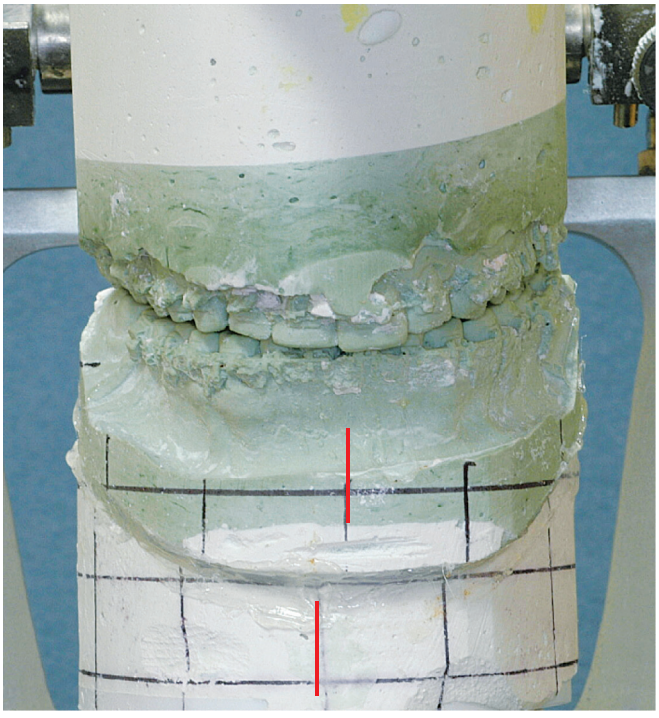
When a displaced condylar fracture occurs in an adult and healing involves the loss of the ipsilateral posterior facial height, a predictable shift in the occlusion is also expected (Figs. 35-10 through 35-15). Depending on the individual's masticatory musculature and condylar (TMJ) adaptations, a significant centric relation–centric occlusion discrepancy may have occurred.[52,130,134] This can result in difficulty with chewing, swallowing, speech, secondary occlusal trauma, symptomatic TMD, and significant facial asymmetry. If so—and assuming satisfactory mandibular mobility (i.e., vertical mouth opening)—standard sagittal split ramus osteotomies can generally be carried out to reestablish the following: 1) the ipsilateral posterior facial height; 2) a consistent preinjury occlusion; 3) the restoration of facial

Text continued on p. 1521

• **Figure 35-11** A woman in her late 20s was in a bicycle accident and sustained a displaced right condylar fracture and anterior maxillary dentoalveolar trauma. She underwent open reduction and internal fixation of the right condyle fracture with plate and screw fixation. There was partial necrosis of the proximal (condylar) segment with a loss of posterior facial height. This resulted in a shift of the mandible on the ipsilateral side. There was a significant centric relation–centric occlusion discrepancy; the patient had difficulty chewing, symptomatic temporomandibular disorder, and facial asymmetry. She was referred to this surgeon and underwent perioperative orthodontics, bilateral sagittal split ramus osteotomies, and an osseous genioplasty. **A,** Frontal facial and occlusal views before reconstruction. **B,** Panorex radiograph before reconstruction.

Continued





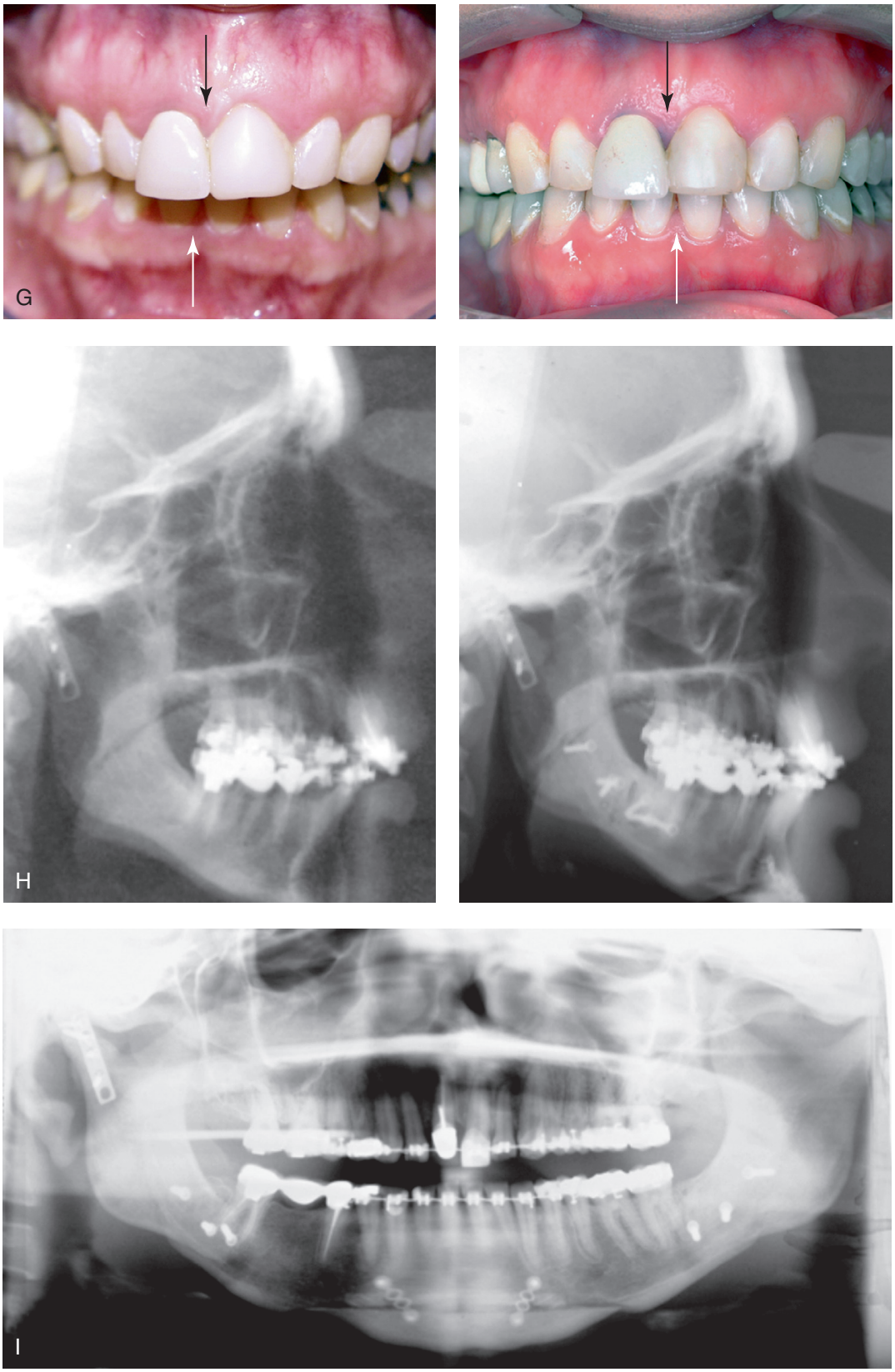
©Elsevier Inc.
Orthognathic Surgery; J.C. Posnick

• **Figure 35-11, cont'd C,** Profile and cephalometric views before reconstruction. **D,** Articulated dental casts that indicate analytic model planning.

©Elsevier Inc.
Orthognathic Surgery; J.C. Posnick

• **Figure 35-11, cont'd E,** Frontal views before and after reconstruction. **F,** Oblique views before and after reconstruction.

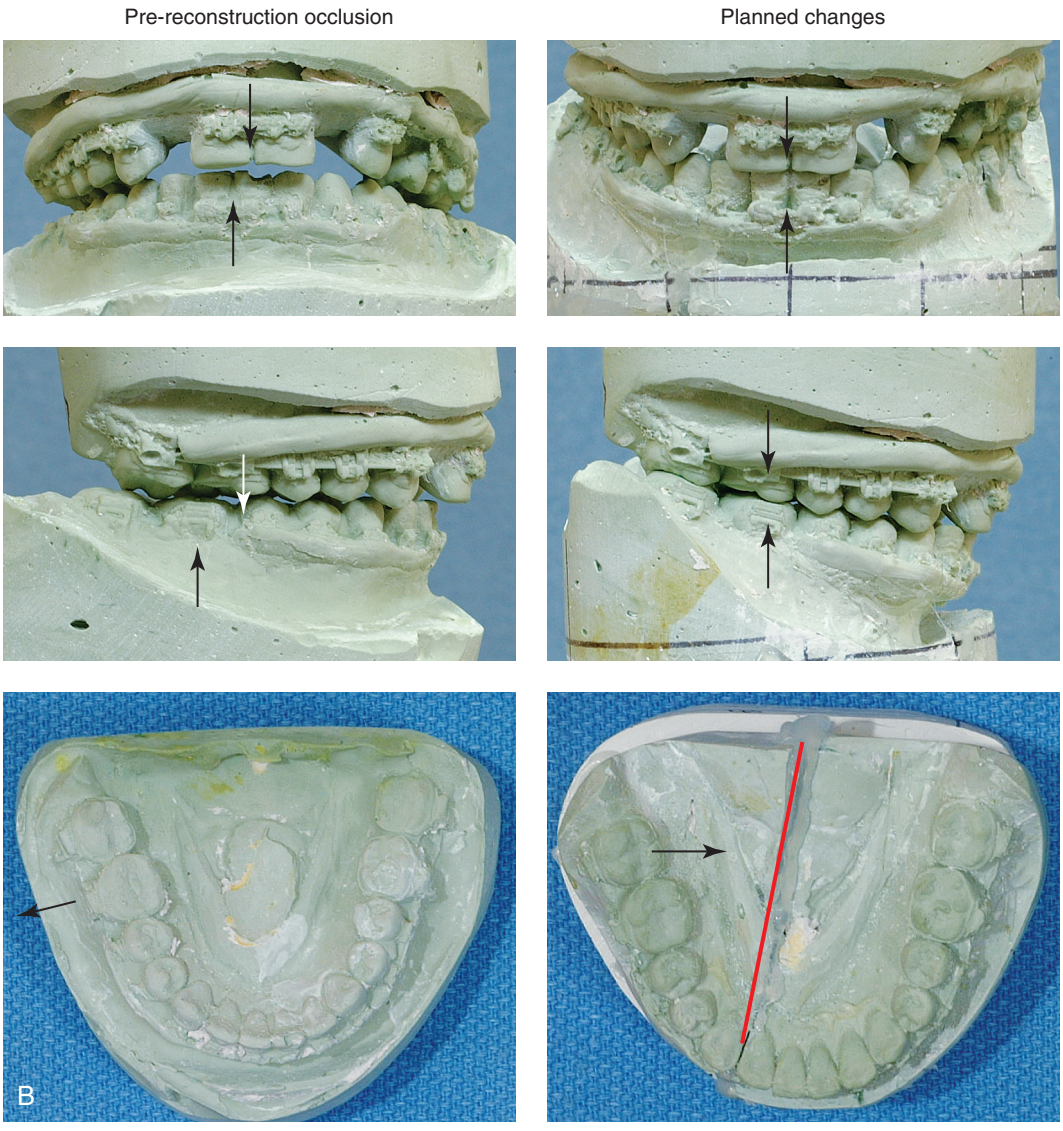
Continued



• **Figure 35-11, cont'd G,** Occlusal views before and after reconstruction. **H,** Lateral cephalometric views before and after reconstruction. **I,** Panorex radiograph after reconstruction.

• **Figure 35-12** A woman in her late 20s was a pedestrian who was struck by a motor vehicle. She sustained a right condylar fracture, a right parasymphyseal fracture, and maxillary dentoalveolar trauma. Primary fracture management included open reduction and internal fixation of the right condyle fracture, closed reduction of the right parasymphyseal fracture, and closed reduction of the dentoalveolar injury. She arrived to this surgeon for the evaluation of secondary deformities that included the partial resorption of the condylar segment, malunion of the parasymphyseal fracture (widening), and the loss of maxillary dentoalveolar components. Comprehensive evaluation included additional consultations with an orthodontist, a periodontist, and a restorative dentist. Surgery included bilateral sagittal split ramus osteotomies (correction of asymmetry), right parasymphyseal osteotomy (narrowing), and bloc iliac grafting of the maxillary dentoalveolar defects. Dental implants were placed 4 months later. Restorative work was completed 6 months after implant placement. **A,** Frontal view before secondary reconstruction. The Panorex radiograph indicates the loss of condylar height. The anteroposterior cephalometric radiograph and facial views indicate mandibular width asymmetry.

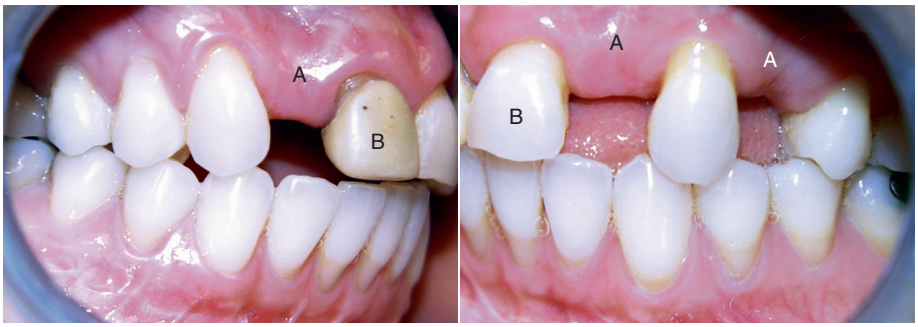
Continued



• **Figure 35-12, cont'd B,** Dental casts before and after model surgery planning.

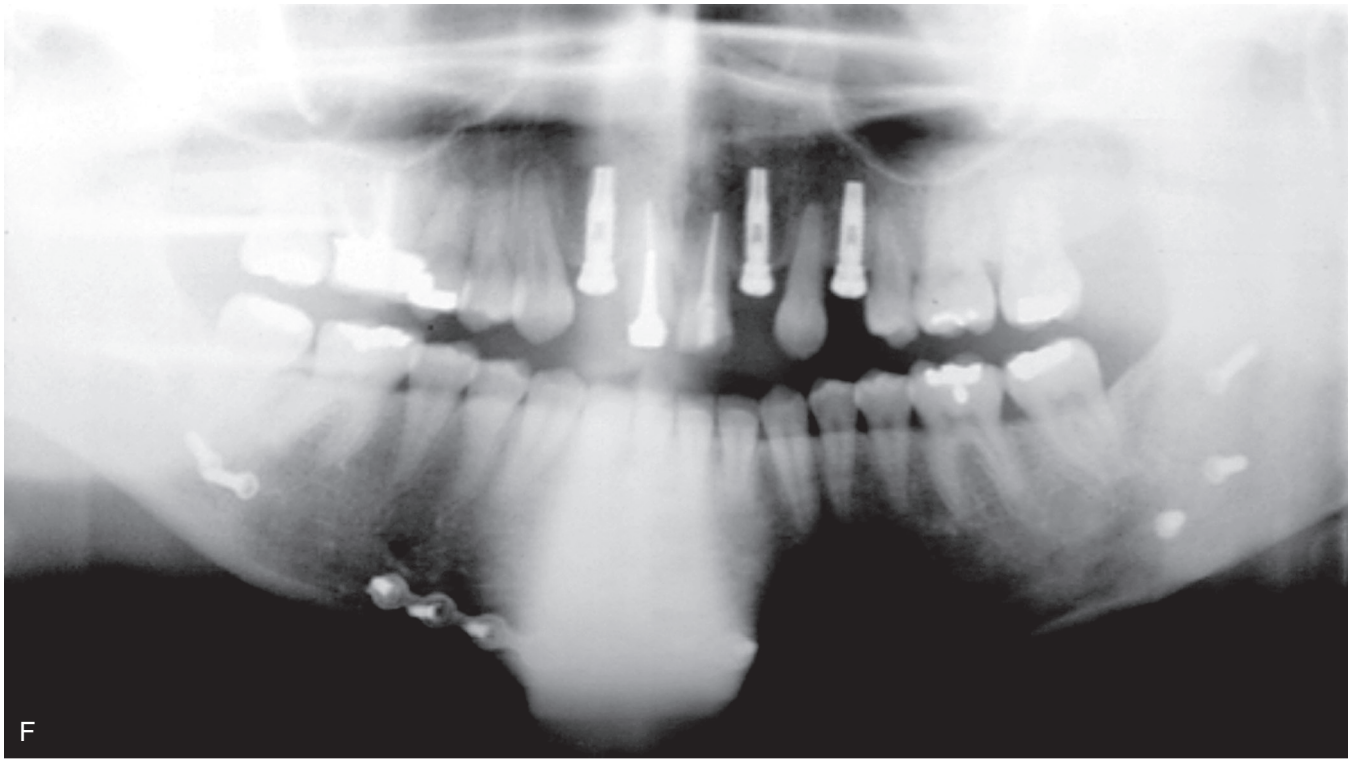
• **Figure 35-12, cont'd** **C,** Facial views before and after reconstruction and rehabilitation. **D,** Oblique views before and after reconstruction and rehabilitation.

Continued



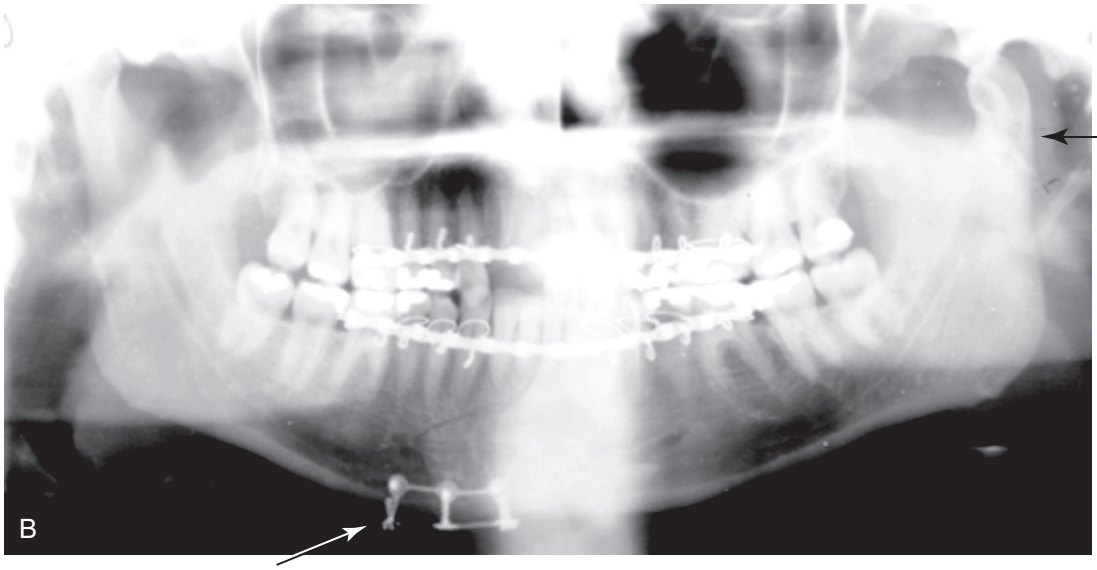
A - Bone defect requires grafting
B - Injured teeth with root canals/crowns in place

After reconstruction/dental rehabilitation



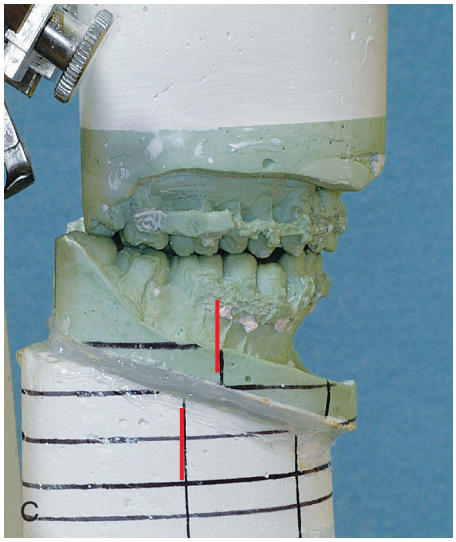
• **Figure 35-12, cont'd E,** Occlusal views before and after reconstruction and rehabilitation. **F,** Panorex radiograph after reconstruction and rehabilitation.

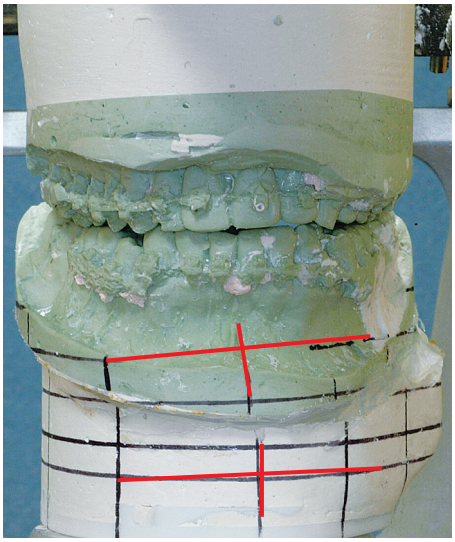
X - required extractions

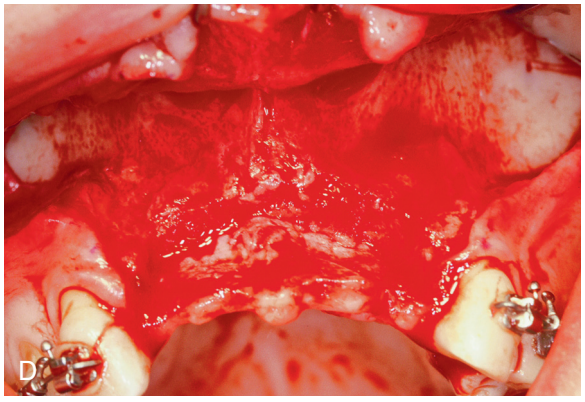


• **Figure 35-13** A woman in her 20s was in a moped accident and sustained maxillofacial trauma that included a displaced left condyle fracture, a right parasymphyseal fracture, maxillary dentoalveolar trauma, and the laceration of the upper lip. She was initially treated with closed reduction of the left condyle fracture, open reduction and internal fixation of the right parasymphyseal fracture, debridement of the right maxillary incisors, and reimplantation of the left maxillary incisors. By 3 months after treatment, malunion of the mandible resulted in malocclusion, difficulty chewing, symptomatic temporomandibular disorder, and facial asymmetry. The patient was referred to this surgeon for comprehensive evaluation included additional consultation with a periodontist, and an orthodontist. She underwent preoperative orthodontics and reconstruction that included bilateral sagittal split ramus osteotomies. A bloc corticocancellous graft was simultaneously crafted and secured to the anterior maxillary dentoalveolar defect. Four months later, four dental implants were placed. This was followed 4 months later by the placement of crowns. **A,** Frontal and occlusal views 1 week after the accident and primary fracture management. **B,** Panorex radiograph taken just after primary fracture management.

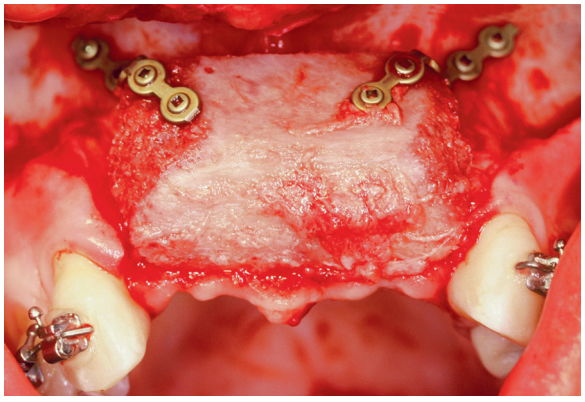
Continued





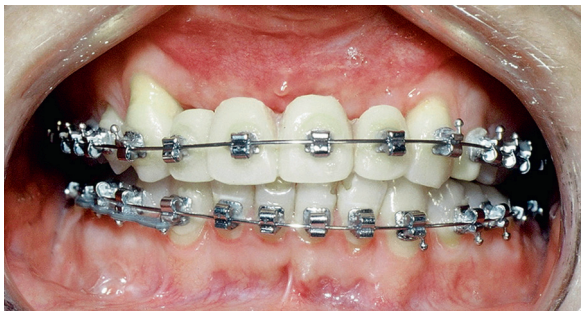


Pre-maxillary defect



Bloc iliac graft in place

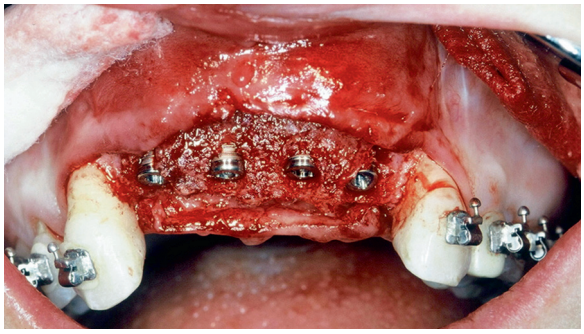
• **Figure 35-13, cont'd C,** Three months after primary fracture management, analytic model planning indicates details for the mandibular reconstruction. **D,** Intraoperative view of the maxillary dentoalveolar defect and the crafted bloc corticocancellous iliac graft fixed in place.



4 months after injury with orthodontics in progress
Prior to grafting

6 months after injury
2 months after iliac grafting and jaw reconstruction

4 months after grafting
Stint-implant placement



4 months after grafting
4 implants have just been placed

• **Figure 35-13, cont'd E,** Close-up views of the anterior maxilla indicate the sequence of events: 4 months after injury but before grafting; 6 months after injury and 2 months after grafting; 4 months after grafting with stent and implant placement; and then just after the placement of four implants. **F,** Panorex radiograph after mandibular and maxillary reconstruction and dental rehabilitation.

Continued

• **Figure 35-13, cont'd G,** Frontal views before and after reconstruction and rehabilitation. **H,** Oblique views before and after reconstruction and rehabilitation. **I,** Occlusal views before and after reconstruction and dental rehabilitation.

Centric relation (CR) bite

Centric occlusion (CO) bite

• **Figure 35-14** When he was between 13 and 15 years old, this patient underwent standard orthodontic treatment and achieved a normal occlusion without a centric relation–centric occlusion (CR-CO) discrepancy. When he was 16 years of age, he was in a motor vehicle accident and sustained injuries that included a displaced left condyle of the mandible fracture. The fracture was treated with closed treatment and intermaxillary fixation for 2 weeks followed by guiding elastics for an additional 4 weeks. The patient also sustained fracture of the left mandibular first and second molars, which required the placement of full crowns. With healing, there was a loss of posterior facial height and a shift of the mandible toward the fracture. There was a significant CR-CO slide with masticatory muscle discomfort and left temporomandibular joint noise. The patient underwent computed tomography scanning and magnetic resonance imaging of the temporomandibular joint, which indicated the following: left condylar head flattening and remodeling; the meniscus being in good alignment with a deformed condylar head; and, on the right side, the mandibular condyle and meniscus appear to be unremarkable. The patient maintained an adequate range of mandibular motion. He was being treated with a splint that prevented CR-CO slide, and his temporomandibular disorder has improved. Bilateral sagittal split osteotomies of the mandible were recommended to establish a consistent CR-CO without prematurities. **A,** Facial views 2 years after left condyle fracture with closed reduction. **B,** Intraoral views with the patient in CR bite. **C,** Intraoral views with the patient in CO bite.

©Elsevier Inc.
Orthognathic Surgery; J.C. Posnick

• **Figure 35-15** A middle-aged man had a syncopal episode and sustained a displaced right condyle fracture of the mandible and maxillary dentoalveolar trauma. This was managed with closed treatment and 2 weeks of intermaxillary fixation followed by elastics, masticatory muscle retraining, and then dental rehabilitation. A comfortable occlusion with minimal centric relation–centric occlusion discrepancy and resolution of temporomandibular disorder was possible without the need for surgical intervention. **A,** Facial and occlusal views 2 days after injury. **B,** Panorex radiograph 4 weeks after injury that indicates right subcondylar fracture and dislocation and left maxillary dentoalveolar trauma.

• **Figure 35-15, cont'd C,** Frontal view at 18 months after injury. Effective muscle retraining achieved a stable occlusion. With wide vertical opening, there is a shift of the chin to the right. The centric occlusion view is shown 18 months after injury with successful dental rehabilitation.

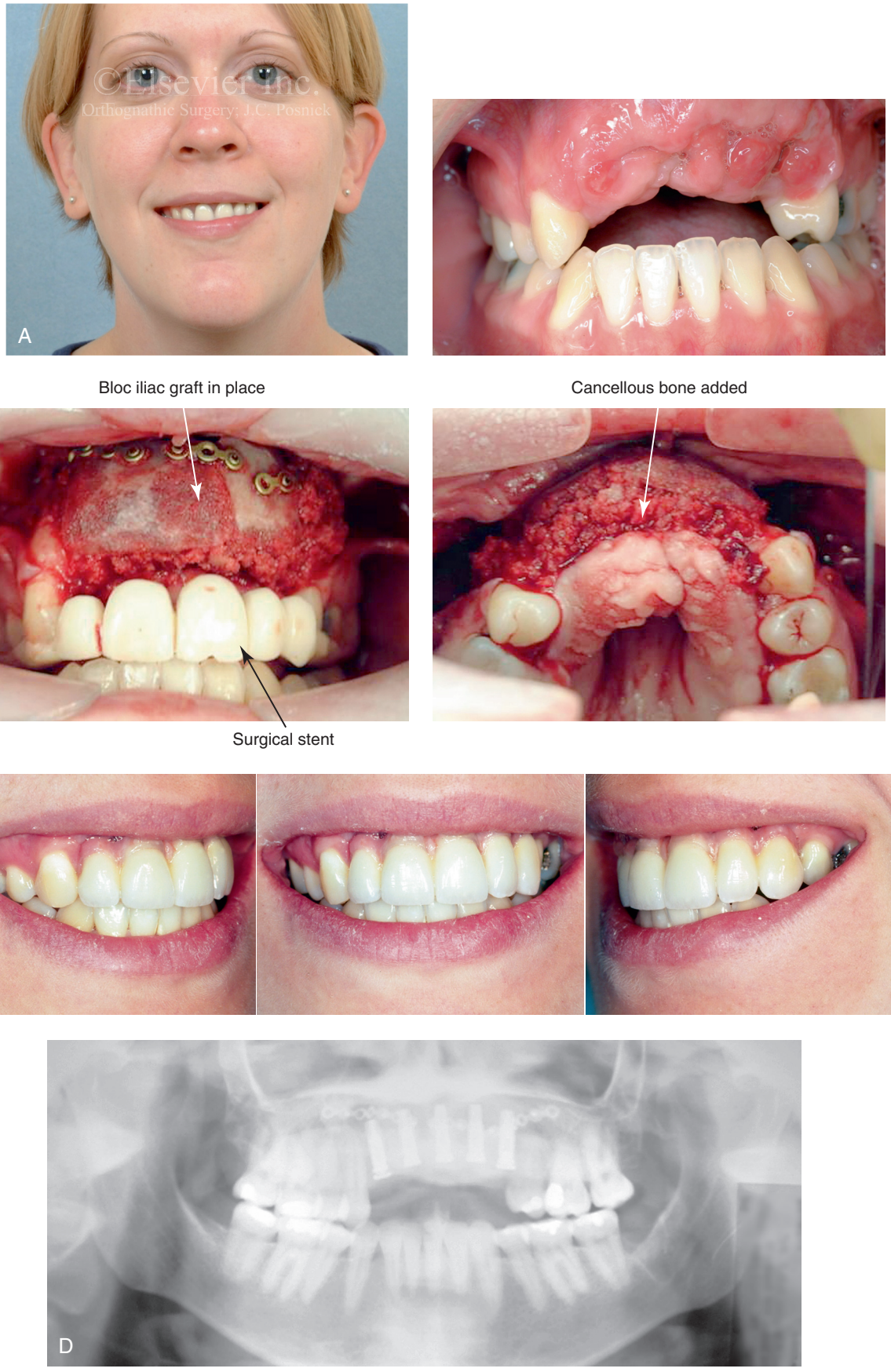
symmetry; and 4) the relief of TMD (see Chapter 4 and Figs. 35-10 through 35-15). In most cases, after the individual sustains a displaced condyle fracture, the combination of condylar remodeling and masticatory muscle adaptation will result in a manageable occlusion and an adequate range of motion without the need for secondary reconstruction (see Fig. 35-15).

Posttraumatic Segmental Dentoalveolar Defects

A frequently seen pattern of posttraumatic secondary deformity occurs after the avulsion or devascularization of an anterior dentoalveolar segment (i.e., the maxilla, the mandible, or both).[39] This may occur as an isolated injury or in conjunction with other jaw fractures (Figs. 35-16 and 35-17; see also Figs. 35-4, 35-9, 35-12, and 35-13). To secondarily correct this segmental dentoalveolar deformity with a loss of teeth, an autogenous corticocancellous iliac bone graft fixed in place with microplates and screws is generally a reliable option (see Figs. 35-4, 35-9, 35-12, 35-13, 35-16, and 35-17). This is followed by a soft-tissue vestibuloplasty and a keratinized mucosa grafting procedure, when required. Approximately 4 months after successful bone grafting and soft tissue re-arrangment, dental implants are placed. This is followed several months later with crown restorations. Comprehensive skeletal reconstruction and dental rehabilitation of segmental dentoalveolar defects generally involves a surgeon, a periodontist, and a restorative dentist and is best coordinated from the onset.[87]

Conclusions

Despite every attempt to ensure the optimal primary treatment of craniomaxillofacial fractures, a percentage of individuals present with residual facial deformities and head and neck dysfunction that require secondary reconstruction. Specific patterns of secondary maxillofacial deformities are frequently seen, including TMJ ankylosis, saddle-nose

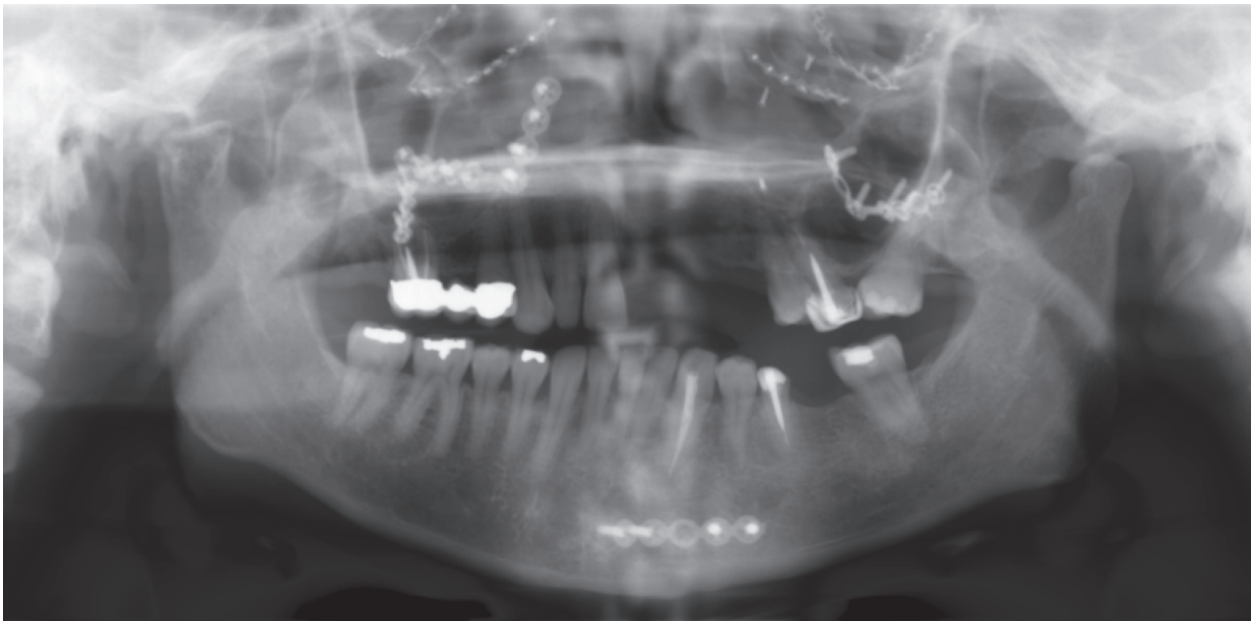


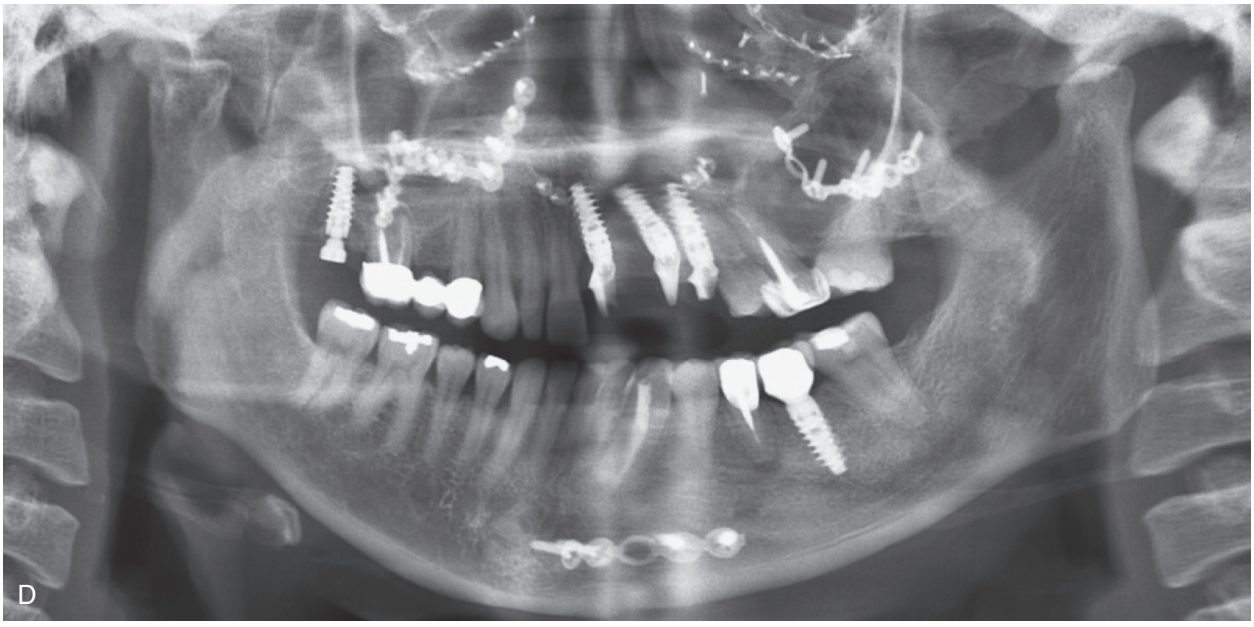
• **Figure 35-16** A 28-year-old woman was in a motor vehicle accident and sustained maxillary dentoalveolar trauma with a loss of five anterior teeth and associated alveolar bone. She had previously undergone orthodontic treatment including 4 bicuspid extractions to close an anterior open bite. **A,** Frontal view with temporary partial denture in place. Occlusal view with the removal of the partial denture indicates the extent of dentoalveolar injury and bone loss. **B,** Intraoperative view with a crafted corticocancellous iliac graft fixed in place. A prefabricated stent was used to assist with graft positioning and contouring. **C,** Occlusal views after successful grafting, placement of 5 implants and then dental restorative work. **D,** Panorex radiograph after successful grafting and the placement of five dental implants.

• **Figure 35-17** A 21-year-old woman was in a motor vehicle accident and sustained naso–orbital–ethmoid fractures, left anterior maxillary dentoalveolar trauma with a loss of three teeth, and associated alveolar bone and left posterior mandibular dental injuries. She was initially treated at another institution. She was referred to this surgeon 6 months later, with left and right enophthalmus, saddle-nose deformity, and right and left medial canthal drift. She underwent left and right orbital reconstruction, left and right medial canthopexies, and the correction of the saddle-nose deformity with a full-thickness cranial graft (radix to tip) through a coronal scalp incision. She was treated by her local dentist with a removable maxillary partial denture. She returned to this surgeon for further maxillofacial evaluation 14 years later in the hopes of achieving improved dental rehabilitation. Examination confirmed left anterior maxillary dentoalveolar defect, left posterior mandibular absent second bicuspid and first molar, and several teeth with root canals. She agreed to a comprehensive dental rehabilitation approach. Additional evaluation by a periodontist, and a prosthodontist, was carried out. The patient also complained of chronic obstructed nasal breathing. Intranasal examination confirmed septal deviation and enlarged inferior turbinates. She underwent periodontal treatment followed by temporization of the dentition. Surgical procedures included septoplasty; inferior turbinate reduction; and reconstruction of the maxillary dentoalveolar defect with an autogenous corticocancellous iliac bloc graft. Four months later, three dental implants were placed in the anterior maxillary region. She also underwent implant placement in the left posterior mandible and the right posterior maxilla. Six months after implant placement, definitive crown placement and bridgework were initiated. **A,** Frontal views with smile before and after comprehensive secondary jaw and dental rehabilitation.

Continued

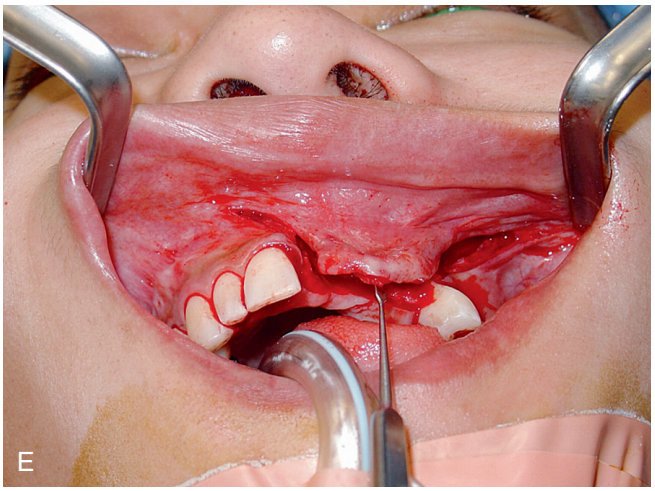
• **Figure 35-17, cont'd B** and **C,** Occlusal and palatal views before grafting and then after grafting, implant placement, and dental restorative work.



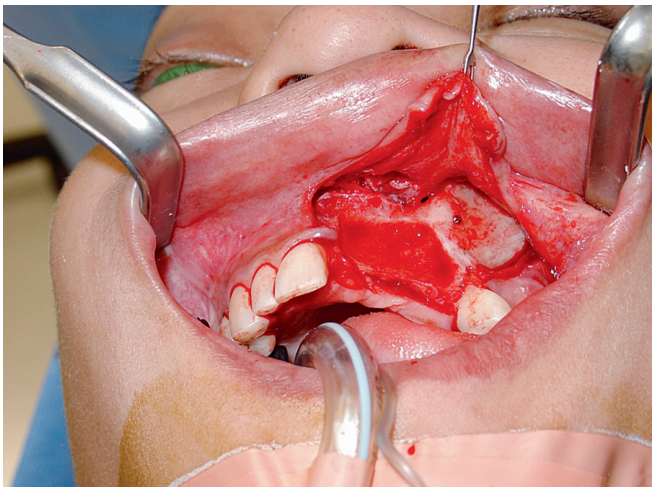


• **Figure 35-17, cont'd D,** Panorex radiographs before and then after successful secondary jaw and dental rehabilitation including grafting and the placement of dental implants.

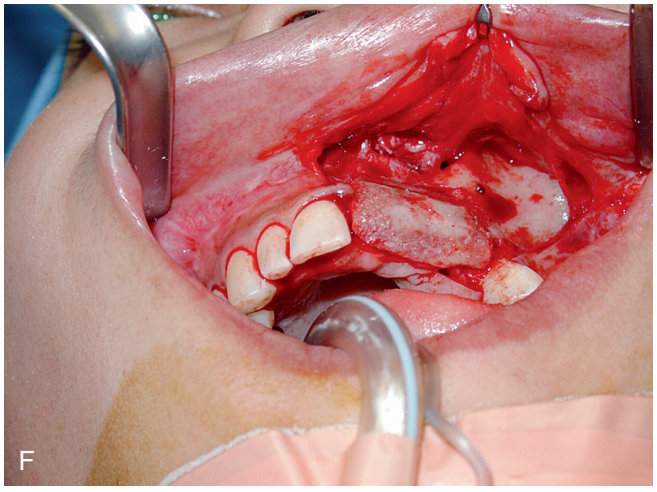
Continued



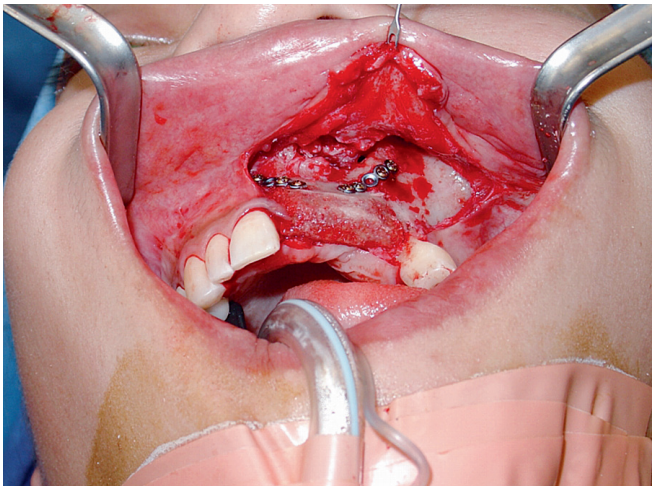
Labial flap elevated



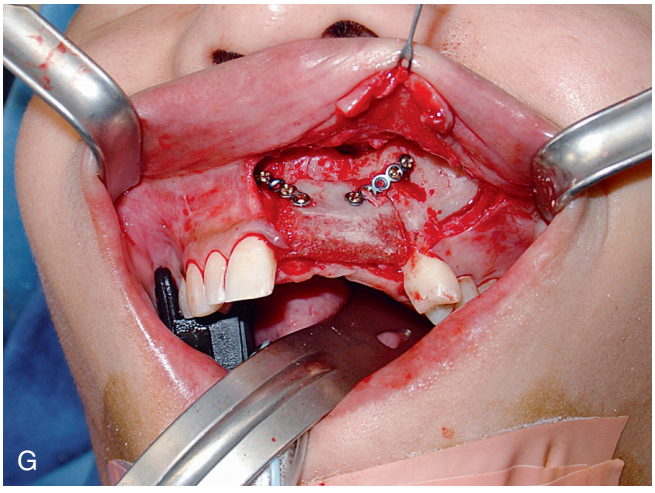
Dento-alveolar defect demonstrated



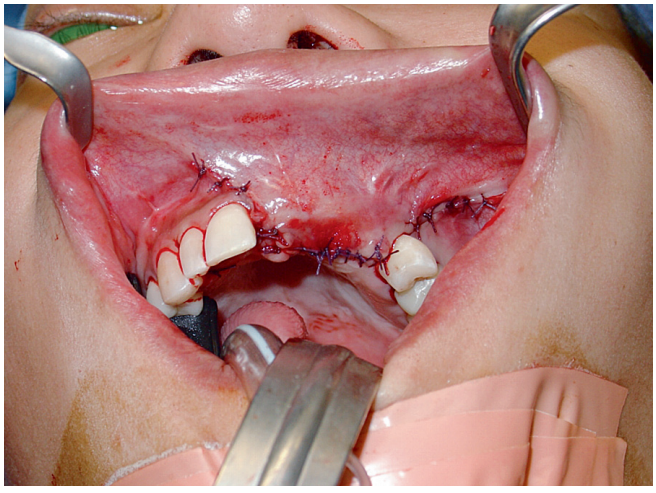
Iliac (corticocancellous) graft in place



Fixation securing graft



Fixation securing graft



Labial flap advanced and sutured in place

• **Figure 35-17, cont'd E, F,** and **G,** Intraoperative views of autogenous iliac corticocancellous graft reconstruction of the left maxillary dento-alveolar defect.

deformities, orthognathic deformities, and segmental dentoalveolar defects. Although there are few published reports that specifically address the management of posttraumatic secondary maxillofacial deformities, the application of basic orthognathic and dento-alveolar restorative principles will result in a favorable outcome for most patients. A team approach with coordinated efforts by the maxillofacial surgeon, the orthodontist, the restorative dentist, and the periodontist is often advantageous for the achievement of optimal results.

References

1. Adekeye EO: Ankylosis of the mandible: Analysis of 76 cases. *J Oral Maxillofac Surg* 41:442, 1983.
2. Allori AC, Chang CC, Fariña R, et al: Current concepts in pediatric temporomandibular joint disorders: Part 1. Etiology, epidemiology, and classification. *Plast Reconstr Surg* 126:1263–1275, 2010.
3. Becking AG, Zijderveld SA, Tuinzing DB: Management of posttraumatic malocclusion caused by condylar process fractures. *J Oral Maxillofac Surg* 56:1370–1374, 1998.
4. Becking AG, Zijderveld SA, Tuinzing DB: Management of posttraumatic malocclusion caused by condylar process fractures. *J Oral Maxillofac Surg* 56:1370–1377, 1998.
5. Blair VP: Operative treatment of ankylosis of the mandible. *Surg Gynecol Obstet* 19:436–456, 1914.
6. Blair VP: The consideration of contour as well as function in operations for chronic ankylosis. *Surg Gynecol Obstet* 46:167–179, 1928.
7. Bloomquist DS: Mandibular body sagittal osteotomy in the correction of malunited mandibular fractures. *J Maxillofac Surg* 10:18–23, 1982.
8. Bounds GA, Hopkins R, Sugar A: Septic arthritis of the temporomandibular joint: A problematic diagnosis. *Br J Oral Maxillofac Surg* 25:61–67, 1987.
9. Carlson DS: (Discussion of) Growth of a costochondral graft in the rat temporomandibular joint. *J Oral Maxillofac Surg* 50:857, 1992.
10. Coccaro PJ: Restitution of mandibular form after condylar injury in infancy. *Am J Orthod Dentofacial Orthop* 55:32, 1969.
11. Converse JM, Smith B, Wood-Smith D: Deformities of the midface resulting from malunited orbital and naso-orbital fractures. *Clin Plast Surg* 2:107, 1975.
12. Dahlstrom L, Kahnberg K-E, Lindahl L: 15 years follow-up on condylar fractures. *Int J Oral Maxillofac Surg* 18:18–23, 1989.
13. Daramola JO, Ajagbe HA, Oluwasanmi JO: Ankylosis of the temporomandibular joint following mandibular injury: Case reports. *Niger Med J* 9:395, 1979.
14. De Amaratunga NA: Mouth opening after release of maxillomandibular fixation in fracture patients. *J Oral Maxillofac Surg* 45:383–385, 1987.
15. Dodson TB, Bays RA, Pfeffle RC, et al: Cranial bone graft to reconstruct the mandibular condyle in *Macaca mulatta*. *J Oral Maxillofac Surg* 55:260, 1997.
16. He D, Yang C, Chen M, et al: Traumatic temporomandibular joint ankylosis: Our classification and treatment experience. *J Oral Maxillofac Surg* 69:1600–1607, 2011.
17. El-Mofty S: Cephalometric studies of patients with ankylosis of the temporomandibular joint following surgical treatment. *Oral Surg* 48:92, 1979.
18. El-Sheikh MM, Medra AM: Management of unilateral temporomandibular ankylosis associated with facial asymmetry. *J Craniomaxillofac Surg* 25:109, 1997.
19. El-Sheikh MM, Medra AM, Warda MH: Bird face deformity secondary to bilateral temporomandibular joint ankylosis. *J Craniomaxillofac Surg* 24:96, 1996.
20. Ellis E, III: Mobility of the mandible following advancement using maxillomandibular fixation and rigid internal fixation: an experimental investigation in *Macaca mulatta*. *J Oral Maxillofac Surg* 46:118–123, 1988.
21. Ellis E, III, Throckmorton GS: Treatment of mandibular condylar process fractures: Biological considerations. *J Oral Maxillofac Surg* 63:115–134, 2005.
22. Ellis E, III, Throckmorton GS, Palmieri C: Open treatment of condylar process fractures: assessment of adequacy of repositioning and maintenance of stability. *J Oral Maxillofac Surg* 58:27–34, 2000.
23. Ellis E, III, Walker R: Treatment of malocclusion and TMJ dysfunction secondary to condylar fractures. *Craniomaxillofac Trauma Reconstr* 2:1–18, 2009.
24. Engel MB, Brodie AG: Condylar growth and mandibular deformities. *Surgery* 22:976, 1947.
25. Evans HJ, Burwell RG, Merville LC, et al: *Residual deformities.* In Rowe NL, Williams JL, editors: *Maxillofacial injuries,* vol 2, Edinburgh, UK, 1985, Churchill Livingstone, p 1765.
26. Funke FW: Quantitative evaluation of mandibular condylar growth using tritiated thymidine in autoradiographic analysis. *J Dent Res* 50:1500, 1971.
27. Furlan S: Correction of saddle nose deformities by costal cartilage grafts: A technique. *Ann Plast Surg* 9:32, 1982.
28. Gibson T, Davis WB: The distortion of autogenous cartilage grafts: Its cause and prevention. *Br J Plast Surg* 10:257, 1957–1958.
29. Gilhus-Moe O: *Fractures of the mandibular condyle in the growth period*, Stockholm, Sweden, 1969, Scandinavian University Books, Universitatsforlaget.
30. Gillies HD: *Plastic surgery of the face*, London, UK, 1920, Oxford University Press.
31. Greenfield FM, Hirsch AC: Delayed treatment of fractured condyles. *J Oral Surg* 19:295, 1965.
32. Gruber RP: Lengthening the short nose. *Plast Reconstr Surg* 91:1252, 1993.
33. Hekkenberg RJ, Piedade L, Mock D, et al: Septic arthritis of the temporomandibular joint. *Otolaryngol Head Neck Surg* 120:780–782, 1999.
34. Hennig TB, Ellis E, III, Carlson DS: Growth of the mandible following replacement of the mandibular condyle with the sternal end of the clavicle: An experimental investigation in *Macaca mulatta*. *J Oral Maxillofac Surg* 50:1196, 1992.
35. Heurlin RJ, Gans BJ, Stuteville OH: Skeletal changes following fracture dislocation of the mandibular condyle in the adult rhesus monkey. *Oral Surg Oral Med Oral Pathol* 14:1490–1500, 1961.
36. Hincapie JW, Tobon D, Diaz-Reyes GA: Septic arthritis of the temporomandibular joint. *Otolaryngol Head Neck Surg.* 121:836–837, 1999.
37. Hinds EC, Parnes EL: Late management of condylar fractures by means of subcondylar osteotomy: Report of cases. *J Oral Surg* 24:54–59, 1966.
38. Jackson IT, Smith J, Mixter RC: Nasal bone grafting using split skull grafts. *Ann Plast Surg* 11:533, 1983.
39. Järvinen S: Traumatic injuries to upper permanent incisors related to age and incisal overjet. *Acta Odontol Scand* 37:335–338, 1979.
40. Joondeph DR, Bloomquist D: Open-bite closure with mandibular osteotomy. *Am J Orthod Dentofacial Orthop* 126:296–298, 2004.
41. Kaban LB, editor: Acquired abnormalities of the temporomandibular joint. In *Pediatric oral and maxillofacial surgery*, Philadelphia, 1990, Saunders, pp 307–342.
42. Kaban LB, Perrott DH, Fisher K: A protocol for management of temporomandibular joint ankylosis. *J Oral Maxillofac Surg* 48:1145, 1990.
43. Kawamoto HK, Jr: *Correction of established traumatic deformities of the facial skeleton using craniofacial principles in facial injuries*, St Louis, 1988, Mosby-Year Book, p 601.

44. Kazanjian VH: Ankylosis of the temporomandibular joint. *Surg Gynecol Obstet* 67:333–348, 1938.
45. Kennett S: Temporomandibular joint ankylosis: The rationale for grafting in the young patient. *J Oral Surg* 31:744, 1973.
46. Kiehn CI, DesPrez JD, Converse CF: Total prosthetic replacement of the temporomandibular joint. *Ann Plast Surg* 2:5, 1979.
47. Ohno K, Michi K, Ueno T: Mandibular growth following ankylosis operation in childhood. *Int J Oral Surg* 10(Suppl 1):324–328, 1981.
48. Leighty SM, Spach DH, Myall RW, Burns JL: Septic arthritis of the temporomandibular joint: Review of the literature and report of two cases in children. *Int J Oral Maxillofac Surg* 22:292–297, 1993.
49. Lindahl L, Hollender L: Condylar fractures of the mandible: II. Radiographic study of remodeling processes in the temporomandibular joint. *Int J Oral Surg* 6:153–165, 1977.
50. Lindquist C, Pihakari A, Tasanen A, Hampf G: Autogenous costochondral graft in temporomandibular joint arthroplasty. A survey of 66 arthroplasties in 60 patients. *J Maxillofac Surg* 14:143–149, 1986.
51. Link JO, Hoffman DC, Laskin DM: Hyperplasia of a costochondral graft in an adult. *J Oral Maxillofac Surg* 51:1392–1394, 1993.
52. Lund K: Mandibular growth and remodeling processes after mandibular fractures. *Acta Odontol Scand Suppl* 32:64, 1974.
53. MacIntosh RB, Henry FA: A spectrum of application of autogenous costochondral grafts. *J Maxillofac Surg* 5:257–267, 1977.
54. Manchester WM: Immediate reconstruction of the mandible and temporomandibular joint. *Br J Plast Surg* 18:291, 1965.
55. Manchester WM: Some technical improvements in the reconstruction of the mandible and temporomandibular joint. *Plast Reconstr Surg* 50:249, 1972.
56. Martinez-Garcia WR: Surgical correction of recurrent bony ankylosis of the temporomandibular joint. *Br J Oral Surg* 9:110, 1971.
57. Matukas VJ, Szymela VF, Schmidt JF: Surgical treatment of bony ankylosis in a child using a composite cartilage-bone iliac crest graft. *J Oral Surg* 38:903, 1980.
58. McNamara JA, editor: *Determinants of mandibular form and growth*, Ann Arbor, Mich, 1995, Center for Human Growth and Development, University of Michigan.
59. Melsen B, Bjerregaard J, Bundgaard M: The effect of treatment with functional appliance on a pathologic growth pattern of the condyle. *Am J Orthod Dentofacial Orthop* 90:503, 1986.
60. Millard R: (Discussion of) Nasal reconstruction with full-thickness cranial bone grafts and rigid internal skeletal fixation through a coronal incision. *Plast Reconstr Surg* 86:903, 1990.
61. Moorthy AP, Finch LD: Interpositional arthroplasty for ankylosis of the temporomandibular joint. *Oral Surg* 55:545, 1983.
62. Mosby EL, Hiatt WR: A technique of fixation of costochondral grafts for reconstruction of the temporomandibular joint. *J Oral Maxillofac Surg* 47:209, 1989.
63. Moss ML: The primacy of functional matrices in orofacial growth. *Dent Pract Dent Rec* 19:65–73, 1968.
64. Moss ML: The role of muscular functional matrices in development and maintenance of occlusion. *Bull Pac Coast Soc Orthod* 45:29–30, 1970.
65. Moss ML, Salentijn L: The primary role of functional matrices in facial growth. *Am J Orthod* 55:566–577, 1969.
66. Moss ML, Salentijn L: The compensatory role of the condylar cartilage in mandibular growth: Theoretical and clinical implications. *Dtsch Zahn Mund Kieferheilkd Zentralbl Gesamte* 56:5–16, 1971.
67. Munro IR, Chen YR, Park BY: Simultaneous total correction of temporomandibular ankylosis and facial asymmetry. *Plast Reconstr Surg* 77:517, 1986.
68. Nelson CL, Buttrum JD: Costochondral grafting for posttraumatic temporomandibular joint reconstruction: A review of six cases. *J Oral Maxillofac Surg* 47:1030, 1989.
69. Nørholt SE, Krishnan V, Sindet-Pedersen S, Jensen I: Pediatric condylar fractures: a long-term follow-up study of 55 patients. *J Oral Maxillofac Surg* 51:1302, 1993.
70. Nowak AJ, Casamassimo PS: Oral opening and other selected facial dimensions of children 6 weeks to 36 months of age. *J Oral Maxillofac Surg* 52:845, 1994.
71. Nwoku AL: Rehabilitating children with temporomandibular joint ankylosis. *Int J Oral Surg* 8:271, 1979.
72. Obeid G, Guttenberg SA, Connole PW: Costochondral grafting in condylar replacement and mandibular reconstruction. *J Oral Maxillofac Surg* 46:177, 1988.
73. Ohno K, Michi K, Ueno T. Mandibular growth following ankylosis operation in childhood. *Int J Oral Surg* 10(Suppl 1):324, 1981.
74. Peer L: *Transplantation of tissues, vol 1*, Baltimore, Md, 1955, Williams & Wilkins, p 181.
75. Peltomaki T: Growth of a costochondral graft in the rat temporomandibular joint. *J Oral Maxillofac Surg* 50:851, 1992.
76. Pensler JM, Christopher RD, Bewyer DC: Correction of micrognathia with ankylosis of the temporomandibular joint in childhood. *Plast Reconstr Surg* 91:799, 1993.
77. Perren SM: Physical and biological aspects of fracture healing with special reference to internal fixation. *Clin Orthop* 138:175, 1979.
78. Perrott DH: (Discussion of) Clinical and computed tomographic findings in costochondral grafts replacing the mandibular condyle. *J Oral Maxillofac Surg* 54:1400, 1996.
79. Perrot DH, Umeda H, Kaban LB: Costochondral graft construction/reconstruction of the ramus/condyle unit: Long-term follow-up. *Int J Oral Maxillofac Surg* 23:321–328, 1994.
80. Pickerill HP: Ankylosis of the jaw: Cartilage graft restoration of the joint: A new operation. *Aust N Z J Surg* 11:197, 1942.
81. Politis C, Fossion E, Bossuyt M: The use of costochondral grafts in arthroplasty of the temporomandibular joint. *J Craniomaxillofac Surg* 15:345–354, 1987.
82. Posnick JC: Craniomaxillofacial fractures in children. *Oral Maxillofac Surg Clin North Am* 6:169–185, 1994.
83. Posnick JC: (Discussion of) Mandibular fractures in infants: Review of the literature and report of seven cases. *J Oral Maxillofac Surg* 52:245–246, 1994.
84. Posnick JC: Management of facial fractures in children and adolescents. *Ann Plast Surg* 33(4):442–457, 1994.
85. Posnick JC: (Discussion of) Pediatric facial fractures: A demographic analysis outside an urban environment. *Ann Plast Surg* 38:584–585, 1997.
86. Posnick JC: Primary craniomaxillofacial fracture management. In Posnick JC, editor: *Craniofacial and maxillofacial surgery in children and young adults,* vol 30, Philadelphia, 2000, W.B. Saunders, pp 697–745.
87. Posnick JC: Secondary craniomaxillofacial traumatic deformities: Evaluation and treatment: TMJ ankylosis, skull defects, orthognathic deformities, saddle nose deformities, enophthalmus. In Posnick JC, editor: *Craniofacial and maxillofacial surgery in children and young adults,* vol 31, Philadelphia, 2000, W.B. Saunders, pp 746–781.
88. Posnick JC, Goldstein JA: Surgical management of temporomandibular joint ankylosis in the pediatric population. *Plast Reconstr Surg* 91:791, 1993.
89. Posnick JC, Ruiz R: (Discussion of) Age-related changes in the pattern of midface fractures in children: A chronographic analysis. *J Craniomaxillofac Trauma* 12–13, 2001.
90. Posnick JC, Ruiz R: (Discussion of) Management of condylar fractures in children. *J Craniomaxillofac Trauma* 6:16–17, 2001.
91. Posnick JC, Seagle MB, Armstrong D: Nasal reconstruction with full-thickness cranial bone grafts and rigid internal skeletal fixation through a coronal incision. *Plast Reconstr Surg* 86:894, 1990.
92. Posnick JC, Wells M, Pron GE: Pediatric facial fractures: Evolving patterns of treatment. *J Oral Maxillofac Surg* 51(8):836–844, 1993.
93. Poswillo D: Experimental reconstruction of the mandibular joint. *Int J Oral Surg* 3:400, 1974.

94. Poswillo DE: The late effects of mandibular condylectomy. *Oral Surg* 33:500, 1972.
95. Proffit WR, Vig KWL, Turvey TW: Early fracture of the mandibular condyles: Frequently an unsuspected cause of growth disturbances. *Am J Orthod Dentofacial Orthop* 78:1, 1980.
96. Rajgopal A, Banerji PK, Batura V, et al: Temporomandibular ankylosis. *J Maxillofac Surg* 11:37, 1983.
97. Raustia A, Pernu H, Pyhtinen J, Oikarinen K: Clinical and computed tomographic findings in costochondral grafts replacing the mandibular condyle. *J Oral Maxillofac Surg* 54:1393–1400, 1996.
98. Raveh J, Vuillemin T, Ladrach K, et al: Temporomandibular joint ankylosis: Surgical treatment and long-term results. *J Oral Maxillofac Surg* 47:900, 1989.
99. Regev E, Koplewitz BZ, Nitzan DW, Bar-Ziv J: Ankylosis of the temporomandibular joint as a sequela of septic arthritis and neonatal sepsis. *Pediatr Infect Dis J* 22:99–101, 2003.
100. Risdon F: Ankylosis of the temporomandibular joint. *J Am Dent Assoc* 21, 1933.
101. Rowe NL: Surgery of the temporomandibular joint. *Proc R Soc Med* 65:383, 1972.
102. Rowe NL: Ankylosis of the temporomandibular joint: Part 1. *J R Coll Surg* 27:67, 1982.
103. Rowe NL: Ankylosis of the temporomandibular joint: Part 2. *J R Coll Surg* 27:167, 1982.
104. Rowe NL: Ankylosis of the temporomandibular joint: Part 3. *J R Coll Surg* 27:209, 1982.
105. Rubens BC, Stoelinga PJW, Weaver TJ, Blijdorp PA: Management of malunited mandibular condylar fractures. *Int J Oral Maxillofac Surg* 19:22–25, 1990.
106. Rubenstein LK: (Discussion of) Oral opening and other selected facial dimensions of children 6 weeks to 36 months of age. *J Oral Maxillofac Surg* 52:848, 1994.
107. Samman N, Cheung LK, Tiderman H: Overgrowth of a costochondral graft in an adult male. *Int J Oral Maxillofac Surg* 24:333–335, 1995.
108. Sarnat BG: Developmental facial abnormalities and the temporomandibular joint. *J Am Dent Assoc* 79:108–117, 1969.
109. Sarnat BG, Muchnic H: Facial skeletal changes after mandibular condylectomy in growing and adult monkeys. *Am J Orthod* 60:33–45, 1971.
110. Sarnat BG, Muchnic H: Facial skeletal changes after mandibular condylectomy in the adult monkey. *J Anat* 108:323–338, 1971.
111. Sarnat BG, Muchnic H: Facial skeletal changes after mandibular condylectomy in growing and adult monkeys. *Am J Orthod* 62:428, 1972.
112. Silvennoinen U, Iizuka T, Oikarinen K, Lindqvist C: Analysis of possible factors leading to problems after nonsurgical treatment of condylar fractures. *J Oral Maxillofac Surg* 52:793–799, 1994.
113. Skolnik J, Iranpour B, Westesson PL, Adair S: Pubertal trauma and mandibular asymmetry in orthognathic surgery and orthodontic patients. *Am J Orthod Dentofacial Orthop* 105:73–77, 1994.
114. Snyder CC, Levine GA, Dingman DL: Trial of a sternoclavicular whole joint graft as a substitute for the temporomandibular joint. *Plast Reconstr Surg* 48:447, 1971.
115. Spitzer WJ, Vanderborght G, Dumbach J: Surgical management of mandibular malposition after malunited condylar fractures in adults. *J Craniomaxillofac Surg* 25:91, 1997.
116. Steinhauser EW: The treatment of ankylosis in children. *Int J Oral Surg* 2:129, 1973.
117. Tessier P. Aesthetic aspects of bone grafting to the face. *Clin Plast Surg* 8:279, 1981.
118. Tessier P: Autogenous bone grafts taken from the calvarium for facial and cranial applications. *Clin Plast Surg* 9:531, 1982.
119. Topazian RG: Etiology of ankylosis of temporomandibular joint: Analysis of 44 cases. *J Oral Surg* 22:227, 1964.
120. Topazian RG: (Discussion of) A protocol for management of temporomandibular joint ankylosis. *J Oral Maxillofac Surg* 48:1152, 1990.
121. Trimble LD, Schoenaers JA, Stoelinga PJ: Acute suppurative arthritis of the temporomandibular joint in a patient with rheumatoid arthritis. *J Maxillofac Surg* 11:92–95, 1983.
122. Troulis MJ, Williams WB, Kaban LB: Endoscopic condylectomy and costochondral graft reconstruction of the ramus condyle unit. *J Oral Maxillofac Surg* 61:63, 2003.
123. Troulis MJ, Williams WB, Kaban LB: Endoscopic mandibular condylectomy and reconstruction: Early clinical result. *J Oral Maxillofac Surg* 62:460, 2004.
124. Vargervik K: (Discussion of) Cranial bone graft to reconstruct the mandibular condyle in Macaca mulatta. *J Oral Maxillofac Surg* 55:267, 1997.
125. Walker RV: Traumatic mandibular condyle fracture dislocations: Effect on growth in the Macaca rhesus monkey. *Am J Surg* 100:850, 1960.
126. Walker RV: Condylar abnormalities. Transactions of the 2nd International Conference on Oral Surgery, Copenhagen, 1965, Munksgaard Copenhagen, pp 81–96, 1967.
127. Walker RV: Condylar fractures: Nonsurgical management. *J Oral Maxillofac Surg* 52:1185, 1994.
128. Ware WH, Brown SL: Growth centre transplantation to replace mandibular condyles. *J Maxillofac Surg* 9:50–58, 1981.
129. Ware WH, Taylor RC: Replantation of growing mandibular condyles in rhesus monkeys. *Oral Surg Oral Med Oral Pathol* 19:669–677, 1965.
130. Weiss P: Regeneration of the mandibular and temporomandibular joints following subperiosteal exarticulation of the mandible in the young dog [German]. *Dtsch Zahnarztl Z* 24:355–360, 1969.
131. Wheeler ES, Kawamoto HK, Jr, Zarem HA: Bone grafts for nasal reconstruction. *Plast Reconstr Surg* 69:9, 1982.
132. Zachariades N, Mezitis M, Michelis A: Posttraumatic osteotomies of the jaws. *Int J Oral Maxillofac Surg* 22:328–331, 1993.
133. Zide MF: (Discussion of) An accurate method for open reduction and internal fixation of high and low condylar process fractures. *J Oral Maxillofac Surg* 52:812, 1994.
134. Zou Z-J, Wu W-T, Sun G-X, et al: Remodeling of the temporomandibular joint after conservative treatment of condylar fractures. *Dentomaxillofac Radiol* 16:91–98, 1987.

36

Idiopathic Condylar Resorption: Evaluation and Treatment

JEFFREY C. POSNICK, DMD, MD

- Theories of Etiology
- Clinical Perspectives
- Current Clinical Approach to Correction of the Secondary Jaw Deformities
- Brief Overview of Author's Current Thinking
- Case Presentations
- Conclusions

In patients with the condition of uncertain etiology that is commonly referred to as *idiopathic condylar resorption* (ICR)*, the condyles of the mandible partially resorb, thereby causing a loss of condylar height with secondary alterations of the maxillofacial morphology, occlusion, and head and neck function.[34,43,100] *Progressive condylar resorption* (PCR)* is a general term that is used to describe conditions that result in a similar loss of condylar height and secondary effects but with known associations (e.g., juvenile idiopathic arthritis, including any of its seven subtypes).[13,24,59,76,92,93]

In general, ICR has the following features:

- It generally affects girls and women who are between the ages of 15 and 35 years.
- It is most frequent among teenage girls during the pubertal growth spurt.
- It typically results in bilateral symmetric condylar involvement followed by stabilization (remission). In the majority of affected individuals, limited further resorption occurs. Unfortunately, in some, a second episode will result in the further loss of condylar height.
- There is no agreed-upon and proven etiology. ICR frequently seems to occur as part of the natural course of events rather than in conjunction with active therapy or treatment. It may also coincide with or be observed during or after active dental restorative, orthodontic, or surgical interventions.
- During the active phase of condylar resorption, TMJ and masticatory muscle discomfort is expected. The active phase often subsides within 6 to 12 months.
- When the condition becomes quiescent, the individual is generally left with satisfactory TMJ function without significant limitations in mouth opening or intracapsular pain. Persistent joint noise is frequent.
- Head and neck functions are likely to be negatively impacted including: chewing; breathing; speech articulation; and body image.
- When the condition is in remission, a cartilaginous cap over an intact cortical rim can generally be documented. A deflated or diminished condylar head is generally seen on imaging (e.g., magnetic resonance imaging or computed tomography scanning). The glenoid fossa and the articular disc typically remain biologically intact.

Mandibular and occlusal findings generally include the following:

- Change in the shape of the condylar heads (i.e., flattening and thinning)
- Decrease in condylar height
- Loss of overall posterior facial height
- Mandibular retropositioning with clockwise rotation (high angle)
- Angle Class II anterior open bite, excess overjet malocclusion

Theories of Etiology

The current dominant theory holds that the etiology of ICR is hormonally mitigated, immunologically controlled and may arise in genetically susceptible individuals in conjunction with environmental factors.* Sex hormones are thought to modulate biochemical changes within the TMJ, which

*These terms are likely to change as further knowledge becomes available.

*References 1, 3-7, 15, 23, 28, 29, 31, 42, 51, 58, 65, 79, 86, 99, 111, 113

may then result in condylar resorption. For some time, it has been suggested that there is alteration in the serum hormonal levels in at least some affected girls and women with ICR.[31] Gunson and colleagues described endocrine function among women who arrived at their clinic with a history, physical examination, and radiographic findings that were consistent with ICR. The average age at presentation was 26 years (range, 15 to 45 years).[31] Twenty-five of the 27 patients (93%) had low levels of serum 17-beta estradiol at the mid-menstrual cycle. Two subgroups were further differentiated. The first group did not produce estrogen naturally (8 out of 27 patients), and the second group (19 out of 27 patients) had low 17-beta estradiol levels that were presumed to be the result of oral contraceptive pill usage. The authors theorize that, whether the condition is induced by ethinyl estradiol (i.e., oral contraceptive pills) or by premature ovarian failure, a low circulating 17-beta estradiol serum level negatively affects the natural reparative capacity of the condyles. The authors believe that, when 17-beta estradiol deficiency is coupled with the presence of local factors that cause compressive forces, condylar lysis is more likely to occur and to ultimately result in the observed condition called ICR.[8,14,27,46,64] Unfortunately, the study did not include a control group for oral contraceptive pill use or precise knowledge of the phase of the menstrual cycle at the time of hormone serum sampling. Although hormone influences no doubt play a role, an autoimmune aspect is also likely.[31]

Gunson and Arnett comment that whatever the underlying etiology of ICR, all bone loss at the condylar level involves a *common resorptive pathway* that includes cytokine-activated osteoblasts that then recruit and promote the activity of osteoclasts.[32] This in turn results in the secretion of enzymes that are responsible for the breakdown of hydroxyapatite and collagen. A case in point is *juvenile idiopathic arthritis* (ex. polyarticular arthritis, rheumatoid factor positive), which is caused by a B-cell–mediated autoimmune reaction to synovial tissues.[33,80,105] A consequence of this reaction is the presence of local inflammatory cells, which secrete cytokines that cause the activation of osteoblast-mediated osteoclast catabolism.[89] The end result is bone breakdown through the common resorptive pathway.[30] In patients with juvenile idiopathic arthritis (JIA), a current treatment approach is to minimize articular bone loss by using pharmacologic drugs that interfere with specific cytokines and enzymes along the common resorptive pathway rather than open joint or joint replacement procedures. This may include non-steroidal anti-inflammatories (NSAIDS) or other drugs such as methotrexate or a newer class of medications called biologicals.[10,35,36,39,48,52,80,105] Gunson and Arnett point out that a similar approach to ICR may prove to be useful.[32]

Another theory of the cause of ICR is avascular necrosis of the condyle as a result of the compression of specific vessels that supply the condyle followed by condylysis, loss of condyle height with secondary jaw deformity and malocclusion.[16,17,45,62,72,81-84] Piper and Choung had speculated that pathologic compressive forces of the posterior aspect of the condyle on the ligamentous retrodiscal soft tissues constrict the small vessels, thereby limiting circulation to the condyles with resulting in aseptic necrosis that accounts for the observed condylysis in patients with ICR.[17,72] They went on to hypothesize that either a chronically dislocated non-reducing disc or specific patterns of malocclusion may also cause this cycle of events. As an extension of this thinking, preventative open-joint procedures were offered to alleviate the theoretical compression of the condylar circulation. Those of us that are skeptical of the non-reducing disc cause of ICR point out that the almost universally observed bilateral symmetric simultaneous nature of this condition and its occurrence only in females make the theory an improbable explanation.

Clinical Perspectives

The foregoing theories remain unproven, but the effects of estrogen on condylar resorption and repair with or without an autoimmune component seem indisputable. Further studies will be required to determine the additive effects of occlusion and masticatory muscle forces on the TMJ in the presence of hormonal imbalance, autoimmune influences, genetic predisposition and other environmental factors.

Some clinicians suggest that, with a lack of clarity about either the etiology of the condition or the effects of various treatment modalities and without a guaranteed endpoint to the condylar degeneration in any given patient, it is best to remove the affected condyles and either reconstruct them with a *costochondral (rib) graft* or to remove the whole joint (i.e., the condyle, the disc, and the glenoid fossa) and replace it with an *alloplastic total joint*. Proponents of a partial or total joint replacement approach to the management of ICR are correct in their thinking that the only way to be certain that a further loss of posterior facial height will not continue is to remove and replace the condyle.[55,56,107,110] For the great majority of patients with ICR, joint replacement treatment seems radical on the basis of the following: 1) the unlikely probability of long-term TMJ pain 2) the expected satisfactory long-term mandibular (mouth) opening 3) the expected condylar stabilization ("burn-out") that is typically seen and 4) the known potential for perioperative complications with invasive open joint procedures and the long-term failure rates of total joint replacement.

Mercuri presents an alternative point of view with a recommendation for total joint replacement to "eliminate the variable issue of ongoing condylar resorption."[55,56] He offers total alloplastic reconstruction as a definitive solution. He also states that the non-alloplastic (biologic) condyle option (e.g., costochondral grafting) requires stabilization of the graft with minimal mobility while integration (union) to the host mandibular occurs. This limits early masticatory muscle rehabilitation, which he feels is contrary to the tenets of physical rehabilitation after joint surgery. The

disadvantages of alloplastic total TMJ reconstruction include the following: 1) the cost of the device 2) the expected material wear 3) the potential for long-term instability and failure 4) the incidence of both early and late complications including infections with the associated need for device removal and 5) the fact that alloplastic implants will not follow a patient's normal growth pattern. Joint replacement advocates admit that patients must be advised that alloplastic total TMJ reconstruction devices are expected to have a limited functional life. In addition, the total joint replacement approach requires the removal of the functional glenoid fossa and disc along with excision of the condylar head, neck, and posterior ramus on both sides.

Recommendations regarding how best to limit progression of condylar resorption and then how to treat the consequences of ICR depend on the clinician's beliefs about the following 1) the disorder's likely etiology 2) the effects of malocclusion and the masticatory muscle forces and 3) the natural history of the degenerative process.

Some clinicians speculate that, if "decompressive" TMJ treatment is initiated early during the clinical course of active ICR, then less condylar resorption will occur. Others believe that, after the resorptive process has begun, it runs its course despite any attempt to "unload" the joint. Even with a lack of evidence-based research into the subject, most clinicians recommend attempts to limit mechanical compressive TMJ forces through the use of conservative measures (i.e., splints, muscle relaxants, medications and, diet modifications) in the hopes that less resorption (condylysis) will occur.[11,12,19,21,25,37,40,47,63,66,101] Some authors believe that counterclockwise rotation of the maxillomandibular complex as part of the orthognathic correction will increase the compressive condylar forces, whereas others state that it is actually a method of "unloading" the joint.[20,22,41,44,50,57,61,71,85,88,105,108] I agree with the published studies that document long-term skeletal stability when counterclockwise rotation of the maxillomandibular complex is carried out as part of the correction of mandibular-deficient dentofacial deformity.[74,75,77,109] A common concern is that TMJ "loading" (i.e., the presence of compressive forces) will occur if the condyles are "overseating" or if medial or lateral condylar torquing is allowed to occur during orthognathic surgery.[3-6] Some clinicians speculate that individuals who have persistent TMJ symptoms (e.g., popping, clicking) after the orthodontic treatment and corrective orthognathic surgery are at higher risk for progression.[2] Most published studies confirm that joint noise (e.g., popping, clicking, grating) is not in itself an indicator of future condylar resorption.[60,87,104]

Current Clinical Approach to Correction of the Secondary Jaw Deformities

After the diagnosis of ICR is made, non-invasive measures (e.g., splint therapy, muscle relaxants, medications, and diet modification) to "unload" and "stabilize" the condyles or at least to relieve masticatory muscle hyperactivity and discomfort are initiated. Orthognathic procedures and orthodontic treatment carried out to correct the secondary malocclusion, the facial dysmorphology, and to open the airway are more likely to be maintained if the condylar resorption process has been stable for a period of time.[49,77,112] Stability can be documented by minimal ongoing change in the occlusion, an intact cartilaginous cap, and an underlying rim of cortical bone over the partially resorbed condyle. The judicious use of technetium-99m methylene diphosphonate quantitative condylar bone scintigraphy can be a helpful tool for assessing whether or not condylysis is active.[9,18,26,53,69,70,73,78,90,103] Unfortunately, these and other tests only confirm the current anatomy and activity level; they cannot predict the future.

The resulting maxillomandibular dysmorphology observed in patients with ICR can affect speech, swallowing, chewing, breathing, and lip closure. ICR rarely results in disabling facial or TMJ pain or significant limitations in vertical mouth opening. The observed facial aesthetic consequences of ICR are the result of the maxillo-mandibular changes which distort but do not actually deform the soft-tissue envelope. The overlying soft tissues of the lips, cheeks, and neck and the underlying soft tissues of the tongue and soft palate can be normalized only by correcting the skeletal deformities. The achievement of favorable facial aesthetics and the opening of the airway generally require the surgical repositioning of the maxilla (Le Fort I osteotomy), the mandible (ramus osteotomies), and the chin region (oblique osteotomy). Limiting the surgery to either the maxilla or the mandible with the idea of achieving a more stable occlusal result is not substantiated in the literature and is likely to result in suboptimal results. The key to achieving a successful outcome (i.e., enhanced aesthetics, improved occlusion, and an open airway) for the majority of patients with ICR is to surgically accomplish adequate horizontal advancement of the entire maxillomandibular complex often with counterclockwise rotation.

The suggestion that, for the patient who has experienced ICR, the use of distraction osteogenesis techniques rather than standard orthognathic procedures will achieve a more stable long-term occlusion is not supported by the literature.[38,54,68,91,94,95,102] The distraction approach 1) generally limits the region of reconstruction to the mandible 2) it requires greater patient convalescences and compliance, and 3) it may increase (rather than decrease) the compressive forces on the condyles.

When the condylar resorption process is believed to be "burnt out," a comprehensive approach to correct the secondary deformities that involves an orthodontist, an orthognathic surgeon, a TMJ specialist, and appropriate imaging studies is recommended. Periodontal and restorative dental work may also be needed. Evaluation by a rheumatologist should also be considered. A thorough

evaluation of the upper airway is essential to clarify any day and nighttime breathing difficulties. The simultaneous correction of chronic nasal airway obstruction and any baseline obstructive sleep apnea should be a primary objective (see Chapters 10 and 26).

The key to a favorable reconstruction for an individual with end-stage ICR or PCR is to define the functional disability (i.e., breathing, chewing, speech, swallowing, lip closure) and the extent of skeletal dysmorphology. The establishment of a functional occlusion that limits stress in the entire masticatory system without significant centric relation–centric occlusion discrepancies and without parafunctional habits is desirable (see Chapter 9). The successful performance of orthognathic, intranasal, and dental procedures to achieve these objectives represents our standard approach; see the case presentations later in this chapter.

Kaban and colleagues offers an alternative approach when the patient is not willing to take the risk that a degree of resorption may continue or reoccur at some point in the future.[24,43,67,92,96-98] The authors completed a retrospective case series of 15 patients with the diagnosis of active bilateral ICR who chose a condylar replacement approach between 1999 and 2004. Each patient underwent bilateral endoscopic-assisted condylectomy and autogenous costochondral graft reconstruction. Patients with the following characteristics were included:

1. Active bilateral ICR documented by worsening Class II malocclusion with open bite on serial clinical examinations, radiographs, photographs, and dental casts
2. A positive technetium-99m methylene diphosphonate bone scan
3. A minimum of 12 months of postoperative follow up

Symptoms such as myofascial pain and occlusal discomfort are managed by splint therapy, physical therapy, muscle relaxants, nonsteroidal anti-inflammatory drugs, and other modalities as indicated. Patients who respond to treatment as documented by the relief of symptoms, no occlusal radiograph changes over a 2-year period, and a negative bone scan begin orthodontic treatment in preparation for standard orthognathic surgery. Patients with active ICR who do not respond to the previously described treatment modalities or who prefer not to follow the protocol for a variety of reasons are considered candidates for condylectomy and costochondral graft reconstruction. Fifteen patients fell into this latter category and underwent bilateral endoscopic condylectomies and costochondral graft reconstruction. None of the 15 study patients suffered significant surgical complications, such as fractures or the erroneous placement of the graft within the fossa. Postoperatively, all patients showed Class I occlusion with no anterior open bite. All patients maintained a clinically acceptable and reproducible occlusion one year later with a mean maximum incisor mouth opening of 39 mm. Kaban and colleagues stressed the importance of making a distinction between active and inactive ICR and the patient's refusal to wait for the presumed remission of the active condylar resorption phase. Only those who were unwilling to risk recurrence are offered the condylar replacement option; all others undergo a standard orthognathic approach.

Brief Overview of Author's Current Thinking

With the osteoarthritic condition known as ICR or PCR, after the acute process "runs its course" (i.e., after 6 to 12 months for most individual's), a degree of bilateral condylar resorption will have occurred. This is typically followed by relative condylar stability, satisfactory range of motion of the mandible, and no significant TMJ limitations. The resulting anterior open bite Class II malocclusion may be responsible for masticatory muscle discomfort during function. Temporary relief of these symptoms with a neutralizing splint is generally possible.

The definitive correction of the secondary maxillomandibular deformity through standard osteotomies in combination with appropriate orthodontics has proven beneficial to improve the occlusion, to open the airway, and to enhance facial aesthetics. Concern about further resorption of the "fragile" condyles is best managed by avoiding masticatory activities that "load" (i.e., compress) the joint and with the use of systemic medications when indicated.[32]

The treatment of juvenile idiopathic arthritis (JIA) has improved dramatically during recent years with the advent of disease-modifying anti-rheumatoid drugs. The orthognathic surgeon will encounter patients on a drug regimen that consists of nonsteroidal anti-inflammatory drugs [NSAIDS], glucocorticoids, methotrexate, and biologic agents. In these cases, consultation with a rheumatologist is recommended, but the surgeon should also be aware of these medications, because their presence could affect surgical outcome. Prudent perioperative management of these drugs is necessary to optimize surgical outcome. A balance must be struck between minimizing potential surgical complications and maintaining disease control to prevent further joint damage (i.e., condylar resorption) that would negatively affect the outcome.[41A]

To review, ICR should not be managed either as a standard internal derangement or as a typical temporomandibular disorder (TMD). The concept of resolving the problem through either an open-joint procedure or through joint replacement continues to seem off the mark given the known natural history of this entity for the majority of affected individuals. A joint-replacement approach is generally reserved for those few patient who have undergone failed surgical procedures with additive iatrogenic TMJ dysfunction.

Case Presentations

CASE 36-1

Progressive Condylar Resorption (Fig. 36-1)

History

A 23-year-old Caucasian female was diagnosed with juvenile rheumatoid arthritis when she was 10 years old; the disease primarily affects her knees and ankles. Involvement of both of her TMJs began when she was 12 years old and resulted in PCR that stabilized when she was 14 years old. She underwent compensatory orthodontics when she was between the ages of 14 and 16 years, and she has reported pain-free satisfactory mandibular range of motion since that time. When she was 19 years old, she sustained trauma to

• **Figure 36-1** The patient before and after treatment. Final maxillary restorative dental work is not yet completed. **A,** Frontal views in repose before and after surgery. **B,** Frontal views with smile before and after surgery.

©Elsevier Inc.
Orthognathic Surgery; J.C. Posnick

• **Figure 36-1, cont'd C,** Oblique views before and after surgery. **D,** Profile views before and after surgery.

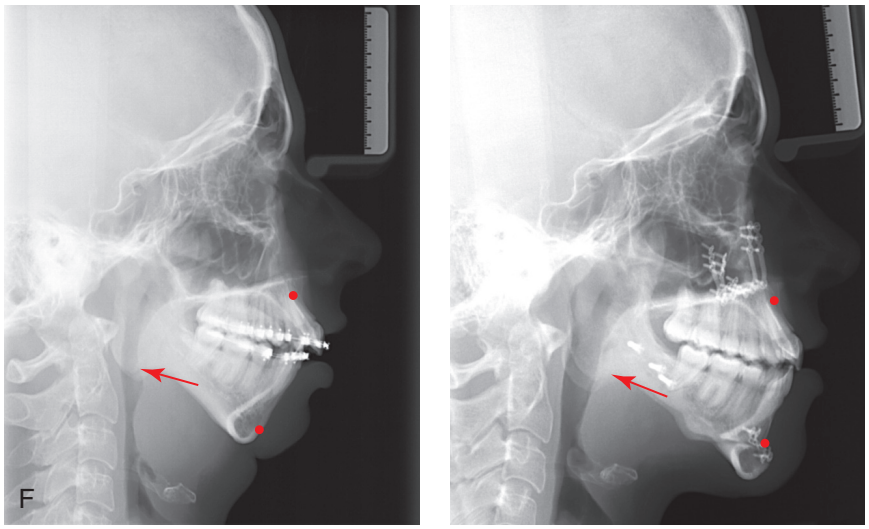
Continued

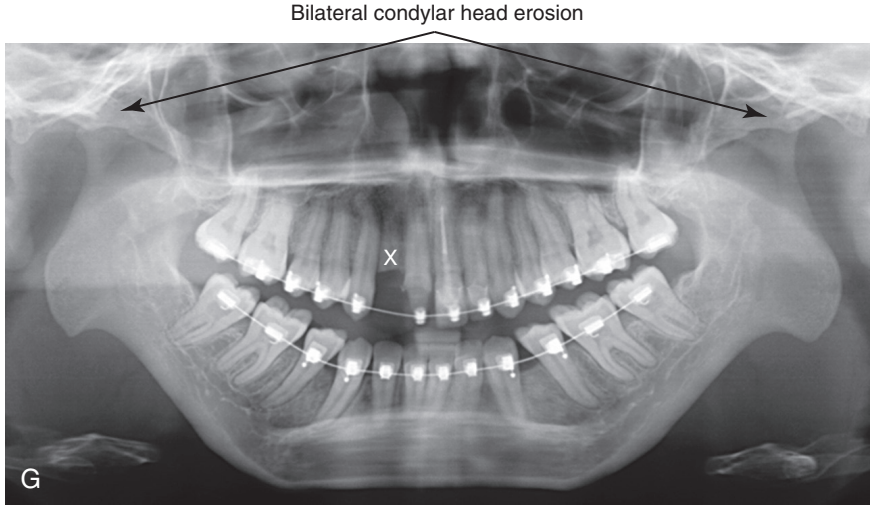
Prior to retreatment

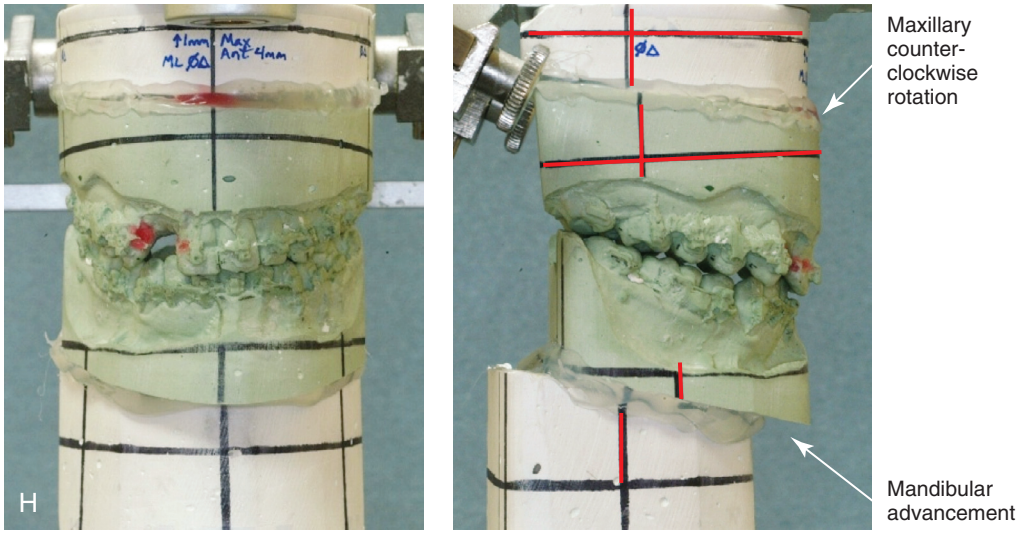
Pre surgery after mandibular bicuspid extractions and orthodontics

After treatment

• **Figure 36-1, cont'd E,** Occlusal views before retreatment, before surgery but after bicuspid extractions, and after surgery.







• **Figure 36-1, cont'd F,** Lateral cephalometric views before and after surgery. **G,** Panorex view before surgery. An "X" is on a root tip that requires extraction. A question mark is on an incisor that may require extraction. **H,** Articulated dental casts that indicate analytic model planning. *From Posnick JC, Fantuzzo JJ: Idiopathic condylar resorption: current clinical perspectives,* J Oral Maxillofac Surg *65:1617-1623, 2007.*

the four maxillary incisors with a loss of the right lateral incisor and the need for root canal treatment of the right central incisor. Temporary restorations were placed. The patient then continued to follow up with a general dentist and orthodontist when she was between 19 and 23 years old, with no changes noted in her occlusion (i.e., no progress of the condylar resorption).

Evaluation

The 23 year old woman arrived for surgical consultation in the hope of achieving enhanced facial aesthetics and improved function (i.e., mastication, breathing, lip posture, and speech articulation). She underwent evaluations by a speech pathologist, an otolaryngologist/head and neck surgeon, an orthodontist, a periodontist, a restorative dentist, a rheumatologist, and a TMJ specialist.

Treatment

Preoperative orthodontic treatment included the removal of the mandibular first bicuspids to uncrowd the arch. Unfortunately, as a result of previous dentoalveolar trauma, only minimal repositioning of the maxillary anterior teeth was possible, thereby preventing optimal incisor positioning. The patient's surgery included the following:

- Maxillary Le Fort I osteotomy
  - Vertical intrusion at the incisors
  - Maxillary plane counterclockwise rotation
  - Horizontal advancement at the incisors
- Bilateral sagittal split ramus osteotomies
  - Mandibular plane counterclockwise rotation
  - Horizontal advancement
- Oblique osteotomy of the chin
  - Horizontal advancement
  - Vertical shortening
- Intranasal procedures
  - Septoplasty
  - Reduction of the inferior turbinates

After the initial surgical healing (5 weeks), finishing orthodontics continued for 6 months. The removal of the orthodontic appliances was followed by maintenance splint therapy and orthodontic retention that was primarily used while the patient was sleeping.

CASE 36-2

Idiopathic Condylar Resorption (Fig. 36-2)

History

This systemically healthy 12-year-old girl experienced severe TMJ discomfort with limited and painful mouth opening. Mandibular retrusion and anterior open-bite deformity were progressive over the subsequent 6 months. When the patient was 13 years old, her orthodontist offered maxillary first bicuspid extractions with orthodontic retraction to neutralize the occlusion. From the ages of 13 to 15 years, orthodontic treatment accomplished this objective. When the patient reached the age of 16 years, concerns about her facial aesthetics, her airway, and her periodontal health were voiced, and alternative treatment was sought.

Evaluation

A 16 year old teenager arrived for surgical evaluation without pain in the TMJ and facial region. Vertical mouth opening was to 35 mm. Radiographs confirmed no progression of condylar resorption for at least 2 years. A complete workup for systemic joint disease was negative. The patient described having difficulty breathing through her nose, and she was found to have a deviated septum and hypertrophic inferior turbinates.

Treatment

Redo orthodontic treatment included the removal of the mandibular first bicuspid teeth to uncrowd the arch and upright the incisors. The patient's surgery included the following:

- Maxillary Le Fort I osteotomy
  - Vertical intrusion at the incisors
  - Maxillary plane counter clockwise rotation
  - Horizontal advancement at the incisors
  - Cant correction
- Bilateral sagittal split ramus osteotomies
  - Mandibular plane counterclockwise rotation
  - Horizontal advancement
- Oblique osteotomy of the chin
  - Horizontal advancement
  - Vertical shortening
- Intranasal procedures
  - Septoplasty
  - Reduction of the inferior turbinates
- Removal of wisdom teeth

After initial surgical healing (5 weeks), finishing orthodontics continued for 6 months. Removal of orthodontic appliances was followed by the use of routine removable retainers.

©Elsevier Inc.
Orthognathic Surgery; J.C. Posnick

• **Figure 36-2** Patient before and after the completion of orthodontics and jaw surgery. **A,** Frontal views in repose before and after surgery. **B,** Frontal views with smile before and after surgery.

Continued

• **Figure 36-2, cont'd C,** Oblique views before and after surgery. **D,** Profile views before and after surgery.

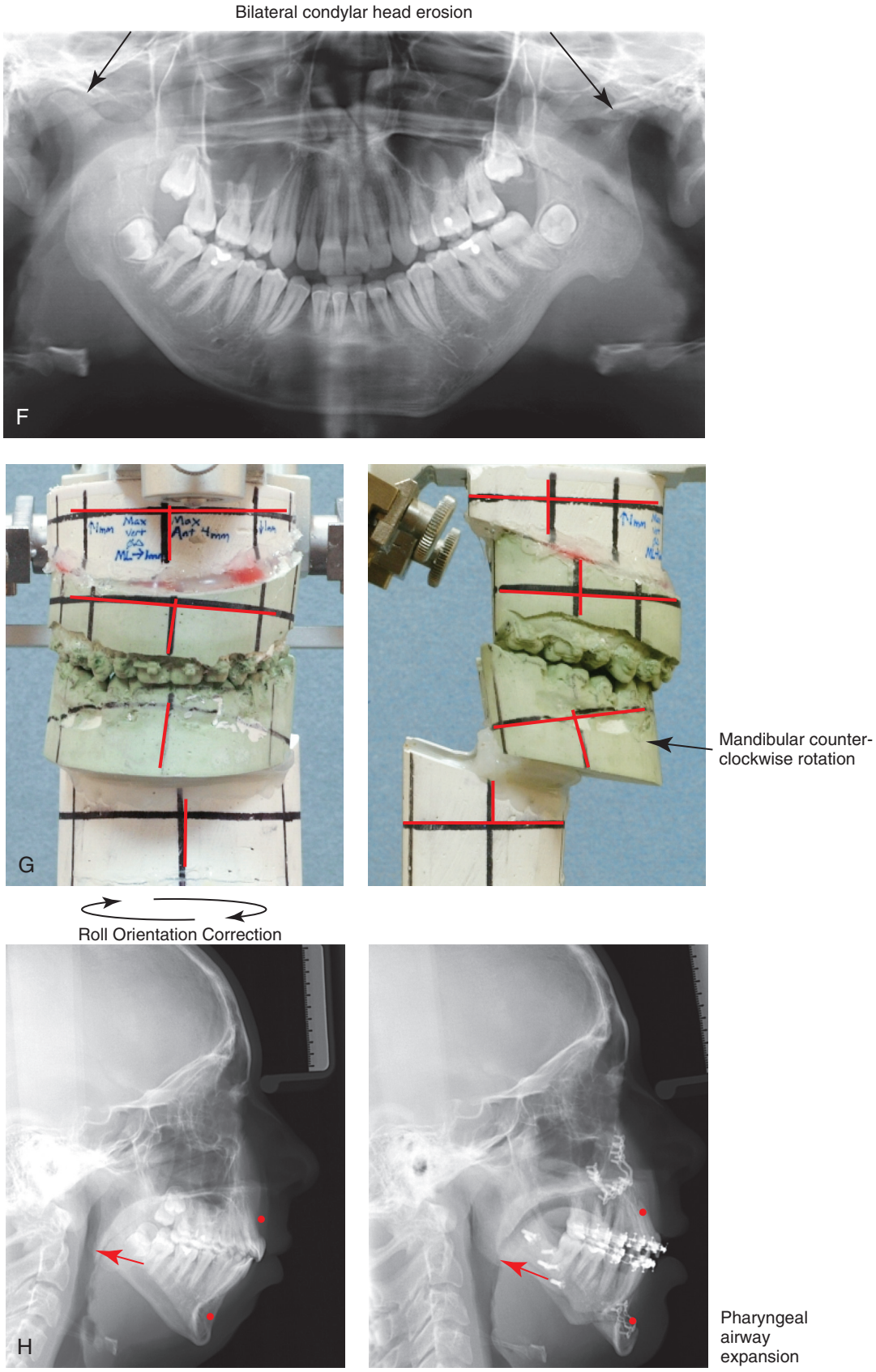
Prior to re-do orthodontics. She previously underwent maxillary bicuspid extractions

Re-do orthodontics in progress including mandibular bicuspid extractions

After treatment

• **Figure 36-2, cont'd E,** Occlusal views before retreatment, before surgery but after mandibular bicuspid extractions, and after surgery.

Continued



• **Figure 36-2, cont'd** **F,** Panorex view before surgery. **G,** Articulated dental casts that indicate analytic model planning. **H,** Lateral cephalometric views before and after surgery. *A, B, C, D, E (center row, bottom row), F, G, H, from Posnick JC, Fantuzzo JJ: Idiopathic condylar resorption: current clinical perspectives,* J Oral Maxillofac Surg *65:1617-1623, 2007.*

CASE 36-3

Idiopathic Condylar Resorption (Fig. 36-3)

History

A 12-year-old female underwent orthodontic treatment when she was between the ages of 12 and 15 years; including four bicuspid extractions. She was felt to have achieved a satisfactory occlusion without the need for further care. When she was 19 years old and away at college, she experienced an episode of temporomandibular disorder that involved TMJ region and masticatory muscle discomfort and that limited her mandibular range of motion and her ability to chew. Over a 6-month period, the symptoms subsided. During this time, a progressive anterior open-bite and a retrusive mandibular profile occurred. For the next two years (i.e., from the ages of 19.5 years to 21.5 years), she was followed by a restorative dentist and an orthodontist; no clinical progression was found. There were no further changes in her occlusion, but she did have intermittent masticatory muscle discomfort that improved with the use of an occlusion neutralization splint.

• **Figure 36-3** Patient before and after treatment. **A,** Frontal views in repose before and after treatment. **B,** Frontal views with smile before and after treatment.

Continued

• **Figure 36-3, cont'd C,** Oblique views before and after treatment. **D,** Profile views before and after treatment.

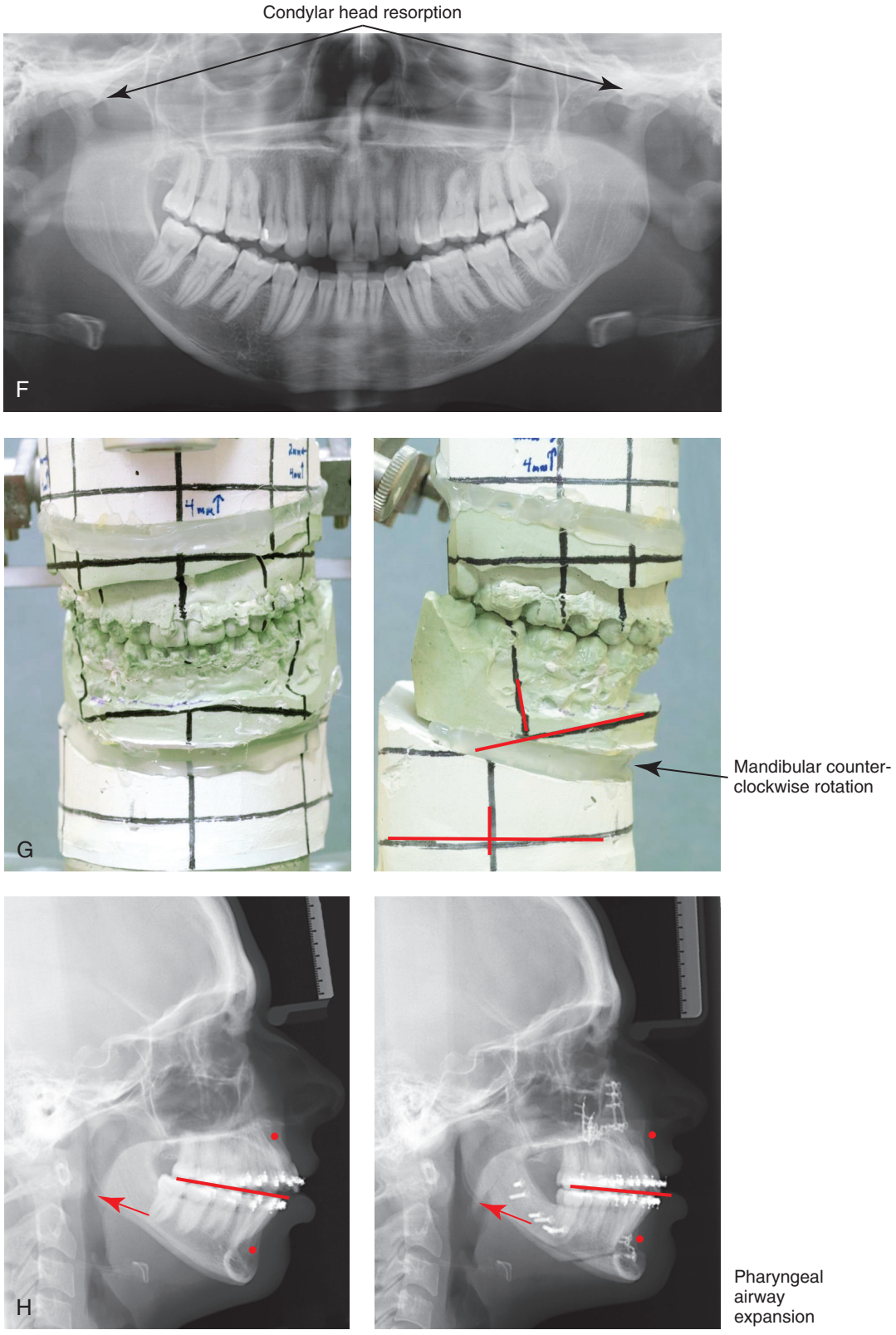
Prior to re-do orthodontics. Earlier treatment included four bicuspid extractions

Pre surgery

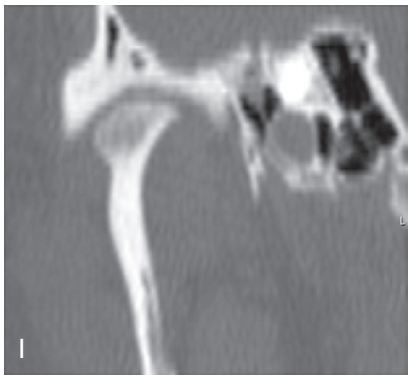
After treatment

• **Figure 36-3, cont'd E,** Occlusal views before retreatment, before surgery, and after treatment.

Continued

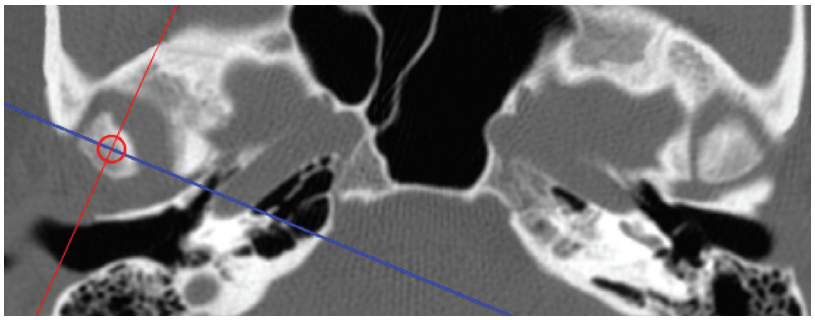


• **Figure 36-3, cont'd** **F,** Panorex view before surgery. **G,** Articulated dental casts that indicate analytic model planning. **H,** Lateral cephalometric views before and after surgery.

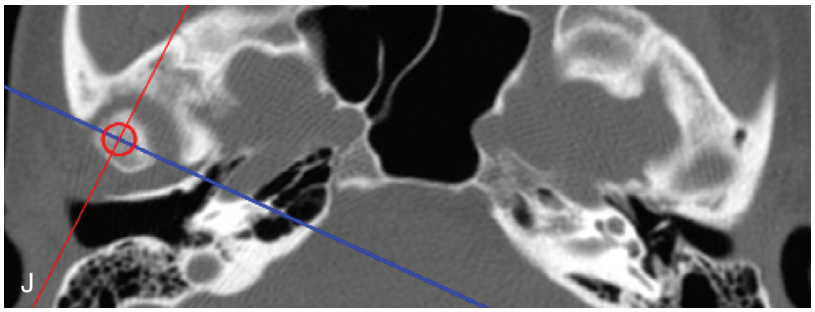


6 months prior to surgery

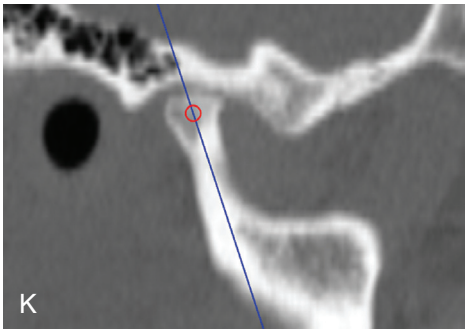
6 months after surgery



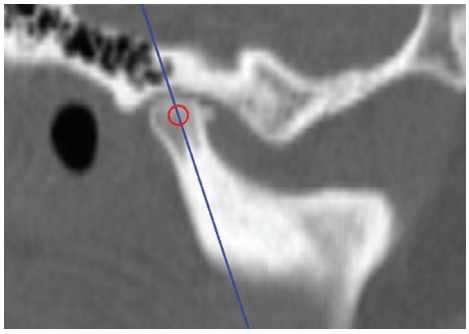
6 months prior to surgery



6 months after surgery



6 months prior to surgery



6 months after surgery

• **Figure 36-3, cont'd I,** Coronal computed tomography views of the condyle 6 months before and 6 months after surgery. **J,** Axial computed tomography views of the condyle 6 months before and 6 months after surgery. **K,** Sagittal computed tomography views of the condyle 6 months before and 6 months after surgery.

Evaluation

The patient arrived for surgical consultation when she was 21 years old having just graduated from college with the hope of achieving improved function (i.e., mastication, breathing, lip posture/closure, and speech articulation) and enhanced facial aesthetics (i.e., an improved profile and a less gummy smile). She underwent evaluations by a speech pathologist, an otolaryngologist/head and neck surgeon, an orthodontist, a restorative dentist, and a TMJ specialist.

Treatment

With successful splint therapy for the relief of masticatory muscle discomfort and a 2-year history of no progression of condylar resorption (i.e., stable occlusion), orthodontic dental decompensation was initiated. A computed tomography scan of the TMJ was obtained before surgery. The patient's surgery included the following:

- Maxillary Le Fort I osteotomy
  - Vertical intrusion at the incisors
  - Horizontal advancement at the incisors
- Bilateral sagittal split ramus osteotomies
  - Mandibular plane counterclockwise rotation
  - Horizontal advancement
- Oblique osteotomy of the chin
  - Horizontal advancement
  - Vertical shortening
- Intranasal procedures
  - Septoplasty
  - Reduction of the inferior turbinates
  - Recontouring of the nasal floor
- Removal of retained wisdom teeth

After initial surgical healing (5 weeks), finishing orthodontics continued, with the removal of the appliances 6 months postoperatively. The patient continued with preventative splint therapy and orthodontic retention.

CASE 36-4

Progressive Condylar Resorption/Idiopathic Condylar Resorption? (Fig. 36-4)

History

When she was 15 years old, the occlusion of this female high school student was characterized as having a normal overjet/overbite relationship, with mild anterior crowding. When she was 16 years of age, she experienced an episode of temporomandibular disorder that was characterized by TMJ region and masticatory muscle discomfort. This was accompanied by a painful and limited mandibular range of motion and chewing difficulties. When she was 17 years old, the TMD was successfully managed with the use of conservative measures (e.g., splint therapy, anti-inflammatory agents, and diet modifications). Mouth opening was satisfactory and without direct TMJ region pain but with an anterior open-bite skeletal Class II malocclusion. As a result of other signs and symptoms that were present, a diagnosis of chronic fatigue syndrome and fibromyalgia was made.

Evaluation

The patient arrived for surgical consultation when she was 19 years old in the hopes of achieving improved function (i.e., mastication, breathing, lip closure/posture, speech articulation, and swallowing) and enhanced facial aesthetics (i.e., smile and profile). She underwent evaluation by a speech pathologist, an otolaryngologist/head and neck surgeon, an orthodontist, a TMJ specialist, a restorative dentist, and a general physician.

Treatment

With conservative measures used to control masticatory muscle discomfort, satisfactory TMJ function, and no clinical progression of condylar resorption after 2 years of observation, orthodontic dental decompensation was initiated. No extractions were required to relieve crowding. A computed tomography scan of the TMJ was obtained 2 months before surgery. The patient's surgery included the following

- Maxillary Le Fort I osteotomy
  - Vertical intrusion
  - Horizontal advancement
  - Segmentation for arch expansion and correction of the curve of Spee
- Bilateral sagittal split ramus osteotomies
  - Mandibular plane counterclockwise rotation
  - Horizontal advancement
- Oblique osteotomy of the chin
  - Horizontal advancement
  - Vertical shortening
- Intranasal procedures
  - Septoplasty
  - Inferior turbinate reduction
  - Nasal base recontouring
- Removal of Wisdom Teeth

After surgical healing (5 weeks), finishing orthodontics continued for 9 months. With the removal of the orthodontic appliances, preventative splint therapy and orthodontic retention continued.

©Elsevier Inc.
Orthognathic Surgery; J.C. Posnick

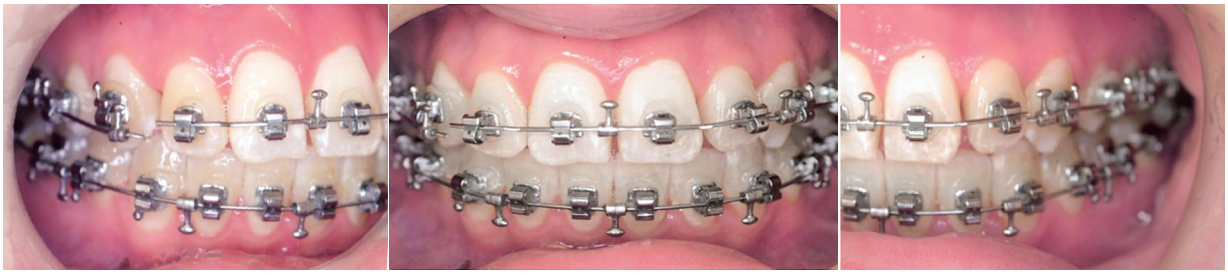
• **Figure 36-4** Patient before and after treatment. **A,** Frontal views in repose before and after surgery. **B,** Frontal views with smile before and after surgery.

Continued

• **Figure 36-4, cont'd C,** Oblique views before and after surgery. **D,** Profile views before and after surgery.

Prior to treatment

Pre surgery

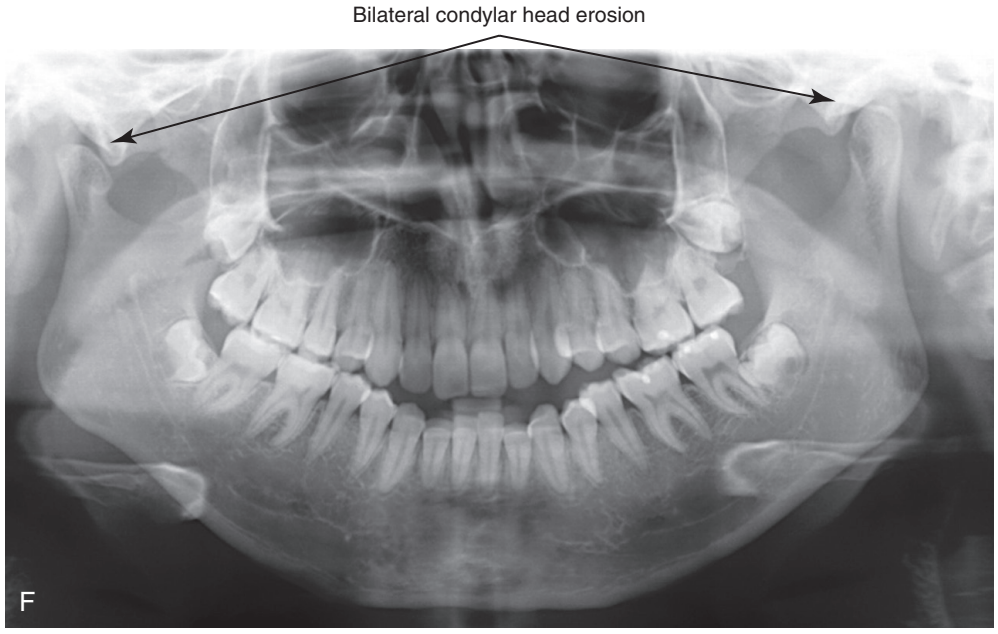


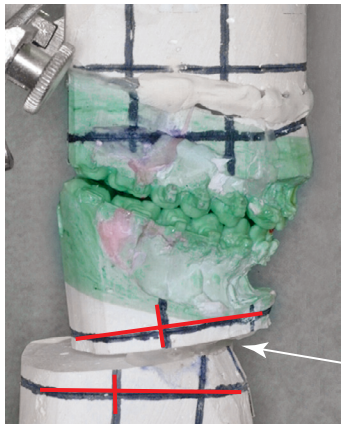
5 weeks after surgery

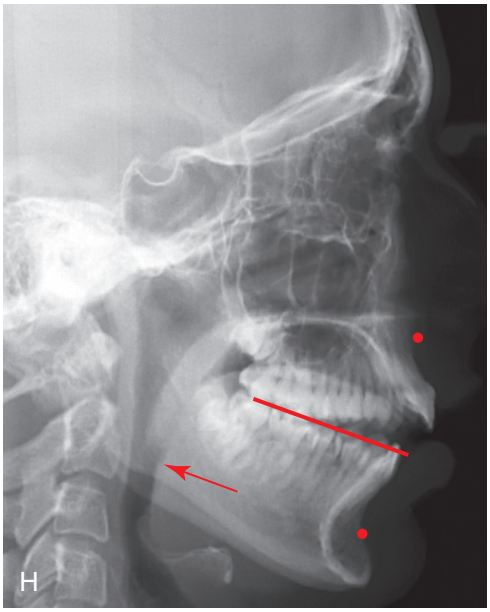
After treatment

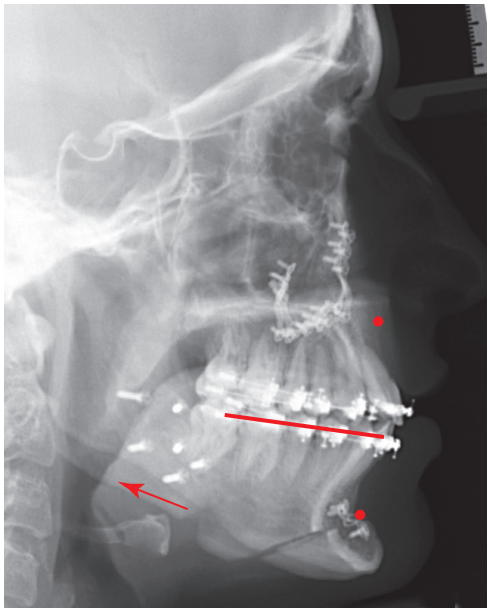
• **Figure 36-4, cont'd E,** Occlusal views before treatment, before surgery, and at 5 weeks and after treatment.

Continued

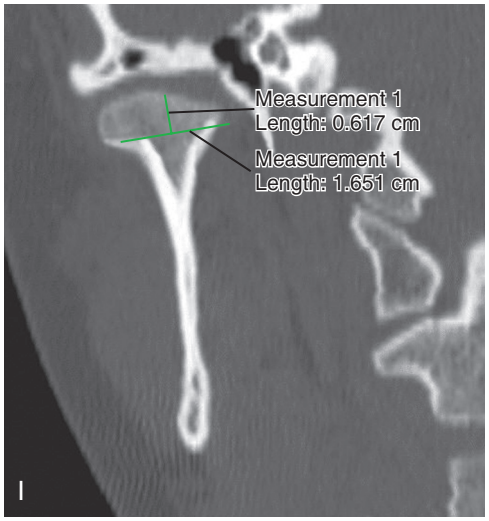






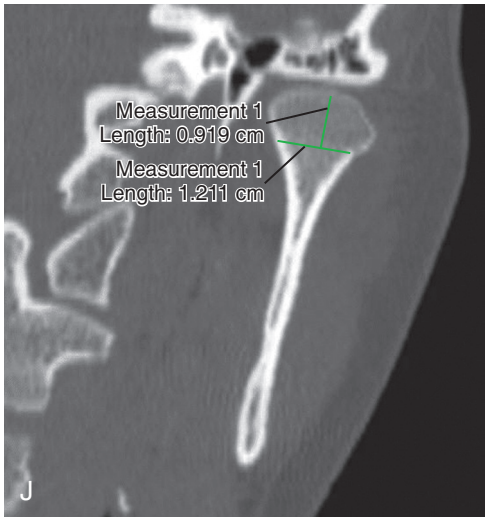


• **Figure 36-4, cont'd F,** Panorex view before surgery. **G,** Articulated dental casts that indicate analytic model planning. **H,** Lateral cephalometric views before and after surgery.

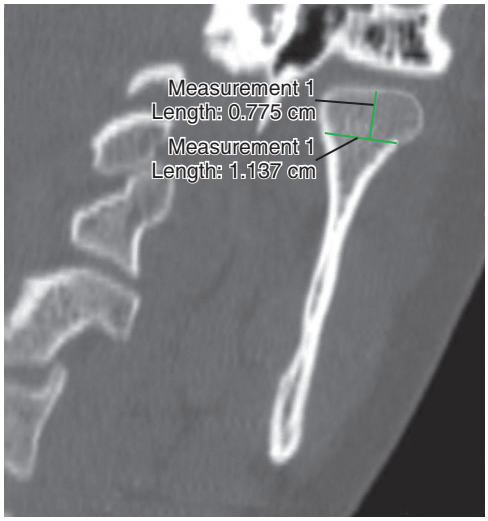


Right side 2 months pre surgery

Right side 14 months after surgery



Left side 2 months pre surgery



Left side 14 months after surgery

• **Figure 36-4, cont'd I,** Coronal computed tomography views of the condyle 2 months before and 14 months after surgery. **J,** Axial computed tomography views of the condyle 2 months before and 14 months after surgery.

CASE 36-5

Idiopathic Condylar Resorption (Fig. 36-5)

History

When she was 12 years old, she was referred by a pediatric dentist to an orthodontist for evaluation of her malocclusion. She was felt to have dental crowding, and she underwent standard orthodontic therapy (without extractions) when she was between 12 and 14 years old, after which time the appliances were removed. She was left with a mild gummy smile, satisfactory lip competence, and a stable Class I occlusion.

When she was approximately 22 years old, she experienced an episode of bilateral TMJ and masticatory muscle discomfort that affected her mandibular range of motion and her chewing ability. An insidious anterior open-bite and retrusive mandibular profile was recognized during the ensuing 6 to 12 months. The symptoms subsided, and the patient was left with a satisfactory mandibular range of motion without TMJ region discomfort. She returned to the treating orthodontist when she was 26 years old complaining of difficulty with chewing as a result of malocclusion and masticatory muscle discomfort. Radiographic examination confirmed the partial resorption of the condylar heads.

Evaluation

The patient initially arrived for surgical evaluation when she was 26 years old and requested reconstruction. The history, physical examination, and radiographic findings were consistent with ICR. Conservative management of the masticatory muscle discomfort and the confirmation of condylar stability were recommended. The patient continued with splint therapy. Over the next 12 months, a consistent occlusion with good mandibular range of motion confirmed condylar stability. As a result of her work and school schedules, the patient elected to postpone reconstruction until a future date.

She returned when she was 30 years old and again requested reconstruction. Now, with documentation of at least 5 years of no measurable progression of condylar resorption and with a negative quantitative condylar head bone scan, definitive therapy was offered. She underwent current evaluations by a speech pathologist, an otolaryngologist/head and neck surgeon, an orthodontist, a restorative dentist, and a general physician.

Treatment

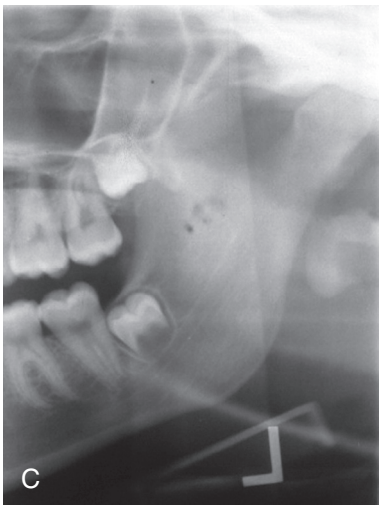
Orthodontic dental decompensation was carried out over the course of 9 months. The patient's surgery included the following:

- Maxillary Le Fort I osteotomy
  - Vertical intrusion at the incisors
  - Horizontal advancement at the incisors
  - Maxillary plane counterclockwise rotation
- Bilateral sagittal split ramus osteotomies
  - Mandibular plane counterclockwise rotation
  - Horizontal advancement
- Oblique osteotomy of the chin
  - Horizontal advancement
  - Vertical change

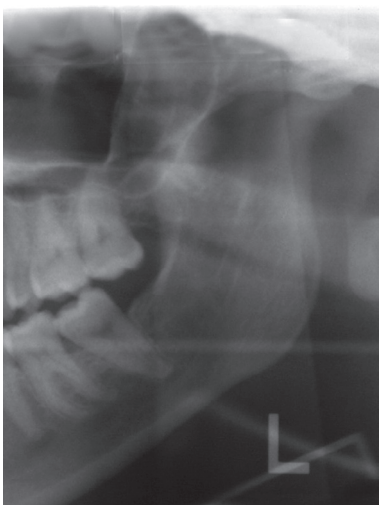
After initial surgical healing (5 weeks), finishing orthodontics continued for approximately 6 months, and orthodontic retention continued. With good relief of masticatory muscle discomfort, the patient had a limited need for splint therapy.

• **Figure 36-5** Patient before and after treatment. **A,** Frontal views at 14, 27, and 30 years of age. **B,** Profile views at 14, 27, and 30 years of age.

Continued

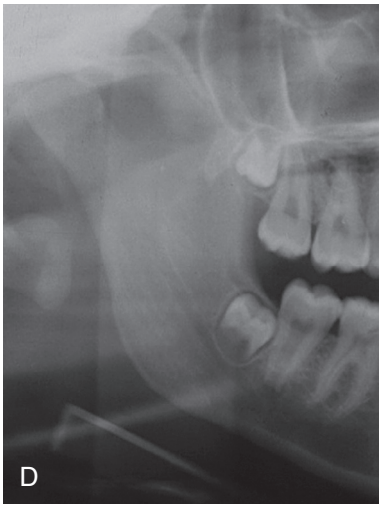


14 years of age

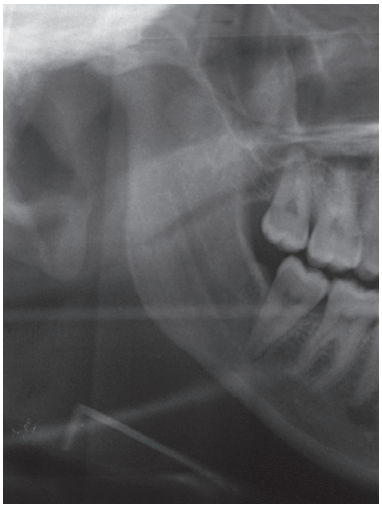


27 years of age

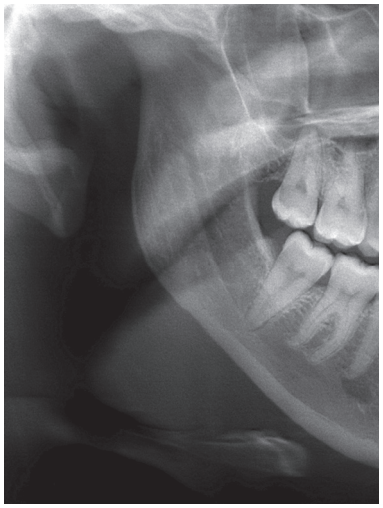
30 years of age



14 years of age



27 years of age



30 years of age

26 years of age

• **Figure 36-5, cont'd C,** Left condyle Panorex views at 14, 27, and 30 years of age. **D,** Right condyle Panorex views at 14, 27, and 30 years of age. **E,** Coronal computed tomography view of the condyles at 26 years of age.

©Elsevier Inc.
Orthognathic Surgery; J.C. Posnick

• **Figure 36-5, cont'd** **F,** Frontal views in repose before and after surgery. **G,** Frontal views with smile before and after surgery.

Continued

• **Figure 36-5, cont'd H,** Oblique views before and after surgery. **I,** Profile views before and after surgery.

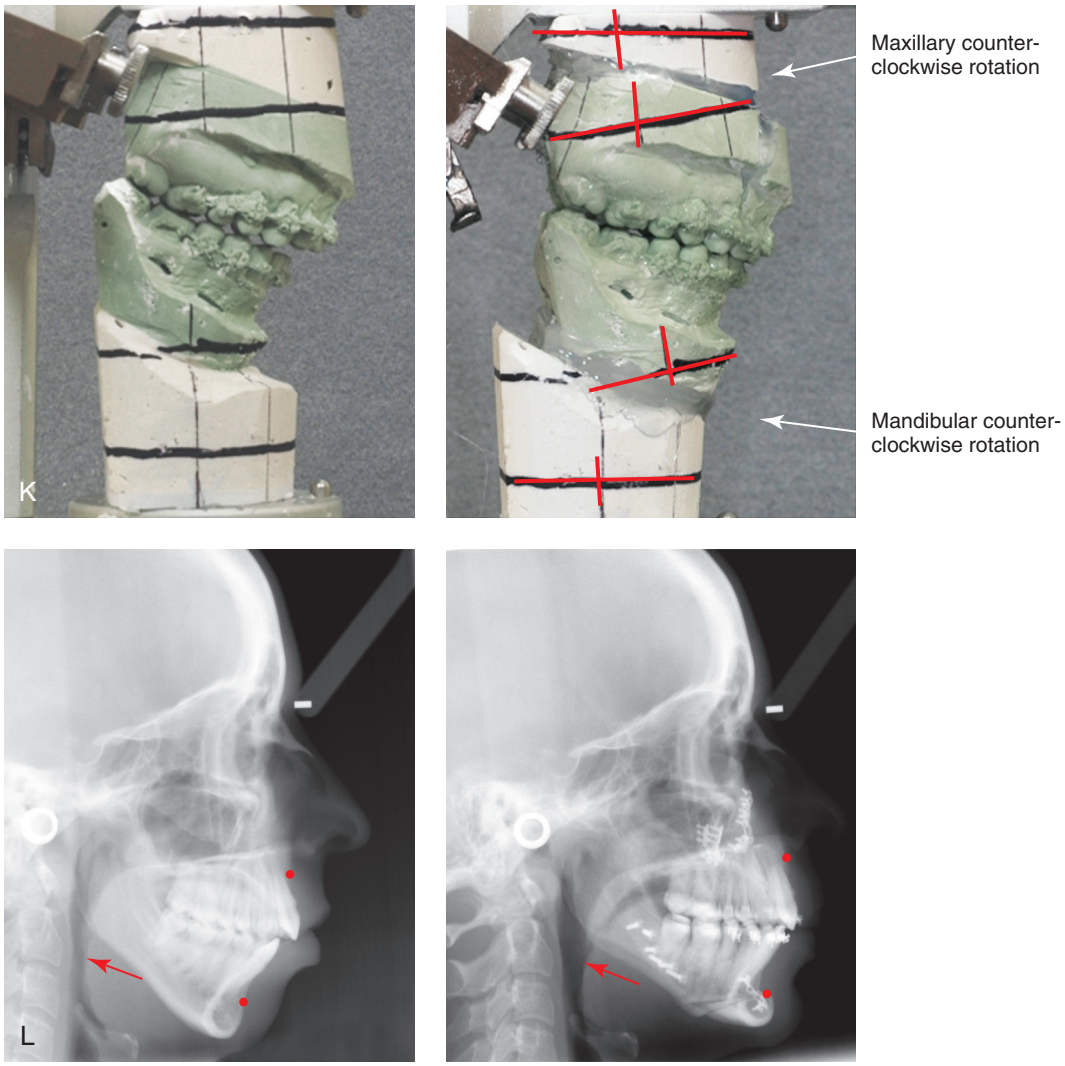
Prior to retreatment

Pre surgery

After treatment

• **Figure 36-5, cont'd J,** Occlusal views before retreatment, before surgery, and after surgery.

Continued



• **Figure 36-5, cont'd K,** Articulated dental casts that indicate analytic model planning. **L,** Lateral cephalometric views before and after surgery.

Conclusions

In patients with the condition of uncertain etiology known as ICR and in those with a known association (PCR), the condyles of the mandible partially resorb, thereby causing a loss of condylar height and secondary alterations of the maxillo-mandibular complex with effects on the occlusion, the upper airway, and the facial aesthetics. Theories of the condition's etiology continue to evolve.

When the condylysis process is believed to be "burnt out," a comprehensive approach to resolve the secondary deformities generally involves an orthodontist, an orthognathic surgeon, a TMJ specialist, and appropriate imaging studies to achieve optimal results. The keys to a favorable result for the secondary maxillo-mandibular deformities are as follows: to await the presumed remission of condylar resorption; to define the functional deficits; and to then successfully perform joint-preserving orthognathic and intranasal procedures in conjunction with orthodontic treatment as indicated. In the future, the use of disease-modifying anti-rheumatic drugs may play a prominent role in limiting condylar resorption in affected individuals.

References

1. Abubaker AO, Arslan W, Sotereanos GC: Estrogen and progesterone receptors in the temporomandibular joint disk of symptomatic and asymptomatic patients. *J Oral Maxillofac Surg* 51:1096, 1993.
2. Anderson Q: Discussion of: Mandibular retrusion, temporomandibular joint derangement, and orthognathic surgery planning, *Plast Reconstr Surg* 2:230–232, 1992.
3. Arnett GW, Milam SB, Gottesman L: Progressive condylar resorption: Host adaptive capacity factors. *Am J Orthod Dentofacial Orthop*, 1995.

4. Arnett GW, Milam SB, Gottesman L: Progressive mandibular retrusion—idiopathic condylar resorption. Part I. *Am J Orthod Dentofacial Orthop* 110:8–15, 1996.
5. Arnett GW, Milam SB, Gottesman L: Progressive mandibular retrusion—idiopathic condylar resorption. Part II. *Am J Orthod Dentofacial Orthop* 110:117–127, 1996.
6. Arnett GW, Tamborello JA: Progressive Class II development: Female idiopathic condylar resorption. *Oral Maxillofac Clin North Am* 2:699, 1990.
7. Aufdemorte TB, Van Sickels JE, Dolwick MF, et al: Estrogen receptors in the temporomandibular joint of the baboon: An autoradiographic study. *Oral Surg* 61:307, 1986.
8. Bain SD, Gross TS: Structural aspects of bone resorption. In Bronner F, Farach-Carson MC, editors: *Bone resorption*, London, UK, 2005, Springer-Verlag, pp 58–66.
9. Barrett MB, Smith PHS: Bone imaging with 99Tc in polyphosphate: A comparison with 18F and skeletal radiology. *Br J Radiol* 17:387, 1974.
10. Bergink AP, Uitterlinden AG, Van Leeuwen JP, et al: Vitamin D status, bone mineral density, and the development of radiographic osteoarthritis of the knee: The Rotterdam study. *J Clin Rheumatol* 15:230, 2009.
11. Bilodeau JE: Retreatment of a patient who presented with condylar resorption. *Am J Orthod Dentofacial Orthop* 131:897, 2007.
12. Bouwman JPB, Kerstens HCJ, Tuinzing DB: Condylar resorption in orthognathic surgery: The role of intermaxillary fixation. *Oral Surg Med Oral Pathol* 78:138–141, 1994.
13. Brennan MT, Patronas NJ, Brahim JS: Bilateral condylar resorption in dermatomyositis: A case report. *Oral Surg Oral Med Oral Path Oral Radiol Endod* 87:446, 1999.
14. Burr DB: Muscle strength, bone mass, and age-related bone loss. *J Bone Miner Res* 12:1547, 1997.
15. Censi S, Weitzmann MN, Roggia C, et al: Estrogen deficiency induces bone loss by enhancing T-cell production of TNF-alpha. *J Clin Invest* 106:1229, 2000.
16. Chuong R, Piper MA: Avascular necrosis of the mandibular condyle: Pathogenesis and concept of management. *Oral Surg Oral Med Oral Pathol* 75:428, 1993.
17. Chuong R, Piper MA, Boland TJ: Osteonecrosis of the mandibular condyle: Pathophysiology and core decompression. *Oral Surg Oral Med Oral Pathol* 79:539, 1995.
18. Cisneros G, Kaban LB: Computerized skeletal scintigraphy for assessment of mandibular asymmetry, *J Oral Maxillofac Surg* 42:513, 1985.
19. Copray JCVM, Jansen HWB, Duterloo HS: The role of biomechanical factors in mandibular condylar cartilage growth and remodeling in vitro. In McNamara JA, Jr, editor: *Craniofacial growth series. Center for Human Growth and Development*, Ann Arbor, Mich, 1984, University of Michigan Press.
20. Crawford JG, Stoelinga JW, Blijdorp PA, Brouns JJ: Stability after reoperation for progressive condylar resorption after orthognathic surgery: Report of seven cases. *J Oral Maxillofac Surg* 52:460–466, 1994.
21. Cutbirth M, Sickels JE, Thrash WJ: Condylar resorption after bicortical screw fixation of mandibular advancement. *J Oral Maxillofac Surg* 56:178–182, 1998.
22. De Clercq CA, Neyt LF, Mommaerts MY, et al: Condylar resorption in orthognathic surgery: A retrospective study. *Int J Adult Orthodon Orthognath Surg* 9:233–240, 1994.
23. Eghbali-Fatoureschi G, Khosla S, Sanyal A, et al: Role of RANK ligand in mediating increased bone resorption in early postmenopausal women. *J Clin Invest* 111:1221, 2003.
24. Ferguson JW, Luyk NH, Parr NC: A potential role for costo-chondral grafting in adults with mandibular condylar destruction secondary to rheumatoid arthritis: A case report. *J Craniomaxillofac Surg* 21:15, 1993.
25. Forwood MR, Turner CH: Skeletal adaptations to mechanical usage: Results from tibial loading studies in rats. *Bone* 17(4 Suppl):197S, 1995.
26. Frost HM: Tetracycline-based histological analysis of bone remodeling. *Calcif Tissue Res* 3:211, 1969.
27. Frost HM: The skeletal intermediary organization. *Metab Bone Dis Relat Res* 4:281, 1983.
28. Frost HM: Changing concepts in skeletal physiology: Wolff's Law, the mechanostat and the "Utah Paradigm." *J Hum Biol* 10:599, 1998.
29. Frost HM: Perspective on the estrogen-bone relationship and postmenopausal bone loss: A new model. *J Bone Miner Res* 14:1473, 1999.
30. Guler N, Yatmaz PI, Ataoglu H, et al: Temporomandibular internal derangement: Correlation of MRI findings with clinical symptoms of pain and joint sounds in patients with bruxing behavior. *Dentomaxillofac Radiol* 32:304, 2003
31. Gunson MJ, Arnett GW, Formby B, et al: Oral contraceptive pill use and abnormal menstrual cycles in women with severe condylar resorption: A case for low serum 17B-estradiol as a major factor in progressive condylar resorption. *Am J Orthod Dentofacial Orthop* 136:772–779, 2009.
32. Gunson MJ, Arnett GW, Milam SB: Pathophysiology and pharmacologic control of osseous mandibular condylar resorption. *J Oral Maxillofac Surg* 70:1918–1934, 2012.
33. Hajati AK, Alstergren P, Masstrom K, et al: Endogenous glutamate in association with inflammatory and hormonal factors modulates bone tissue resorption of the temporomandibular joint in patients with early rheumatoid arthritis. *J Oral Maxillofac Surg* 67:1895, 2009.
34. Handelman CS: Ask us: Condylar resorption. *Am J Orthod Dentofacial Orthop* 125:16A, 2004.
35. Heidari B, Hajian-Tilaki K, Heidari P: The status of serum vitamin D in patients with rheumatoid arthritis and undifferentiated inflammatory arthritis compared with controls. *Rheumatol Int* 32:991–995, 2012.
36. Heidari B, Heidari P, Hajian-Tilaki P: Association between serum vitamin D deficiency and knee osteoarthritis. *Int Orthop* 35:1627–1631, 2011.
37. Herford AS, Hoffman R, Demirdji S, et al: Comparison of synovial fluid pressure after immediate versus gradual mandibular advancement in the miniature pig. *J Oral Maxillofac Surg* 63:775, 2005.
38. Hikiji H, Takato T, Matsumoto S, et al: Experimental study of reconstruction of the temporomandibular joint using a bone transport technique. *J Oral Maxillofac Surg* 58:1270, 2000.
39. Holick MF: The vitamin D epidemic and its health consequences. *J Nutr* 135:2739S, 2005.
40. Hoppenreijs TJ, Freihofer HP, Stoelinga PJ, et al: Condylar remodeling and resorption after Le Fort I and bimaxillary osteotomies in patients with anterior open bite: A clinical and radiological study. *Int J Oral Maxillofac Surg* 27:81–91, 1998.
41. Hoppenreijs TJ, Stoelinga PJ, Grace KL, et al: Long-term evaluation of patients with progressive condylar resorption following orthognathic surgery. *Int J Oral Maxillofac Surg* 28:411, 1999.

41A. Howe CR, Gardner GC, Kadel NJ: Perioperative medication management for the patient with rheumatoid arthritis. *J Am Acad Orthop Surg* 14:544–551, 2006.

42. Hoyland JA, Mee AP, Baird P, et al: Demonstration of estrogen receptors in association with primary knee osteoarthritis in Korean population. *Arthritis Res Ther* 6:R415, 2004.
43. Huang YL, Pogrel MA, Kaban LB: Diagnosis and management of condylar resorption. *J Oral Maxillofac Surg* 55:114, 1997.
44. Hwang SJ, Haers PE, Zimmermann A, et al: Surgical risk factors for condylar resorption after orthognathic surgery. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 89:542–552, 2000.
45. Jacobs JS: Discussion of: Osteonecrosis of the temporomandibular joint: Correlation of magnetic resonance imaging and histology. *J Oral Maxillofac Surg* 57:899, 1999.
46. Jilka RL: Biology of the basic multicellular unit and the pathophysiology of osteoporosis. *Med Pediatr Oncol* 41:182, 2003.
47. Kannus P, Sievanen H, Vuori L: Physical loading, exercise and bone. *Bone* 18(Suppl 1):1, 1996.
48. Kapila S, Wang W, Uston K: Matrix metalloproteinase induction by relaxin causes cartilage matrix degradation in target

synovial joints. *Ann N Y Acad Sci* 1160:322, 2009.
49. Kato Y, Hiyama S, Kuroda T, et al: Condylar resorption 2 years following active orthodontic treatment: A case report. *Int J Adult Orthodon Orthognath Surg* 14:243, 1999.
50. Kerstens HC, Tuinzing DB, Golding RP, et al: Condylar atrophy and osteoarthrosis after bimaxillary surgery. *Oral Surg* 69:274, 1990.
51. Kusec V, Virdi AS, Prince R, et al: Localization of estrogen receptor-alpha in human and rabbit skeletal tissues. *J Clin Endrocrinol Metab* 83:2421, 1998.
52. Lane NE, Gore LR, Cummings SR, et al: Serum vitamin D levels and incident changes of radiographic hip osteoarthritis: A longitudinal study. Study of osteoporotic fractures research group. *Arthritis Rheum* 42:854, 1999.
53. Larheim TA, Westesson PL, Hicks D, et al: Osteonecrosis of the temporomandibular joint: Correlation of magnetic resonance imaging and histology. *J Oral Maxillofac Surg* 57:888, 1999.
54. McCormick SU, McCarthy JG, Grayson BH, et al: Effect of mandibular distraction on the temporomandibular joint: Part 1. Canine study. *J Craniofac Surg* 6:358, 1995.
55. Mercuri LG: A rationale for total alloplastic temporomandibular joint reconstruction in the management of idiopathic/progressive condylar resorption. *J Oral Maxillofac Surg* 65:1600–1609, 2007.
56. Mercuri LG: Osteoarthritis, osteoarthrosis, and idiopathic condylar resorption. *Oral Maxillofac Surg Clin North Am* 20:169–183, 2008.
57. Merkx MA, Van Damme PA: Condylar resorption after orthognathic surgery: Evaluation of treatment in eight patients. *J Craniomaxillofac Surg* 22:53–58, 1994.
58. Milam SB, Aufdemorte TB, Sheridan PJ, et al: Sexual dimorphism in the distribution of estrogen receptors in the temporomandibular joint complex of the baboon. *Oral Surg Oral Med Oral Pathol* 64:527, 1987.
59. Milam SB: Pathogenesis of degenerative temporomandibular joint arthritides. *Odontology* 93:7–15, 2005.
60. Mongini F: Anatomic and clinical evaluation of the relationship between the temporomandibular joint and occlusion. *J Prosthet Dent* 38:539, 1977.
61. Moore K, Gooris P, Stoelonga P: The contributing role of condylar resorption to skeletal relapse following mandibular advancement surgery: Report of five cases. *J Oral Maxillofac Surg* 49:448, 1991.
62. Morales-Ryan CA, Garcia-Morales P, Wolford LM: Idiopathic condylar resorption: Outcome assessment of TMJ disc repositioning and orthognathic surgery. *J Oral Maxillofac Surg* 60(Suppl 1):53, 2002.
63. Moseley JR, Lanyon LE: Strain rate as a controlling influence on adaptive remodeling in response to dynamic loading of the ulna in growing male rats. *Bone* 23:313, 1998.
64. Nanes MS, Pacifici R: Inflammatory cytokines. In Bronner F, Farach-Carson MC, Rubin J, editors: *Bone resorption*, London, UK, 2005, Springer-Verlag, pp 67–90.
65. Okuda T, Yasuoka T, Nakashima M, et al: The effect of ovariectomy on the temporomandibular joints of growing rats. *J Oral Maxillofac Surg* 54:1202, 1996.
66. O'Ryan F, Epker BN: Temporomandibular joint function and morphology: Observations on the spectra of normalcy. *Oral Surg Oral Med Oral Pathol* 58:272, 1984.
67. Papadaki ME, Tayebaty F, Kaban LB, Troulis MJ: Condylar resorption. *Oral Maxillofac Surg Clin North Am* 19:223–234, 2007.
68. Papageorge MB, Apostolidis C: Simultaneous mandibular distraction and arthroplasty in a patient with temporomandibular joint ankylosis and mandibular hypoplasia. *J Oral Maxillofac Surg* 57:328, 1999.
69. Parfitt AM: The coupling of bone formation to bone resorption: A critical analysis of the concept and its relevance to the pathogenesis of osteoporosis. *Metab Bone Dis Relat Res* 4:1, 1982.
70. Parfitt AM: The cellular basis for bone remodeling: The quantum concept reexamined in light of recent advances in the cell biology of bone. *Calcif Tissue Int* 36:S37, 1984.
71. Phillips RM, Bell WH: Atrophy of mandibular condyles after sagittal ramus split osteotomy: Report of a case. *J Oral Maxillofac Surg* 36:45, 1978.
72. Piper MA: Microscope disk preservation surgery of the temporomandibular joint. *Oral Maxillofac Clin North Am* 1:279, 1989.
73. Pogrel MA, Kopf J, Dodson TB, et al: A comparison of single photon emission computed tomography and planar imaging for quantitative skeletal scintigraphy of the mandibular condyle. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 80:226, 1995.
74. Posnick J, Fantuzzo J, Orchin J: Deliberate operative rotation of the maxillo-mandibular complex to alter the A-point to B-point relationship for enhanced facial esthetics. *J Oral Maxillofac Surg* 64:1687–1695, 2006.
75. Posnick JC: (Discussion of) Occlusal plane rotation: Aesthetic enhancement in mandibular micrognathia. *Plast Reconstr Surg* 91:1241, 1993.
76. Posnick JC, Fantuzzo JJ: Idiopathic condylar resorption: Current clinical perspective. *J Oral Maxillofac Surg* 65(8):1617–1623, 2007.
77. Rosen HM: Occlusal plane rotation: Aesthetic enhancement in mandibular micrognathia. *Plast Reconstr Surg* 91:1231, 1993.
78. Roser SM, Mena I: Diphosphonate dynamic imaging of experimental bone grafts and soft tissue injury. *Int J Oral Surg* 7:488, 1978.
79. Salem ML, Hossain MS, Nomoto K: Mediation of the immunomodulatory effect of beta-estradiol on inflammatory responses by inhibition of recruitment and activation of inflammatory cells and their gene expression of TNF-alpha and IFN-gamma. *Int Arch Allergy Immunol* 121:235, 2000.
80. Sano H, Arai K, Murai T, et al: Tight control is important in patients with rheumatoid arthritis treated with an anti-tumor necrosis factor biological agent: Prospective study of 91 cases who used a biological agent for more than 1 year. *Mod Rheumatol* 19:390, 2009
81. Schellhas KP, Piper MA, Bessette R, et al: Mandibular retrusion, temporomandibular joint derangement, and orthognathic surgery planning. *Plast Reconstr Surg* 2:218, 1992.
82. Schellhas KP, Piper MA, Omlie MR: Facial skeletal remodeling due to temporomandibular joint degeneration: An imaging study of 100 patients. *AJR Am J Roentgenol* 155:373–383, 1990.
83. Schellhas KP, Pollei SR, Wilkes CH: Pediatric internal derangements of the temporomandibular joint: Effect on facial development. *Am J Orthod Dentofacial Orthop* 104:52, 1993.
84. Schellhas KP, Wilkes CH, Fritts HM, et al: MR of osteochondritis dissecans and avascular necrosis of the mandibular condyle. *AJR Am J Roentgenol* 152:551–560, 1989.
85. Schendel S, Tulasne JF, Linck DW: Idiopathic condylar resorption and micrognathia: The case for distraction osteogenesis. *J Oral Maxillofac Surg* 65:1610–1616, 2007.
86. Schiessl H, Frost HM, Jee WSS: Perspectives: Estrogen and bone-muscle strength and mass relationships. *Bone* 22:1, 1998.
87. Schiffman E, Fricton JR: Epidemiology of TMJ and craniofacial pain. In Fricton JR, Hathaway KM, editors: *TMJ and craniofacial pain: Diagnosis and management*, St. Louis, 1988, IEA.
88. Sesenna E, Raffaini M: Bilateral condylar atrophy after combined osteotomy for correction of retrusion. A case report. *J Maxillofac Surg* 13:263, 1985.
89. Simopoulos AP: Omega-3 fatty acids in inflammation and autoimmune diseases. *J Am Coll Nutr* 21:495–505, 2002
90. Stevenson JS, Bright RW, Dunson GL, et al: Technetium 99m phosphate bone imaging: A method for bone graft healing. *Radiology* 110:391, 1974.
91. Stucki-McCormick SU, Fox RM, Mizrahi RD: Reconstruction of a neocondyle using transport distraction osteogenesis. *Semin Orthod* 5:59, 1999.
92. Svensson BG, Adell R: Costochondral grafts to replace mandibular condyles in juvenile chronic arthritis patients: Long-term effects on facial growth. *J Craniomaxillofac Surg* 26:275–285, 1998.
93. Svensson BG, Feldmann G, Rindler A: Early surgical-orthodontic treatment of mandibular hypoplasia in juvenile chronic arthritis. *J Craniomaxillofac Surg* 21:67–75, 1993.

94. Thurmuller P, Troulis MJ, Rosenberg A, et al: Changes in the condyle and disc in response to distraction osteogenesis of the minipig mandible. *J Oral Maxillofac Surg* 60:1327, 2002.
95. Thurmuller P, Troulis MJ, Rosenberg A, et al: Microscopic changes in the condyle and disc in response to distraction osteogenesis of the minipig mandible. *J Oral Maxillofac Surg* 64:249, 2005.
96. Troulis MJ, Tayebaty FT, Papadaki M, et al: Condylectomy and costochondral graft reconstruction for treatment of active idiopathic condylar resorption. *J Oral Maxillofac Surg* 66:65–72, 2008.
97. Troulis MJ, Williams WB, Kaban LB: Endoscopic condylectomy and costochondral graft reconstruction of the ramus condyle unit. *J Oral Maxillofac Surg* 61:63, 2003.
98. Troulis MJ, Williams WB, Kaban LB: Endoscopic mandibular condylectomy and reconstruction: Early clinical result. *J Oral Maxillofac Surg* 62:460, 2004.
99. Tsai CL, Liu TK, Chen TJ: Estrogen and osteoarthritis: A study of synovial estradiol and estradiol receptor binding in human osteoarthritic knees. *Biochem Biophys Res Commun* 183:1287, 1992.
100. Tuinzing DB: Diagnosis and management of condylar resorption [discussion]. *J Oral Maxillofac Surg* 55:119, 1997.
101. Umemura Y, Ishiko T, Yamaguchi M, et al: Five jumps per day increase bone mass and breaking force in rats. *J Bone Miner Res* 12:1480, 1997.
102. Van Strijen PJ, Breuning KH, Becking AG, et al: Condylar resorption following distraction osteogenesis: A case report. *J Oral Maxillofac Surg* 59:1104, 2001.
103. Vidra MA, Rozema FR, Kostense PJ, et al: Observer consistency in radiographic assessment of condylar resorption. *Oral Surg Oral Med Oral Path Oral Radiol Endod* 93:399, 2002.
104. Wanman A: Longitudinal course of symptoms of craniomandibular disorders in men and woman: A 10-year followup study of an epidemiologic sample. *Acta Odontol Scand* 54:337–342, 1996.
105. Weiss JE, Ilowite NT: Juvenile Idiopathic Arthritis. Pediatr Clin N Am 52:413–442, 2005.
106. Will LA, West RA: Factors influencing the stability of the sagittal split osteotomy for mandibular advancement. *J Oral Maxillofac Surg* 47:813, 1989.
107. Wolford LM: Concomitant temporomandibular joint and orthognathic surgery. *J Oral Maxillofac Surg* 61:1198–1204, 2003.
108. Wolford LM, Cardenas L: Idiopathic condylar resorption: Diagnosis, treatment protocol, and outcomes. *Am J Orthodon Dentofacial Orthop* 116:667–677, 1999.
109. Wolford LM, Chemallo PD, Hilliard FW: Occlusal plane alteration in orthognathic surgery. *J Oral Maxillofac Surg* 51:730, 1993.
110. Wolford LM, Cottrell DA, Henry CH: Temporomandibular joint reconstruction of the complex patient with the Techmedica custom-made total joint prosthesis. *J Oral Maxillofac Surg* 52:2–10, 1994.
111. Wronski TJ, Dann LM, Scott KS, et al: Endocrine and pharmacological suppressors of bone turnover protect against osteopenia in rats. *Endocrinology* 125:810, 1989.
112. Yamada K, Hanada K, Fukui T, et al: Condylar bony change and self-reported parafunctional habits in prospective orthognathic surgery patients with temporomandibular disorders. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 92:265, 2001.
113. Yasuoka T, Nakashima M, Okuda T, et al: Effect of estrogen replacement on temporomandibular joint remodeling in ovariectomized rats. *J Oral Maxillofac Surg* 58:189, 2000.

37

Aesthetic Alteration of the Chin: Evaluation and Surgery

JEFFREY C. POSNICK, DMD, MD

- Anatomy of the Chin
- Aesthetics of the Chin
- Biologic Foundation of Osseous Genioplasty
- Frequent Patterns of Chin Deformities
- Osseous Genioplasty: Surgical Technique
- Achieving Favorable Chin Aesthetics
- Avoiding Pitfalls during Chin Surgery
- Complications and Unfavorable Results
- Alloplastic Chin Augmentation
- Conclusions
- Patient Education Materials

Hugo Obwegeser introduced the intraoral approach to completing an osseous genioplasty in 1957.[74] More than half a century later, this relatively simple technique still remains underused when surgeons are asked to make cosmetic changes to the chins of their patients.

Anatomy of the Chin

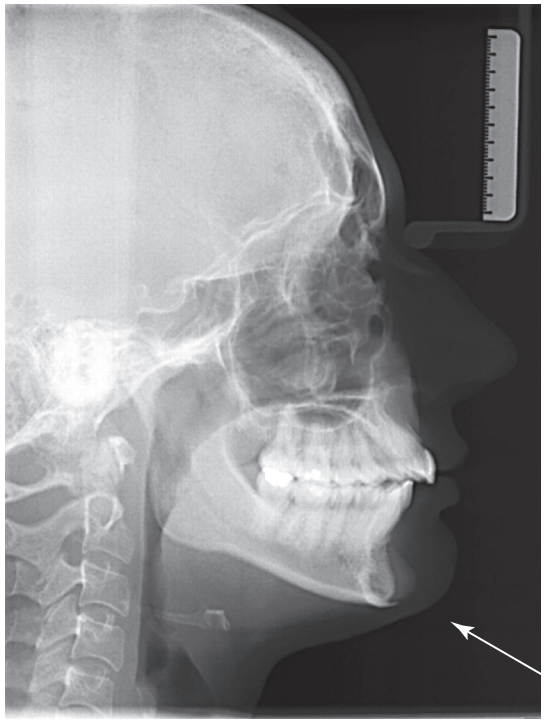
The word *chin* describes a region of the face that includes both soft tissue and bone.[79,125] The soft-tissue portion encompasses the labial mental fold superiorly, the oral commissures laterally (just anterior to the jowls), and the submental–cervical crease inferiorly. The bony portion of the chin encompasses the mandibular symphyseal region. It includes the region inferior to the root apices of the anterior teeth and the area in front of and below the mental foramen on each side. Within the soft tissues of the chin are found a series of paired muscles that include the mentalis, the quadratis labii inferioris, the triangularis, and the superior portions of the platysma that extend over the symphysis region.[22] Attached to the genial tubercle of the chin posteriorly are the tendinous attachments of the geniohyoid and genioglossus muscles.[66] The anterior bellies of the digastric muscles are attached to the posterior portion of the inferior border of the symphysis. The sensory innervation to the cutaneous chin, the lower lip, and the inner vestibule is derived from the mental nerve on each side. The motor innervation to the muscles within the soft tissues of the chin occurs through the marginal mandibular and cervical branches of the facial nerve.

Aesthetics of the Chin

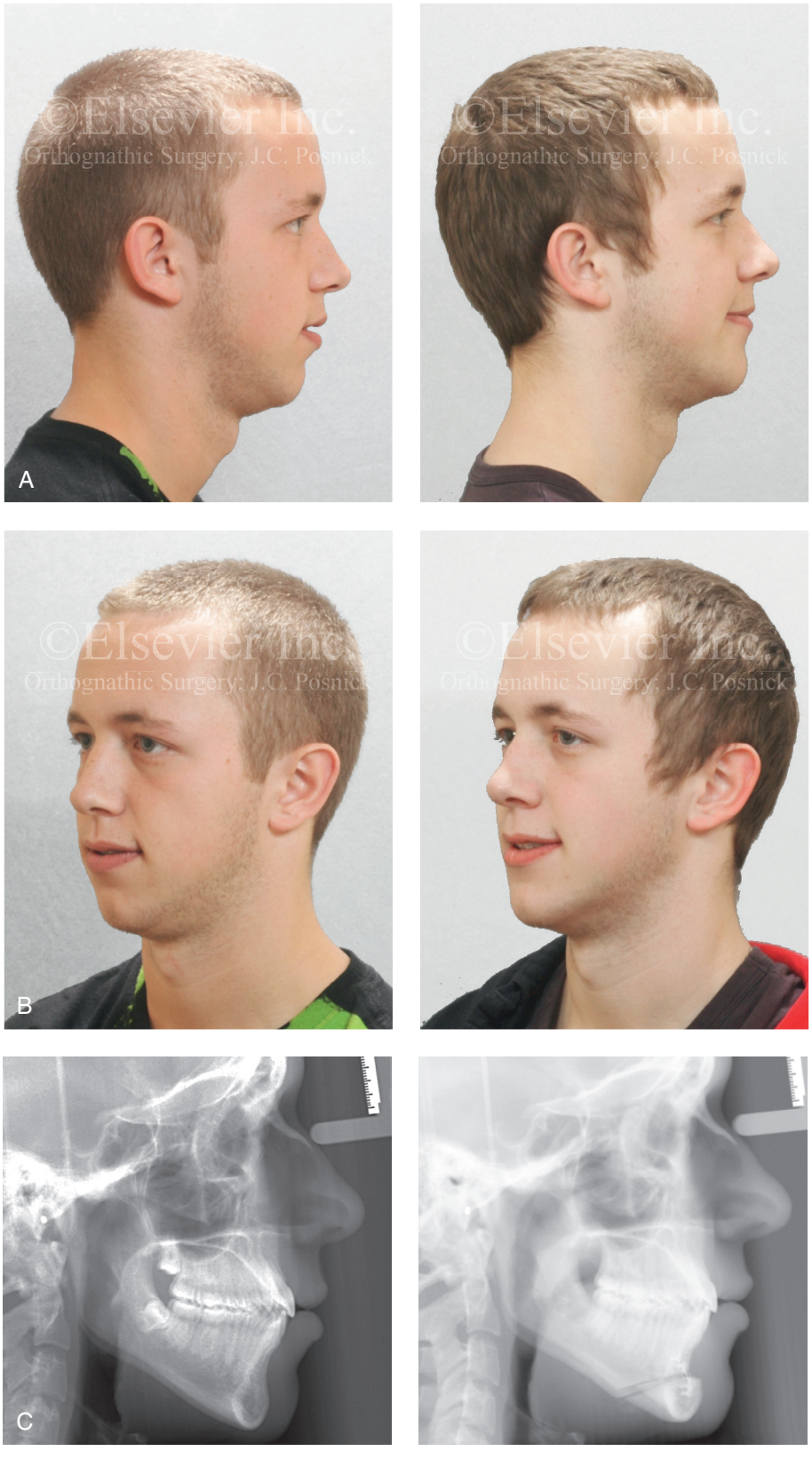
In Western society, a "weak" or deficient chin is associated with timidity, indecision, femininity, a lack of athleticism, and shy behavior. Conversely, a "strong" or forwardly projecting chin is associated with athleticism, aggressiveness, decisiveness, and bold behavior. The fact that an average adult from any number of ethnic backgrounds and nationalities from around the world will frequently characterize a chin as either "weak" or "strong" demonstrates the subliminal messages communicated about the character of a person as a result of the presenting chin morphology. When people speak about chin contour, emphasis is generally placed on the profile view (Fig. 37-1).[2,13,90,91] Although it is true that a retrusive pogonion is a frequently mentioned concern, the chin may also be asymmetric or out of the normal range in more than one dimension.

When it is taken with the patient in the natural head position, with the lips relaxed (i.e., in repose) and with the lower jaw at rest (i.e., the condyles seated in the fossa and with a normal freeway space between the teeth), a lateral cephalometric radiograph provides an accurate image of the facial skeleton and the overlying soft tissues from which to judge profile aesthetics.[28,30,31,43,44,83,105,112] When viewing either a full-face lateral photograph or a soft-tissue lateral cephalometric radiograph taken in this way, a vertical line (i.e., perpendicular to the floor with the patient in the natural head position) can be drawn through the subnasal region to assess the relative prominence of the nose, lips, and chin.[36] Ideally, the chin should lie on this line, with the lips slightly anterior (Figs. 37-2 through 37-5).[9] It is also true that there are many complex interrelationships

Text continued on p. 1571



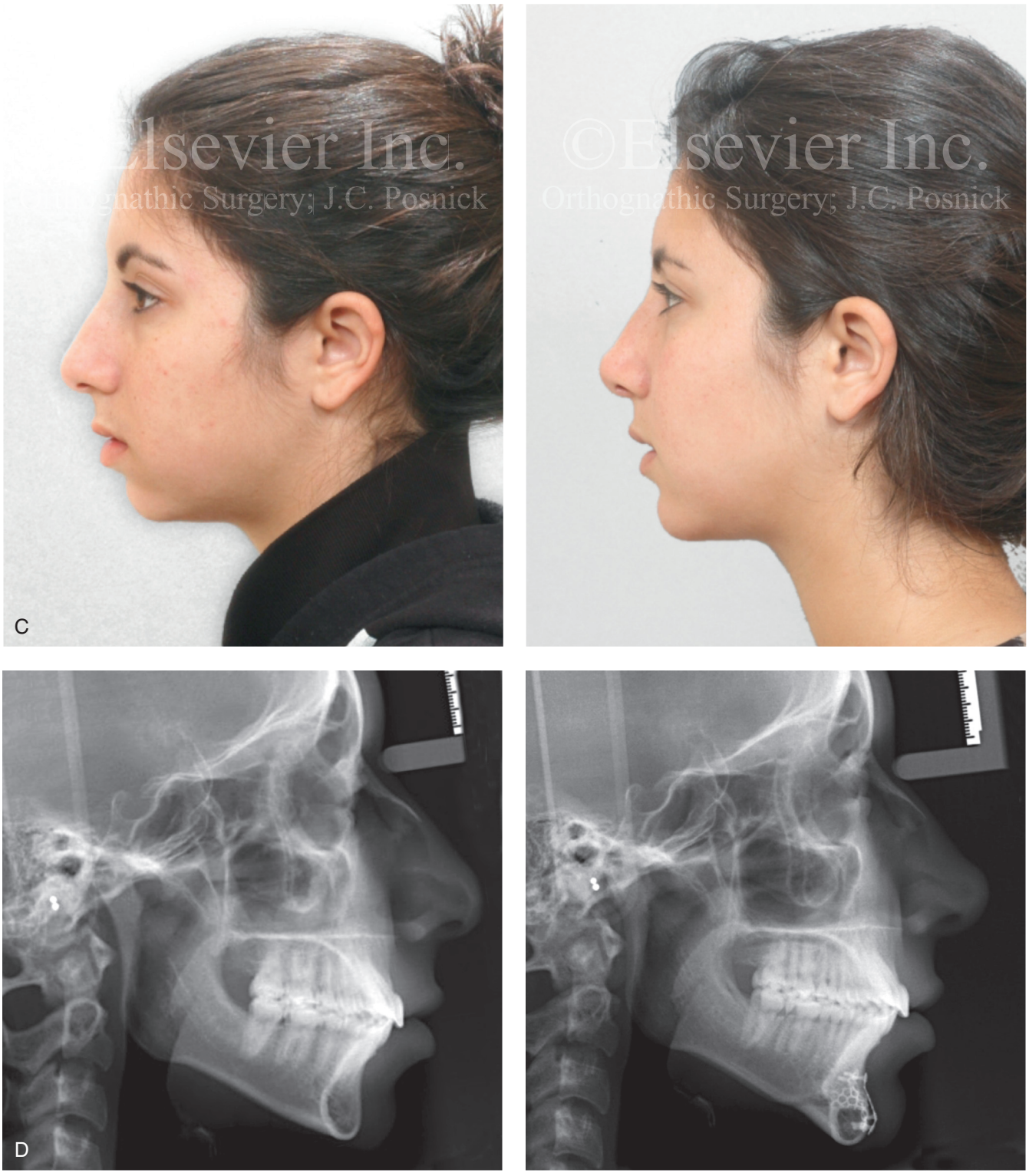
• **Figure 37-1** A 29-year-old woman arrived to discuss her chin aesthetics. During the mixed dentition, she was recognized as having mandibular deficiency and a constricted maxillary arch width. At that time, she underwent growth-modification techniques, including the use of headgear. She also underwent rapid palatal expansion followed by compensatory orthodontic mechanics to neutralize the occlusion, but this resulted in proclination of the incisors. A retrusive profile and a dual bite remained. She recently underwent the placement of an extended silastic chin implant but without fixation by another surgeon. Aesthetic modification of the anterior maxillary dentition was also completed by a restorative dentist. She is displeased with the cosmetic facial results of a deepened labiomental fold, a mobile chin implant, and a "button" appearance during lip closure. There is a dual bite with Class II malocclusion in centric relation. **A,** Oblique and frontal facial views after chin implant placement. **B,** Profile facial view and lateral cephalometric radiograph 6 months after chin implant placement.



• **Figure 37-2** A 17-year-old boy arrived with his father for the evaluation of a "weak chin" and requested chin augmentation. He had undergone orthodontic treatment with the establishment of a satisfactory occlusion that required minimal dental compensation. He agreed to undergo osseous genioplasty (horizontal advancement) to resolve the chief complaint. **A,** Profile views before and after reconstruction. **B,** Oblique facial views before and after reconstruction. **C,** Lateral cephalometric radiographs before and after reconstruction.

• **Figure 37-3** A 16-year-old girl arrived with her mother and requested a cosmetic rhinoplasty to reduce the dorsal hump and to improve the drooped nasal tip seen in profile. She also had a lifelong history of nasal obstruction. Evaluation confirmed the aesthetic nasal needs as well as sagittal deficiency of the chin. She agreed to undergo septorhinoplasty and osseous genioplasty. The rhinoplasty included nasal osteotomies (in-fracture); dorsal reduction (bone and cartilage) and tip maneuvers (modification of lower lateral cartilages and septal cartilage caudal strut grafting); septoplasty; and the reduction of the inferior turbinates. Osseous genioplasty included horizontal advancement. **A,** Profile facial views before and after reconstruction. **B,** Oblique facial views before and after reconstruction.

• **Figure 37-4** A 17-year-old girl arrived with her mother and requested a cosmetic rhinoplasty to reduce the dorsal hump and to improve the drooped nasal tip seen in profile. She also had a lifelong history of nasal obstruction. Evaluation confirmed the aesthetic nasal needs as well as sagittal and vertical deficiency of the chin. She agreed to undergo septorhinoplasty and osseous genioplasty. The rhinoplasty include nasal osteotomies (in-fracture); dorsal reduction (bone and cartilage) and tip maneuvers (modification of lower lateral cartilages and septal cartilage caudal strut grafting); septoplasty; and the reduction of the inferior turbinates. Osseous genioplasty included horizontal advancement and vertical lengthening with interpositional grafting (bloc hydroxyapatite). **A,** Frontal views in repose before and after reconstruction. **B,** Oblique facial views before and after reconstruction.



• **Figure 37-4, cont'd** **C,** Profile facial views before and after reconstruction. **D,** Lateral cephalometric radiographs before and after surgery.

• **Figure 37-5** An 18-year-old girl arrived with her parents and requested a cosmetic rhinoplasty and a chin implant. She hoped for a smaller nose, a reduction of the dorsal hump, and a stronger profile. She also had a history of nasal obstruction after a sports injury earlier during her life. Examination confirmed mild sagittal deficiency of the mandible but with a functional occlusion and minimal dental compensation after earlier orthodontic treatment. She agreed to undergo septorhinoplasty and osseous genioplasty. The patient's nasal procedures included nasal osteotomies (in-fracture); dorsal reduction (bone and cartilage); tip maneuvers (modification of lower lateral cartilages and septal cartilage caudal strut grafting); septoplasty; the reduction of the inferior turbinates; and osseous genioplasty with horizontal advancement. **A,** Frontal views in repose before and 8 years after reconstruction. **B,** Oblique facial views before and 8 years after reconstruction.

• **Figure 37-5, cont'd C,** Profile views before and 8 years after reconstruction. **D,** Lateral cephalometric radiographs before and 8 years after reconstruction.

among the facial structures that should be taken into consideration.[1-3,51,52,60,69,95,118] Critical decisions with regard to where to position the chin through osseous genioplasty are made when viewing the patient "face to face" from varying perspectives in repose and during broad smile in the clinic setting.[4,16,124,126]

Biologic Foundation of Osseous Genioplasty

When carrying out an osseous genioplasty, a horizontal osteotomy below the roots of the anterior teeth and the mental foramen on either side is completed. After osteotomy, the distal segment of the chin remains attached to a musculoperiosteal pedicle. Healing is optimal when the circulation of blood to bone and its enveloping soft tissue is continuously maintained. In 1988, Bell and colleagues completed microangiographic and histologic studies in adult rhesus monkeys that indicated that an intraoral pedicled flap that involved an osteotomy of the inferior mandibular border maintains circulation and osseous viability after manipulation and repositioning of the chin segment.[106] According to Bell's experimental work, circulation to the dental pulp should not be discernibly affected during the process.

Frequent Patterns of Chin Deformities

Horizontal Chin Deficiency with or without Vertical Discrepancy

Contemporary maxillofacial surgeons now recognize that individuals who are seeking a "stronger chin" (i.e., horizontal

projection) frequently also have disproportions in the vertical dimension of their face.[23,33,45,59,61,77,88,89,92,116,120,123,127,131] Studies suggest that approximately 40% of individuals with horizontal (sagittal) chin deficiency have a decreased lower anterior facial height (i.e., short face growth pattern) (Figs. 37-4, 37-6, and 37-7). It is also known that another 25% of these will have the other extreme of an increased lower anterior facial height (i.e., long face growth pattern) (Figs. 37-8 through 37-11). In either case, the bony chin dysmorphology will also result in soft-tissue abnormalities (distortions) of the labiomental fold. This often includes a deep labiomental angle in those with a short face growth pattern and a flat labiomental fold in those with a long face growth pattern. Fifty percent of individuals with a horizontally deficient chin will have an Angle class II malocclusion, which is indicative of mandibular retrusion. Forty percent will have previously undergone orthodontic camouflage treatment in an attempt to mask their micrognathia (see Fig. 37-1). Incisor flaring will have resulted from the orthodontic camouflage approach that attempted to neutralize a Class II excess overjet malocclusion as an alternative to a more biologic correction of the mandibular deficiency through orthognathic surgery. Some of these individuals will have also undergone chin implant augmentation, often with a suboptimal result (see Fig. 37-1, 37-7, and 37-9).

A pleasing labiomental fold is an essential component of optimal lower face aesthetics. A strong correlation exists between the lower anterior facial height and the labiomental fold morphology. Individuals with decreased lower anterior facial height in combination with a retrusive-appearing chin tend to have an exaggerated and excessively deep labiomental fold with acute angulation between the lower lip and the soft-tissue chin pad (i.e. primary mandibular deficiency or maxillomandibular deficiency; see Figs. 37-4, 37-6, and 37-7). Individuals with increased lower anterior facial height in combination with a horizontally deficient chin are likely to have a labiomental fold that is flattened and effaced, with poor fold definition, excess mentalis strain, and difficult lip closure (i.e., long face growth pattern). The long face growth pattern classically consists of vertical excess of the maxillary and mandibular alveolar regions. This is combined with mandibular retrognathia and an anterior open-bite Class II malocclusion (see Figs. 37-8 and 37-9).

Rosen and others have pointed out that many individuals with mandibular deficiency and decreased lower anterior facial height who have a functional occlusion are unwilling to undergo an orthognathic correction and may opt for a compromise approach to at least achieve some aesthetic advantage (Figs. 37-12 through 37-17).[88-93] An osseous genioplasty to simultaneously vertically lengthen and horizontally advance the chin will generally achieve a degree of improved lower facial height and chin projection while reducing the deep labiomental fold. When this compromise is undertaken, no improvement in any baseline malocclusion or any significant opening of the airway should be expected. From an aesthetic perspective, it is essential to prevent an operated look when choosing a chin-only approach. This aesthetic error can be limited by avoiding the temptation to fully correct a moderate to severe mandibular deficiency through an isolated chin procedure (see Figs. 37-13 and 37-15).

In the individual with a functional occlusion, a retrusive chin and an increased lower anterior facial height (i.e., long face growth pattern), the surgical camouflage approach will be different than what is used for the patient with a short face growth pattern, as described previously. As an aesthetic compromise, it will be an advantage to vertically shorten and horizontally advance the chin (see Fig. 40-9). This will at least marginally decrease the mentalis strain to improve the ease of lip closure and also to enhance the appearance of the lower face (see Fig. 37-11).

Note: There are some patients with the long face growth pattern who do not have a long chin as part of their dysmorphology. These individuals do not have a flat labiomental fold, and they should not undergo a camouflage chin-only procedure consisting of horizontal advancement and vertical shortening as described previously.

To review, when evaluating a "weak" profile, it is important to consider not only the horizontal (sagittal) position of the chin but also the vertical dimension (excess or deficiency). In addition, it is essential to analyze the maxillomandibular proportions of the face (i.e., any existing jaw deformity) and the alignment of the teeth in the alveolar bone (e.g., dental compensation such as incisor flaring). Only after all facial and dental aspects are evaluated will the ideal treatment be understood and any reasonable compromise options be rationally considered in the light of their shortcomings.

Horizontal Chin Prominence and Associated Vertical Discrepancy

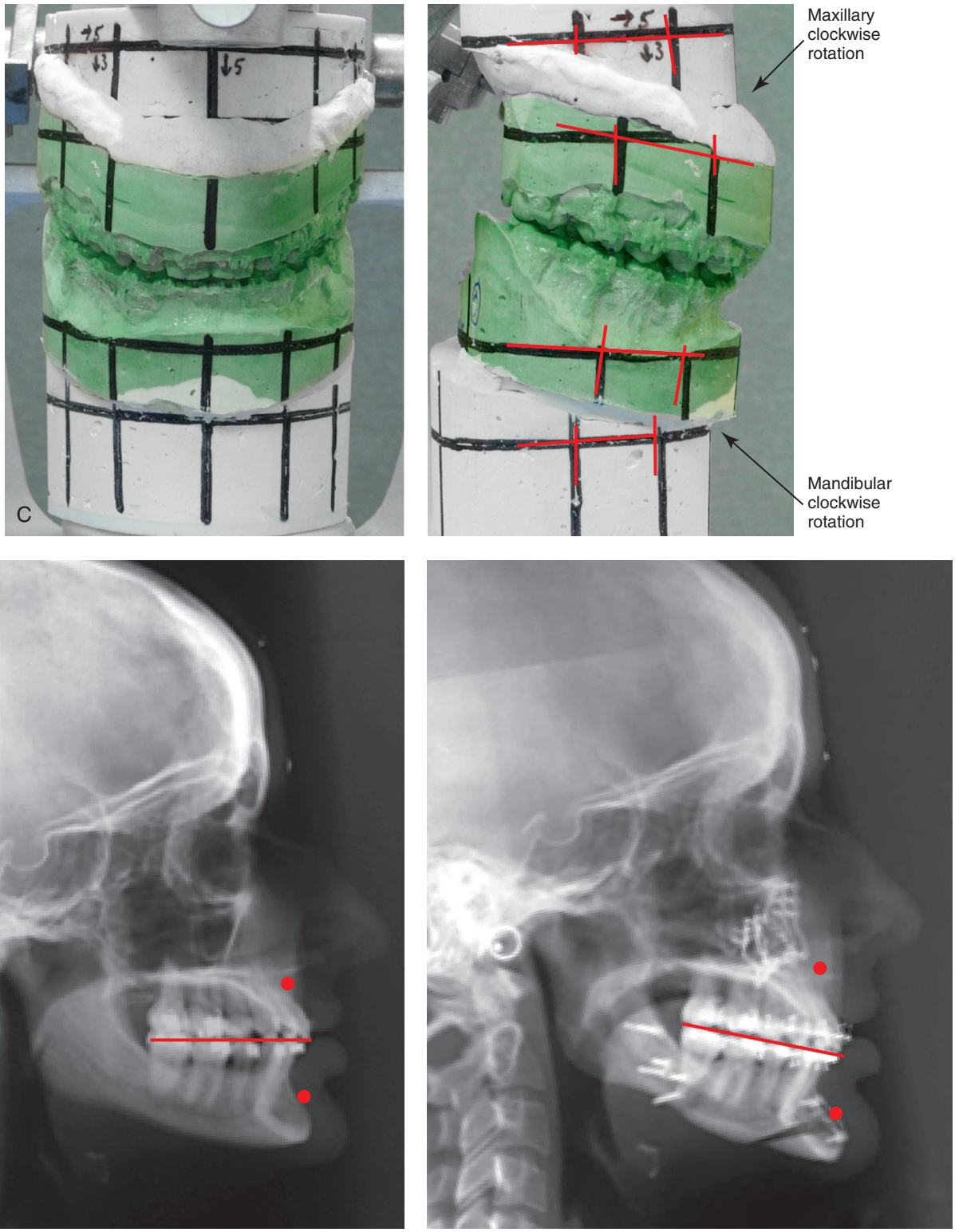
Individuals who present with a prominent pogonion are likely to have disproportions of the lower anterior facial height (i.e., the vertical dimension), which in turn affect labiomental fold morphology.[6,64,99,135] When an individual presents with a prominent pogonion and asks for a chin reduction only, he or she will likely not be pleased with the aesthetic result if this limited request is honored.

A commonly seen pattern of jaw deformity is maxillary deficiency in combination with relative mandibular excess (i.e., skeletal Class III growth pattern; see Chapter 20). This often presents in association with a vertically long (i.e., as measured in millimeters from the mandibular incisor edge to the menton height) and protrusive (i.e. forwardly projected) chin (Fig. 37-18). These individuals generally benefit from Le Fort I advancement with or without simultaneous vertical lengthening and clockwise rotation. Mandibular

Text continued on p. 1595

• **Figure 37-6** A 24-year-old woman arrived for evaluation. She had previously undergone four bicuspid extractions and orthodontics to neutralize her occlusion during her high school years. Analysis confirmed a short face growth pattern that accounted for her apparent large nose, weak profile, obtuse neck chin angle, and edentulous look. She agreed to a combined orthodontic and surgical approach. The patient's surgery included maxillary Le Fort I osteotomy (horizontal advancement, vertical lengthening, and clockwise rotation) with interpositional grafting; sagittal split ramus osteotomies (horizontal advancement and clockwise rotation); and osseous genioplasty (vertical lengthening and minimal horizontal advancement) with interpositional grafting. Minimal change in the occlusion was required (see Fig 23-3). **A,** Frontal views in repose before and after treatment. **B,** Oblique facial views before and after treatment.

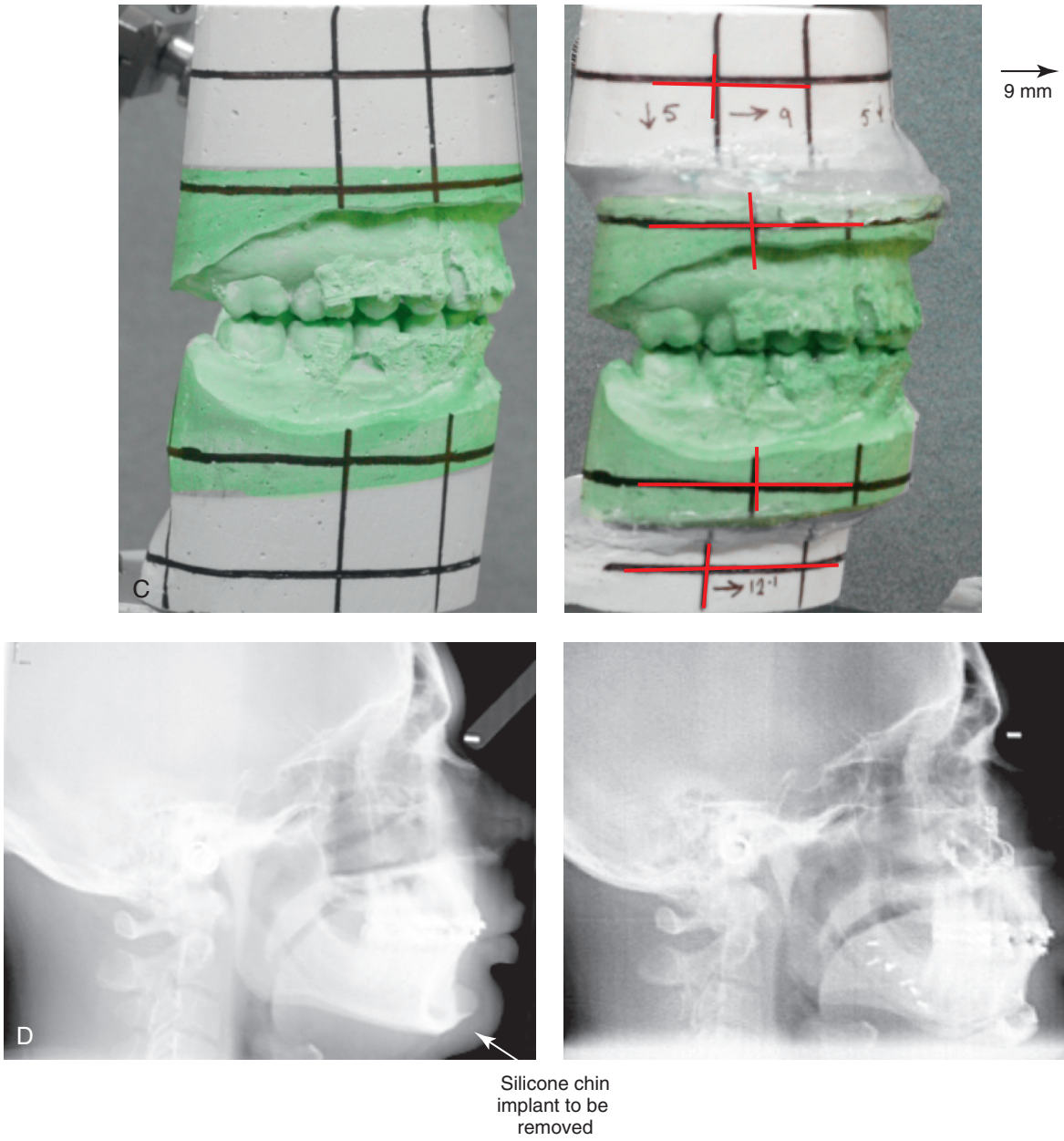
Continued



• **Figure 37-6, cont'd** **C,** Articulated dental casts that indicate analytic surgical planning. **D,** Lateral cephalometric radiographs before and after treatment.

• **Figure 37-7** A 37-year-old man was referred with a request to have a chin implant removed because he was displeased with the aesthetic results. When he was 20 years old, he underwent chin augmentation with a silicone implant in an attempt to camouflage his small lower jaw profile. Ten years later, he underwent four bicuspid extractions and orthodontic alignment to improve his occlusion. He had also undergone a septorhinoplasty procedure, but he was still experiencing continued difficulty breathing through the nose. During evaluation by this surgeon, he was recognized as having a short face growth pattern with decreased lower anterior facial height and limited facial projection. There was an accentuated labiomental lip curl and a "button" appearance to the chin. It became clear during a discussion that the patient suffered with snoring, restless sleeping, and excessive daytime fatigue. An attended polysomnogram confirmed a respiratory disturbance index of 16 events/hr, with desaturations of up to 89%. A titration study confirmed the need for 12 cm of water pressure to reduce the respiratory disturbance index to 3 events/hr. The use of continuous positive airway pressure was attempted but found to be uncomfortable and difficult for the patient to use. **A,** Frontal views with smile before and after surgery. **B,** Profile views before and after surgery.

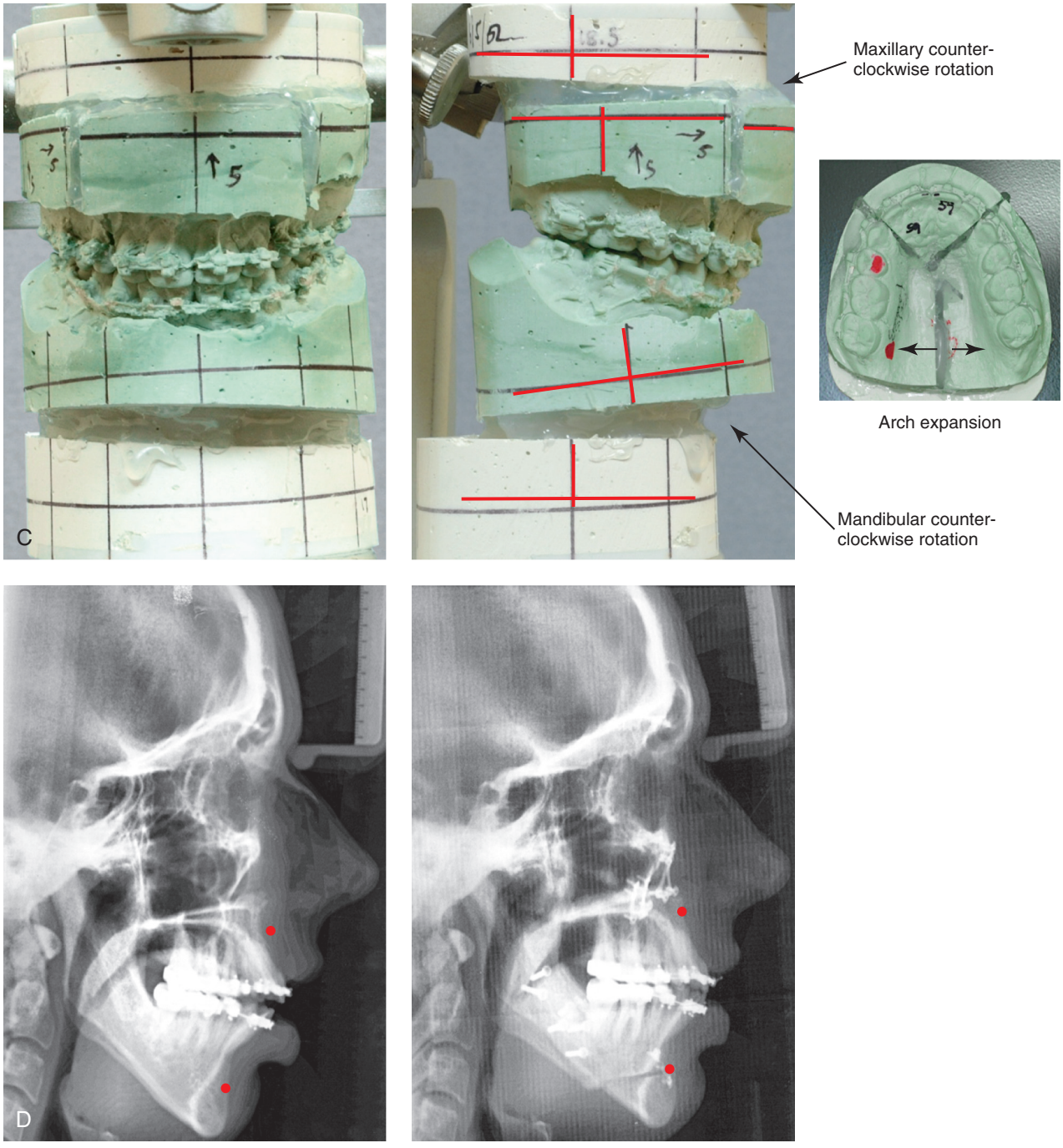
Continued



• **Figure 37-7, cont'd** Consultations with a sleep specialist, an otolaryngologist, and an orthodontist confirmed the advantage of an intranasal and orthognathic surgical approach. The patient agreed to perioperative orthodontics and surgery to improve the airway and facial appearance. His procedures included maxillary Le Fort I osteotomy (horizontal advancement +9 mm, vertical lengthening +5 mm), with interpositional grafting; bilateral sagittal split osteotomies of the mandible (horizontal advancement +12 mm); removal of the chin implant; osseous genioplasty (horizontal advancement and vertical lengthening) with interpositional grafting; reduction of the inferior turbinates; redo septoplasty; and an anterior approach to the neck (cervical flap elevation, defatting, and vertical platysma muscle plication). As a result of the procedures, the patient experienced relief of his snoring, improved quality of sleep, diminished daytime fatigue, and improved breathing during the day. Six months after surgery, he underwent an attended polysomnogram that indicated a normal sleep study with no snoring or apnea events (see Fig. 26-10). **C,** Articulated dental casts that indicate analytic surgical planning. **D,** Lateral cephalometric radiographs before and after surgery (note the preoperative chin implant).

• **Figure 37-8** A 21-year-old college graduate arrived for surgical evaluation. Her history and physical examination confirmed lifelong nasal obstruction, lip incompetence, difficulty chewing, and frequent speech articulation errors. Facial aesthetic concerns include a weak chin, a gummy smile, lip strain, and the impression of having a "big nose." The patient had been under an orthodontist's care since the mixed dentition and through her high school years. Growth modification attempts followed by rapid palatal expansion and full bracketing with four bicuspid extractions were unsuccessful to close the anterior open bite. Early periodontal and dental deteriorization were evident. Further evaluations were carried out by a speech pathologist, an otolaryngologist, and a periodontist; in addition, a fresh look was taken at this patient's orthodontic needs. Orthodontic dental decompensation in combination with orthognathic and intranasal surgery was chosen. The simultaneous procedures that were carried out included Le Fort I osteotomy in segments (horizontal advancement, vertical shortening, counterclockwise rotation, arch expansion, and correction of the curve of Spee); sagittal split ramus osteotomies (horizontal advancement and counterclockwise rotation); osseous genioplasty (vertical shortening and horizontal advancement); and septoplasty, inferior turbinate reduction, and nasal floor recontouring (see Fig. 21-5). **A,** Frontal views in repose before and after reconstruction. **B,** Profile views before and after reconstruction.

Continued



• **Figure 37-8, cont'd** **C,** Articulated dental casts that indicate analytic model planning. **D,** Lateral cephalometric radiographs before and after surgery.

• **Figure 37-9** A 21-year-old college graduate arrived for surgical evaluation. Her history and physical examination confirmed lifelong nasal obstruction, heavy snoring, lip incompetence, difficulty chewing, and frequent speech articulation errors. Facial aesthetic concerns included a weak chin, a gummy smile, and the impression of having a "big nose." She had been under an orthodontist's care from the mixed dentition through her high school years. Growth modification followed by rapid palatal expansion and full bracketing with attempted closure of the anterior open bite were attempted, without success. She had undergone a chin implant in an attempt to improve the profile, but this was also suboptimal. Further evaluations were carried out by a speech pathologist and an otolaryngologist, and a fresh look was taken at this patient's occlusal/dental needs. Orthodontic decompensation in combination with orthognathic and intranasal surgery was chosen. The simultaneous procedures that were carried out included Le Fort I osteotomy in segments (horizontal advancement, vertical shortening, counterclockwise rotation, arch expansion, and correction of the curve of Spee); sagittal split ramus osteotomies (horizontal advancement and counterclockwise rotation); removal of the chin implant; osseous genioplasty (vertical shortening and horizontal advancement); and septoplasty, inferior turbinate reduction, and nasal floor recontouring. **A,** Frontal views in repose before and after reconstruction. **B,** Frontal views with smile before and after reconstruction.

Continued

• **Figure 37-9, cont'd C,** Oblique facial views before and after reconstruction. **D,** Profile views before and after reconstruction.

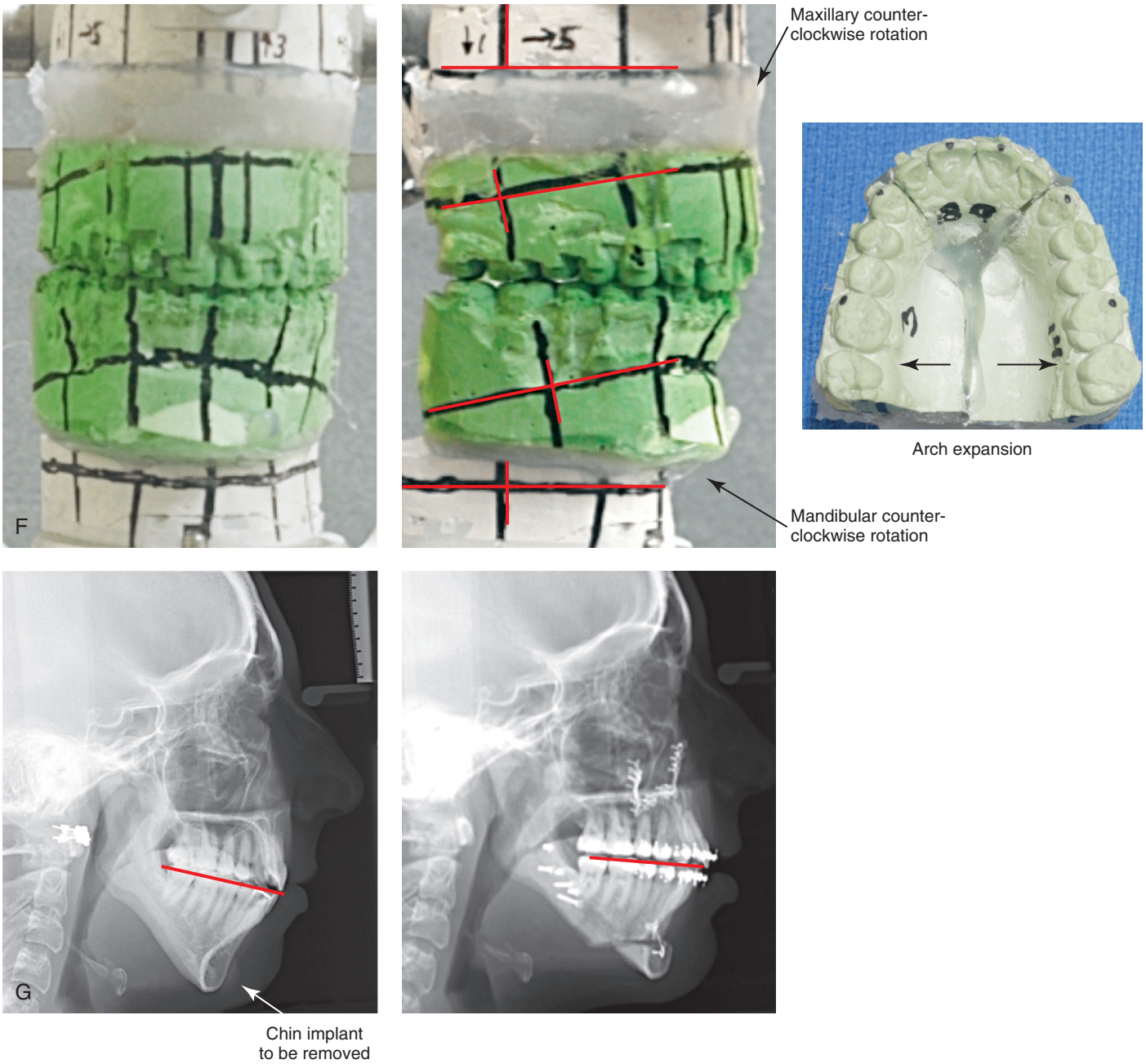
Prior to retreatment

Pre surgery

After treatment

• **Figure 37-9, cont'd E,** Occlusal views before retreatment, with orthodontic decompensation in progress, and after the completion of treatment.

Continued



• **Figure 37-9, cont'd F,** Articulated dental casts that indicate analytic model planning. **G,** Lateral cephalometric radiographs before and after surgery. Note that the chin implant was inadequate to improve the profile. An osseous genioplasty in addition to an orthognathic correction achieved the chosen objectives.

• **Figure 37-10** Intraoperative views of four silicon/Silastic chin implants removed from four separate patients who had suffered complications and suboptimal results and who had requested chin implant removal.

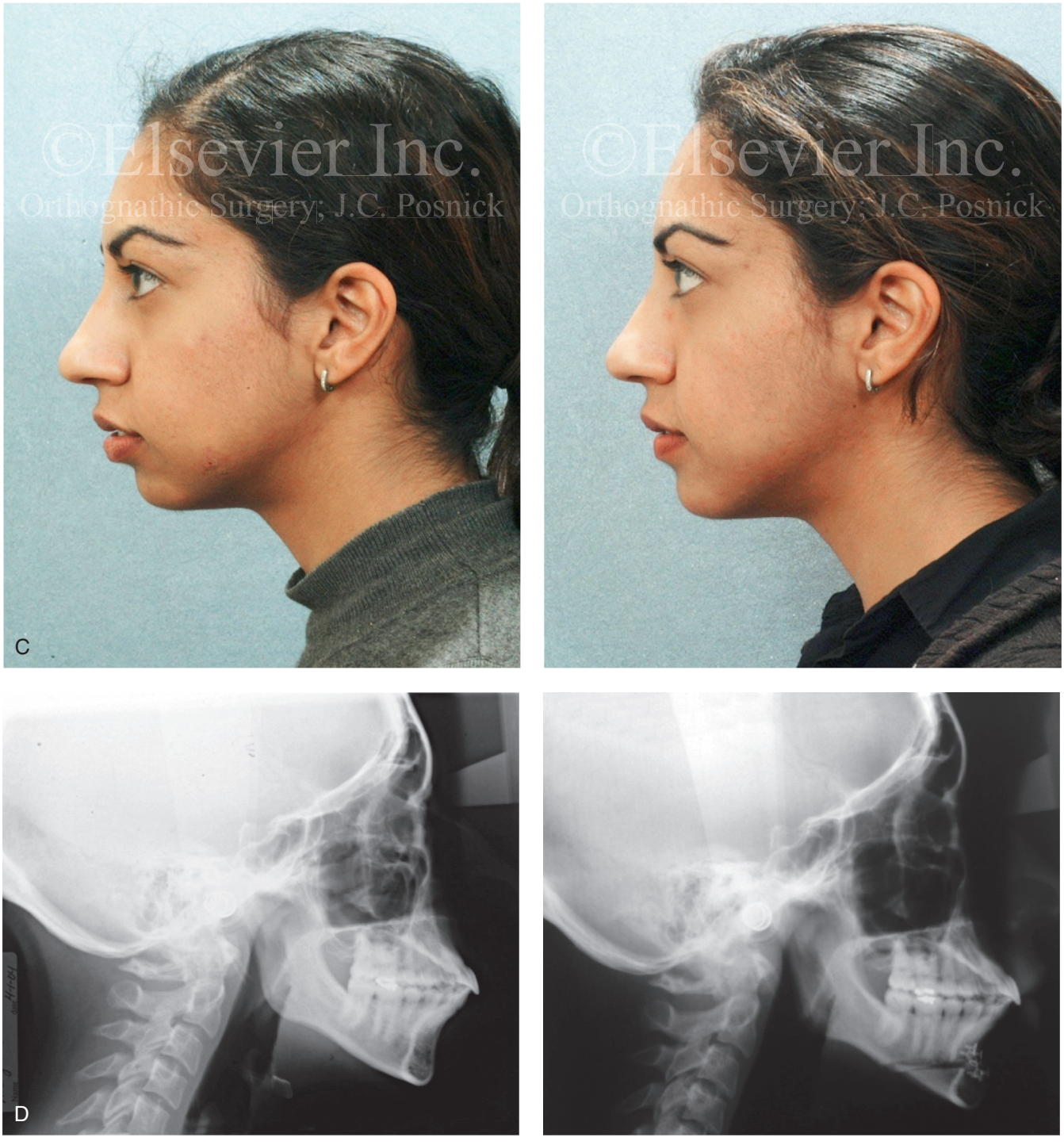
• **Figure 37-11** A woman in her early 50s with facial aging concerns. She had a mild long face growth pattern and a satisfactory occlusion. The lower anterior facial height was increased, which resulted in mild lip incompetence and mentalis strain as well as deep perioral creases. She agreed to blepharoplasty (elliptical excision of upper lid skin and redraping of lower lid tissue); osseous genioplasty (vertical reduction and horizontal advancement); and a face lift (see Fig. 40-9). **A,** Frontal views before and after surgery. **B,** Profile views before and after surgery. Note the relief of the perioral folds and mentalis strain as a result of vertical reduction and advancement genioplasty.

• **Figure 37-12** A Caucasian man in his 30s arrived for the evaluation a "weak chin" and requested aesthetic improvement. Examination confirmed a mild developmental mandibular deficiency (see Chapter 19). Successful orthodontics had been carried out during his teenage years, with only mild flaring of the mandibular incisors. An osseous genioplasty was agreed to that included significant vertical lengthening and limited horizontal advancement with interpositional bloc hydroxyapatite grafting. **A,** Profile views before and after reconstruction. **B,** Lateral cephalometric radiographs before and after reconstruction.

©Elsevier Inc.
Orthognathic Surgery; J.C. Posnick

• **Figure 37-13** A 27-year-old Indian woman arrived and requested of a reduction rhinoplasty and a chin implant to treat her perceived "big nose" and "small chin." Analysis confirmed maxillomandibular sagittal deficiency that was treated during her teenage years with compensatory orthodontics to neutralize the occlusion. There was marked procumbency of the maxillary and mandibular anterior dentition but with adequate tooth and gingival show, without lip incompetence and with good to the periodontium. The nasal aesthetic unit had thick skin but satisfactory proportions. The patient lacked lower anterior facial height and facial projection, especially at the pogonion. She was treated with an osseous genioplasty (vertical lengthening and limited horizontal advancement) and interpositional bloc hydroxyapatite grafting. **A,** Frontal views before and after reconstruction. **B,** Oblique facial views before and after reconstruction.

Continued



• **Figure 37-13, cont'd C,** Profile views before and after reconstruction. **D,** Lateral cephalometric radiographs before and after reconstruction.

©Elsevier Inc.
Orthognathic Surgery; J.C. Posnick

• **Figure 37-14** A woman in her early 20s arrived for the evaluation of her facial aesthetics. She had a developmental jaw deformity that was characterized primarily by mandibular deficiency and a lifelong history of obstructed nasal breathing. During her teenage years, she underwent an orthodontic camouflage approach that included maxillary bicuspid extractions with anterior dentition retraction to achieve an improved occlusion. She perceived a "big nose" and a "small chin" and requested reduction rhinoplasty and a chin implant. She refused redo orthodontics and an orthognathic correction (see Chapter 19). A compromise was agreed to that included open rhinoplasty and osseous genioplasty. The patient's rhinoplasty included nasal osteotomies (in-fracture and straightening); dorsal reduction (bone and cartilage); and tip maneuvers (modification of lower lateral cartilages and septal cartilage caudal strut grafting). Osseous genioplasty included vertical lengthening with interpositional bloc hydroxyapatite grafting and minimal horizontal advancement. **A,** Frontal views with smile before and after reconstruction. **B,** Oblique facial views before and after reconstruction.

Continued

• **Figure 37-14, cont'd C,** Profile views before and after reconstruction. **D,** Lateral cephalometric radiographs before and after reconstruction.

©Elsevier Inc.
Orthognathic Surgery; J.C. Posnick

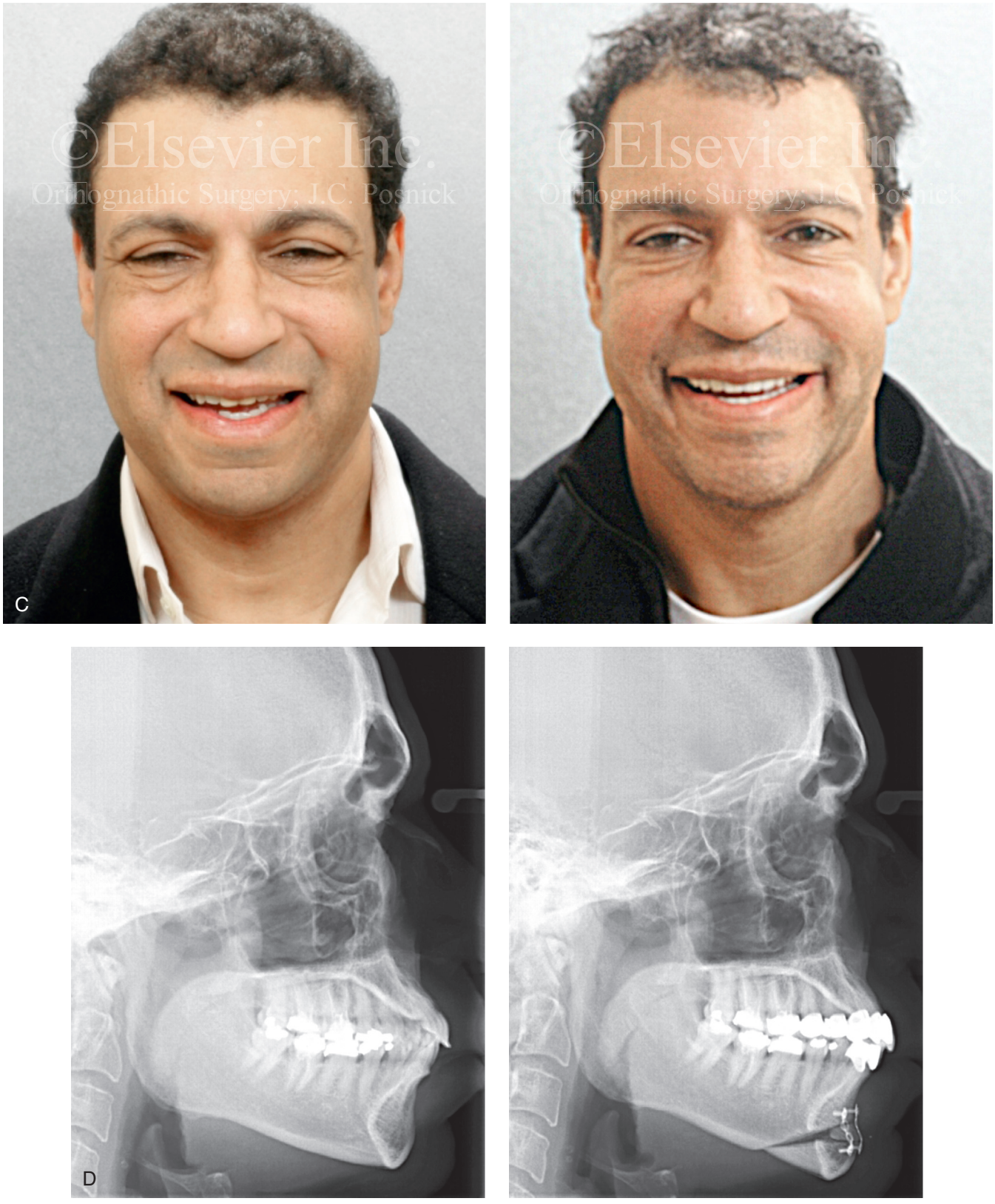
• **Figure 37-15** A woman in her mid 20s arrived for the evaluation of her facial aesthetics. She had a short face growth pattern (i.e., maxillomandibular deficiency) that was treated during her teenage years with compensatory orthodontic alignment only. An orthognathic correction to improve her horizontal facial projection and to increase the lower anterior facial height was recommended (see Chapter 23). She was unwilling to undergo further orthodontics and refused an orthognathic approach. A compromise was agreed to that included open rhinoplasty and osseous genioplasty. The patient's rhinoplasty included nasal osteotomies (in-fracture and straightening); dorsal reduction (bone and cartilage); and tip maneuvers (modification of lower lateral cartilages and septal cartilage caudal strut grafting). Osseous genioplasty included vertical lengthening and minimal horizontal advancement with interpositional bloc hydroxyapatite grafting. **A,** Frontal views in repose before and after reconstruction. **B,** Frontal views with smile before and after reconstruction.

Continued

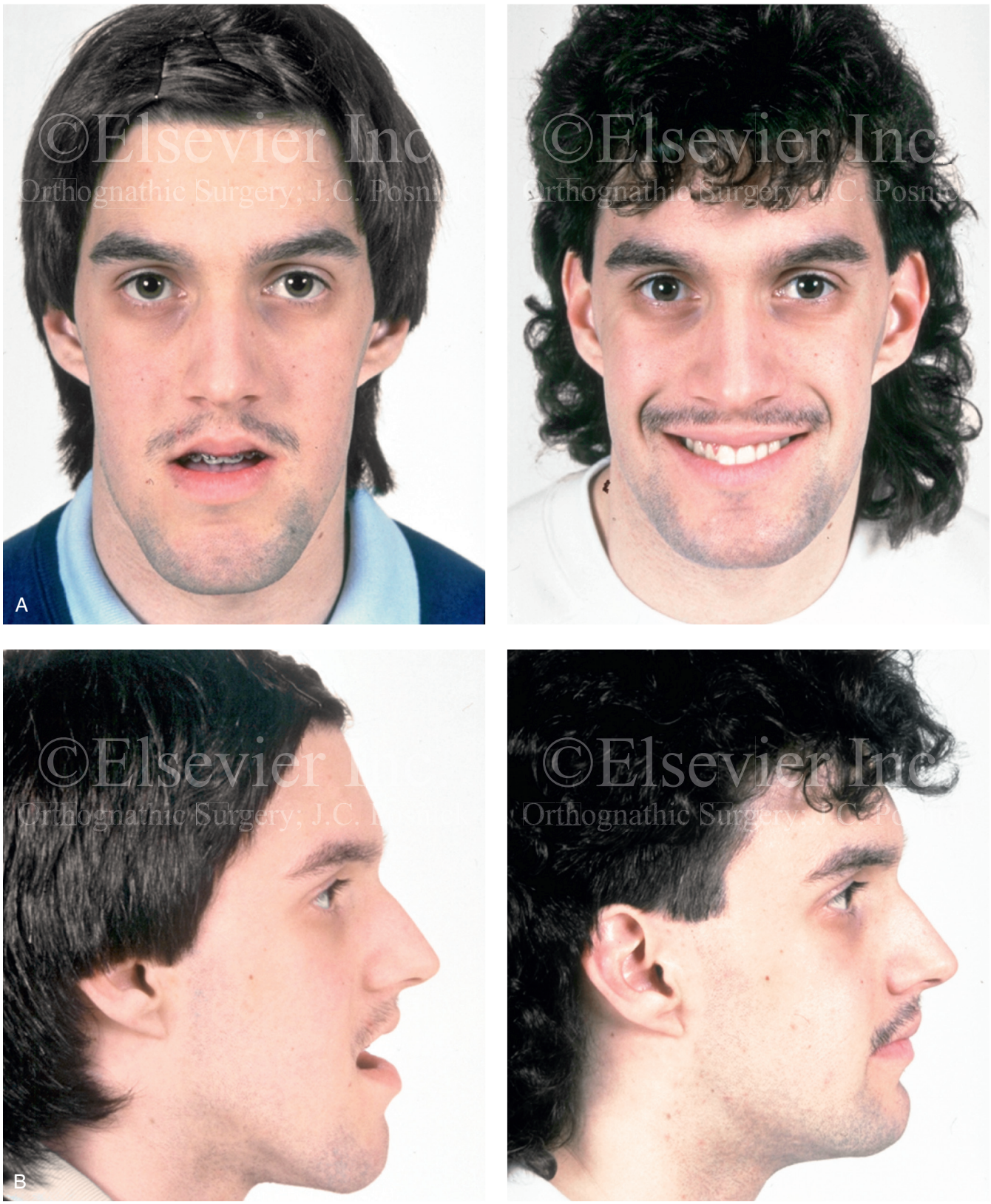
• **Figure 37-15, cont'd C,** Oblique facial views before and after reconstruction. **D,** Profile views before and after reconstruction. **E,** Lateral cephalometric radiographs before and after reconstruction.

• **Figure 37-16** A woman in her early 50s requested an evaluation of a "weak chin," deep perioral creases, mild ptosis of the brow and upper eyelids, and unfavorable nasal aesthetics. Examination confirmed mild drooping of the brow and mild excess skin of the upper eyelids. A developmental mandibular deficiency was unchanged over the years, and she had a functional occlusion. She agreed to a limited incision brow lift, but as a result of dryness of the eyes, decided against the removal of the skin of the upper lid. She also agreed to an osseous genioplasty that included vertical lengthening and horizontal advancement with interpositional bloc hydroxyapatite grafting. Her rhinoplasty included nasal osteotomies (in-fracture); dorsal reduction (bone and cartilage); and tip maneuvers (modification of lower lateral cartilages and septal cartilage caudal strut grafting). All procedures were carried out simultaneously. **A,** Oblique facial views before and after surgery. **B,** Profile views before and after surgery.

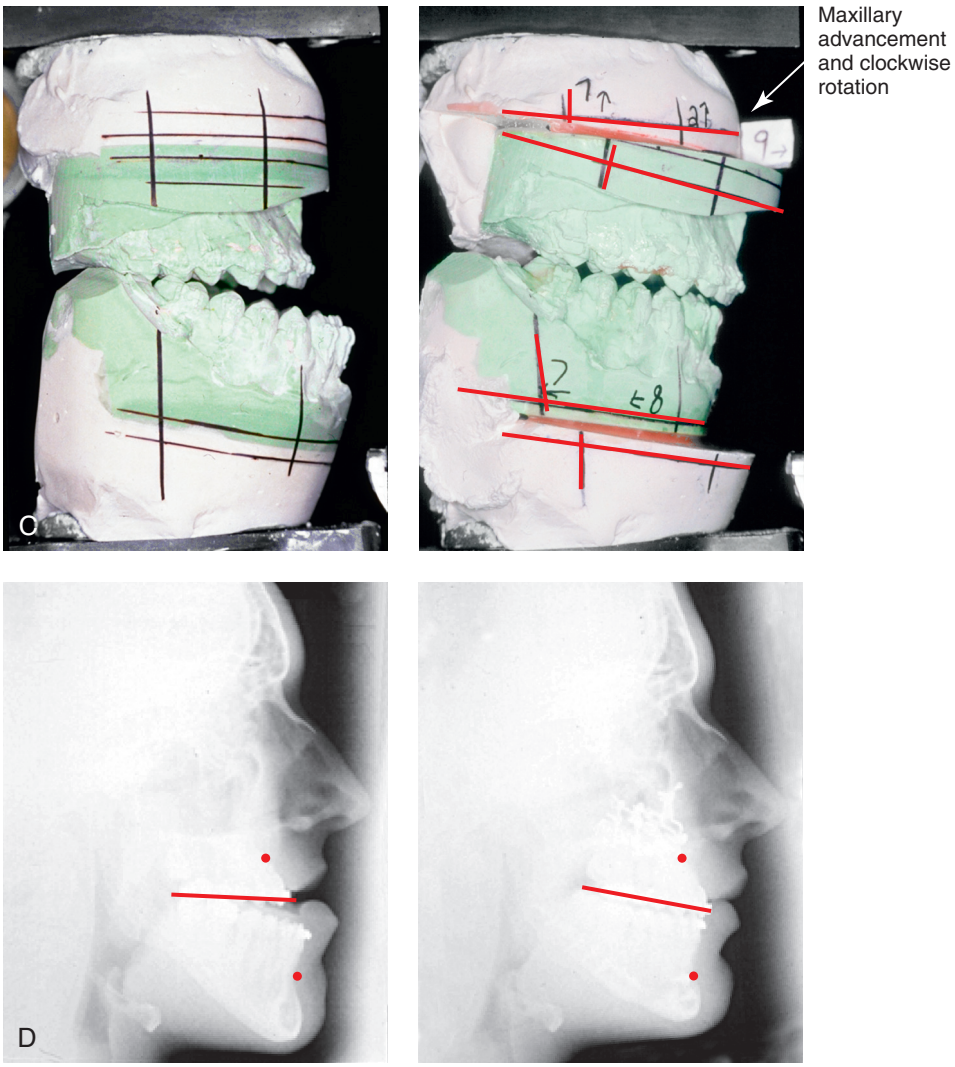
• **Figure 37-17** An African-American man in his early 40s underwent evaluation for a "weak chin" and laxity of the neck soft tissues. There was a degree of procumbency to the maxillary and mandibular dentition but with a stable occlusion. He also planned to undergo cosmetic modification of the anterior dentition. A treatment plan was agreed to that included osseous genioplasty (vertical lengthening with interpositional grafting and minimal horizontal advancement) and an anterior approach to the soft tissues of the neck (cervical flap elevation, neck defatting, and vertical platysma muscle plication). **A,** Profile views before and after surgery. **B,** Oblique facial views before and after surgery.



• **Figure 37-17, cont'd C,** Frontal views before and after surgery. **D,** Lateral cephalometric radiographs before and after surgery.



• **Figure 37-18** A 17-year-old high school student was referred by his orthodontist for surgical evaluation. His maxillary deficiency and mandibular excess Class III negative overjet anterior open-bite malocclusion required orthodontics and jaw surgery. Because the patient had a lifelong history of nasal obstruction and consistent physical findings, intranasal procedures were also required. With orthodontic dental decompensation complete, the patient's surgery included maxillary Le Fort I osteotomy (horizontal advancement, anterior vertical lengthening, and clockwise rotation); sagittal split ramus osteotomies (set-back); osseous genioplasty (vertical reduction); and septoplasty and inferior turbinate reduction. **A,** Frontal views before and after treatment. **B,** Profile views before and after treatment.



• **Figure 37-18, cont'd C,** Articulated dental casts that indicate analytic model planning. **D,** Lateral cephalometric radiographs before and after treatment.

ramus osteotomies are also carried out with limited or no set-back at the incisors but with clockwise rotation to diminish projection at the pogonion. In this case, lower-face aesthetics are often further enhanced via an osseous genioplasty with vertical reduction and minimal sagittal change.

A second frequent pattern of presentation is an individual with a chin that appears prominent in profile because the mandible is overly closed (i.e., short face growth pattern). In these patients, the vertical height (in millimeters) from the mandibular incisal edges to the menton is deficient. With this dentofacial deformity, both horizontal and vertical deficiency of the maxilla and the mandible (i.e., decreased lower anterior facial height and limited facial projection) are present (see Chapter 23). A "pseudoprotrusion" of the chin will result. A patient who asks for a chin set-back only to manage his or her short face (i.e., maxillomandibular deficiency) is unlikely to be pleased with the aesthetic result if this request is honored. In this case, reestablishing the preferred vertical and horizontal dimensions of the face requires a Le Fort I osteotomy with horizontal advancement, vertical lengthening, and, often, clockwise rotation. The mandible—which is also horizontally and vertically deficient—requires sagittal split ramus osteotomies and reorientation to match the new maxillary position. The chin region also benefits from a change in shape. This is accomplished through an oblique osteotomy of the chin with clockwise rotation (i.e., downward and backward positioning of the pogonion) while maintaining contact and pivoting at the posterior osteotomy line. The interpositional gap is grafted with allograft, autograft, or bone substitute, and the chin segment is fixed in place with plates and screws (see Figs. 37-6 and 37-7).

All of these facial aspects must be considered before embarking on any treatment of the chin region if a favorable result and a satisfied patient are to be expected.

Chin Asymmetries

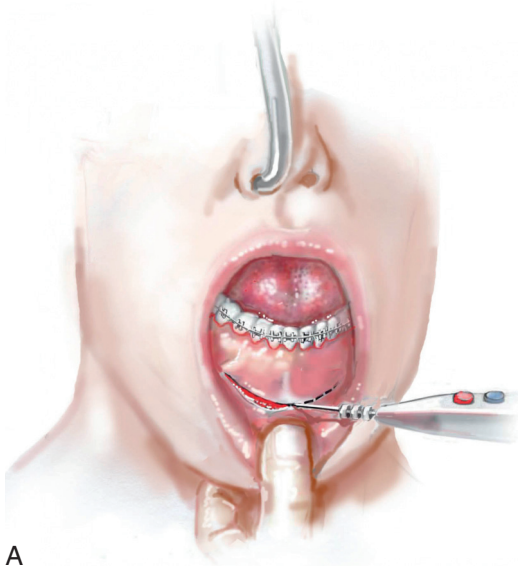
Asymmetries of the chin relative to the face are most frequently encountered in association with specific syndromes and other conditions such as hemifacial microsomia, hemimandibular elongation, hemimandibular hyperplasia, or a condyle fracture sustained during childhood with resultant ipsilateral mandibular undergrowth (Fig. 37-19). For individuals who present with the chin shifted to one side, aesthetic assessment of the entire face, including the soft tissues and the skeletal structures, should be undertaken. Consideration is given to both the lower facial skeleton (i.e., the maxilla, the mandible, and the chin region) and the upper facial skeleton (i.e., the cranial vault, the zygomas, the orbits, and the nasal bones). The chin region may be asymmetric or disproportionate, or it may simply appear to be so as a result of maxillary or mandibular malposition. In several of the conditions mentioned, the extent of actual chin asymmetry is much less than that of the mandible and maxilla. When indicated, a Le Fort I osteotomy and sagittal

• **Figure 37-19** A 16-year-old boy who had sustained a fracture of the right condyle of the mandible during his early childhood presented with resulting facial asymmetry. He previously underwent years of orthodontics to neutralize the occlusion, which also resulted in loss of labial bone along the anterior mandibular teeth. He then underwent an orthognathic approach that included Le Fort I osteotomy (cant correction); left sagittal split ramus osteotomy; reconstruction of the right condyle–ascending ramus with a costochondral graft; and an osseous genioplasty (correction of vertical and horizontal asymmetry) (see Fig. 35-8). **A,** Frontal facial views with smile before and after reconstruction. **B,** Oblique facial views before and after reconstruction. *A, from Posnick JC: Management of facial fractures in children and adolescents,* Ann Plast Surg *33:442-457, 1994.*

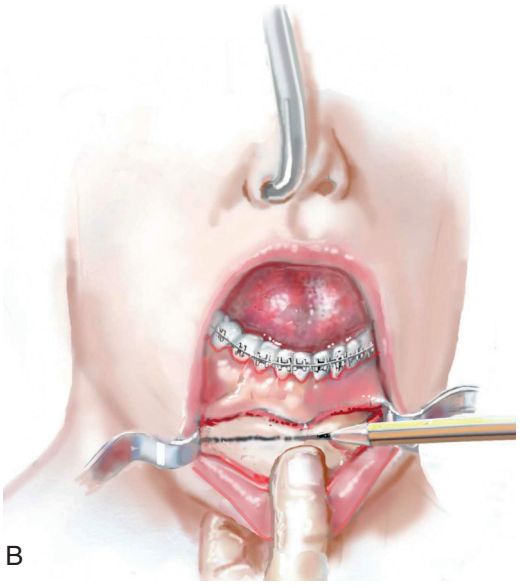
split ramus osteotomies of the mandible with three-dimensional repositioning will greatly improve the chin symmetry. A separate osteotomy to reposition the chin region is also generally required to simultaneously correct disproportion of the lower third of the face.

Osseous Genioplasty: Surgical Technique (Videos 11 and 12)

- Attention is turned to the facial vestibule of the chin. The lower lip is stretched outward to allow for visualization of the mental nerve through the mucosa on each side. In the depth of the vestibule, an incision is made with a Bovie electrocautery device or a knife (no. 15 blade) just through the mucosa from cuspid region to cuspid region, stopping just short of the visualized mental nerve on each side (Fig. 37-20, *A*). The center two thirds of the incision is next extended down to bone. A full cuff of mucosa and muscle is maintained adjacent to the attached gingiva of the anterior teeth; this should allow for adequate layered wound closure of the muscle and mucosa without periodontal sequelae.
- Dissection with an elevator exposes the anterior surface of the chin although not completely to the inferior border of the central chin. The dissection continues laterally and remains inferior to the mental nerve, with lateral exposure to the inferior border of the mandible. There is no need to dissect above the mental nerve on either side. If 360-degree dissection around the nerve is completed, it is more likely that the nerve will be excessively stretched or avulsed.



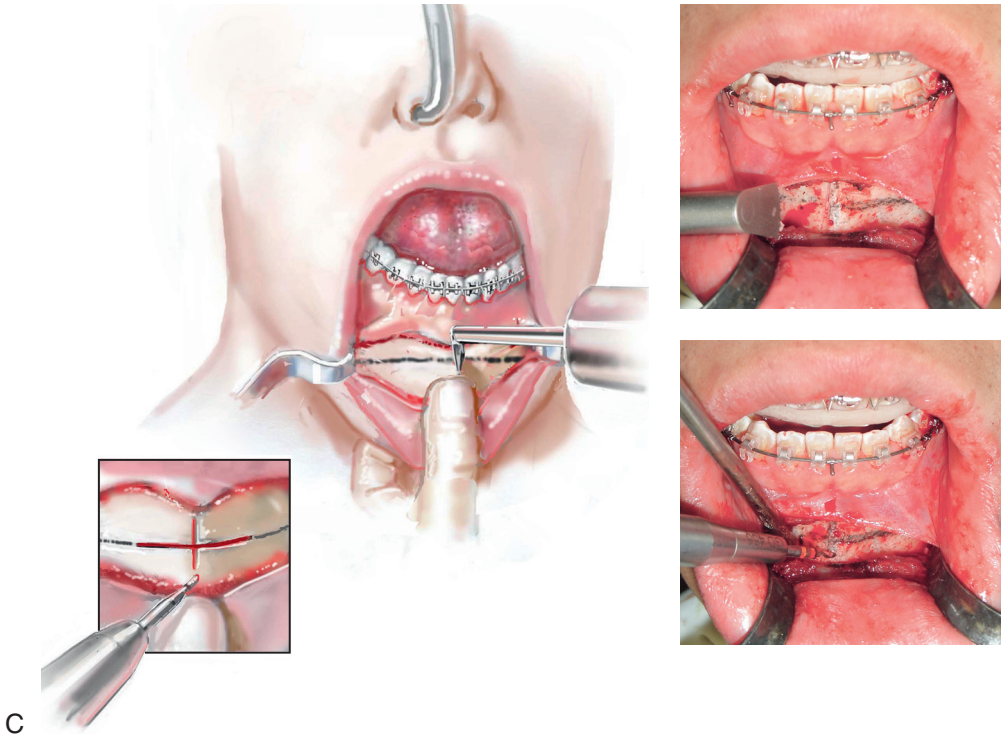
A



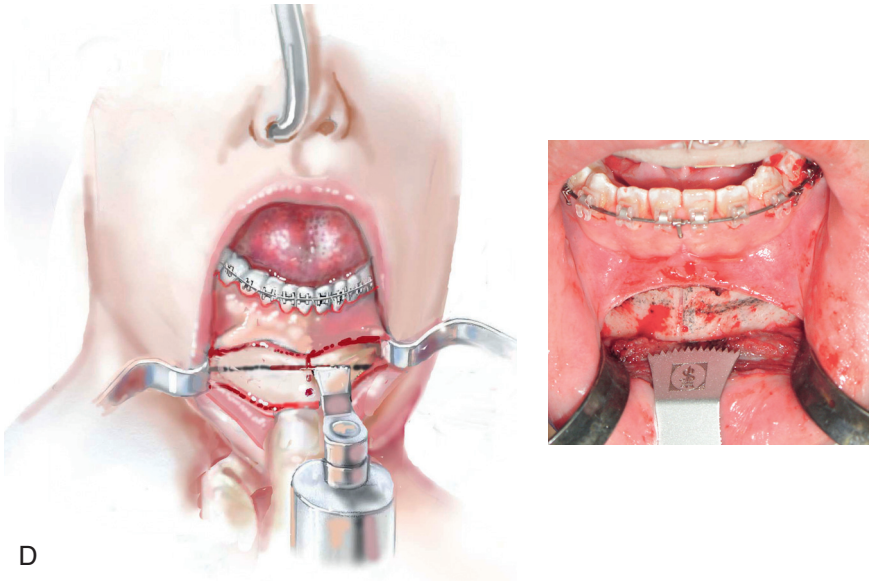
B

• **Figure 37-20** Illustrations of standard osseous genioplasty. See the text for descriptions of the techniques depicted.

Continued

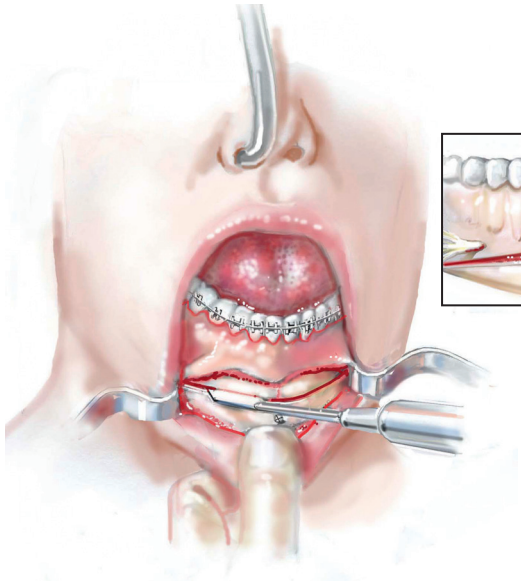


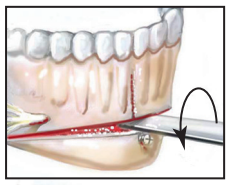
C



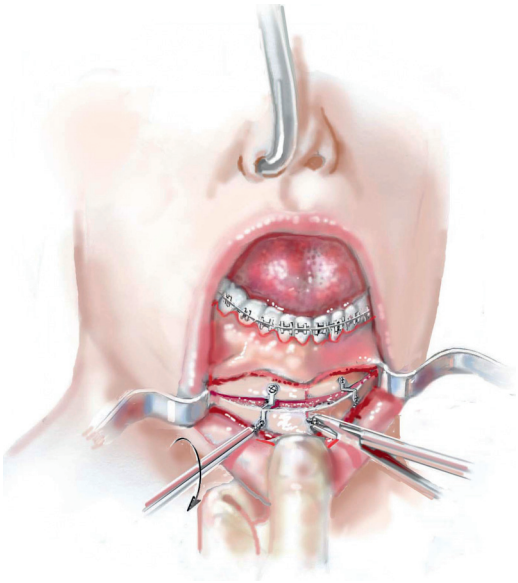
D

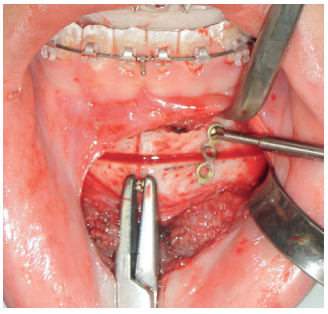
• **Figure 37-20, cont'd**

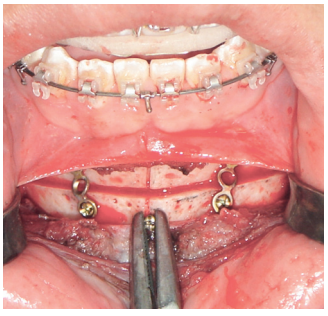




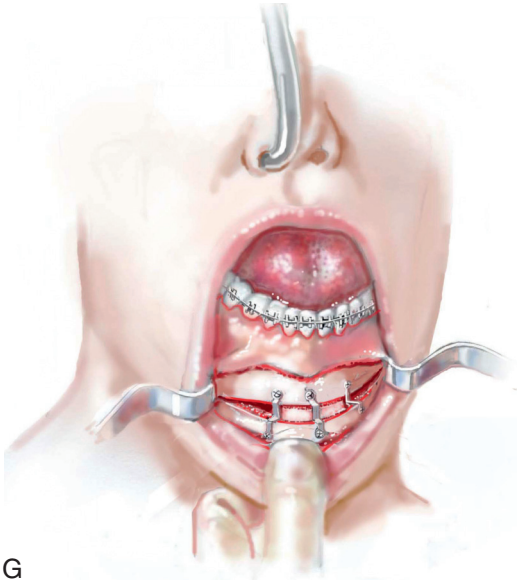
E







F



G

• **Figure 37-20, cont'd**

- A small S-shaped Tessier chin retractor is placed on each side inferior to the mental nerve and laterally to the inferior border. A sterile pencil is used to mark the location of the oblique osteotomy ((see Fig. 37-20, *B*). This should be planned sufficiently below the dental roots and the mental foramen on each side. The exact location of the osteotomy will depend on the presenting chin morphology and the planned reconstruction (i.e., vertical reduction or lengthening and the extent of horizontal advancement).
- With the use of an oscillating saw (i.e., a thin fan blade on a long shaft), a vertical groove is made in the midline across and perpendicular to the proposed horizontal osteotomy. This will help to maintain vertical orientation after the osteotomy and before fixation (see Fig. 37-20, *C*).
- A drill hole is placed in the midline within the distal chin (see Fig. 37-20, *C*). Later, a screw (1.7 mm in diameter and 8 mm in length) will be partially inserted and used as a retractor. The screw is usually not placed in the distal chin until the osteotomy is completed. It will then be held with a wire twister that is used as a retractor to facilitate the repositioning and orientation of the distal chin unit.
- The oblique osteotomy of the chin is initiated in the central portion with the use of an oscillating saw (i.e. wide fan blade on a short shaft). The use of this saw and blade to initiate the osteotomy helps to maintain orientation and to limit canting (see Fig. 37-20, *D*).
- Using a reciprocating saw with a short blade and then with a longer blade is helpful to complete the lateral aspects of the osteotomy and to go through the depth of the bone on each side (see Fig. 37-20, *E*). An osteotome may be inserted with a twisting motion to complete the osteotomy separation (see Fig. 37-20, *E*).
- When vertical shortening of the chin is planned, there are two options. If only limited shortening is required in combination with horizontal advancement, it may be preferred to use a rotary drill with a watermelon bur to remove bone height at the osteotomy line. If a more significant vertical reduction is planned, a second parallel osteotomy (for ostectomy) is completed through the proximal chin (below the dental roots) using a reciprocating saw with a straight blade on a long shaft. Any lateral–inferior bony irregularities are also removed using a rotary drill with a watermelon bur to limit palpable or visible step-offs.
- The distal chin is held in the desired position by the assistant to the surgeon with the use of the wire twister secured to the positioning screw. The surgeon contours each titanium plate across the osteotomy site and then secures each with screws (1.7 mm in diameter and 4 mm in length). Typically, either a three- a four-hole straight plate is contoured with the ends placed on either side of the osteotomy. The superior screw is usually placed in between and inferior to the lateral incisor and canine on each side (see Fig. 37-20, *F*). With the chin secured in its new location, the positioning screw is removed.
- If significant vertical lengthening of the chin is planned, an interpositional graft (e.g., autograft, allograft, bloc hydroxyapatite) is crafted and placed in the gap. The graft fills the central gap between the fixation plates. I do not find it necessary to place graft in the lateral aspect of the gaps. An additional plate and screws are generally placed vertically in the midline across the osteotomy site and directly over the graft (see Chapter 18 and Fig. 37-20, *G*).

Achieving Favorable Chin Aesthetics

Establishing preferred aesthetics in the chin region is heavily dependent on the surrounding structures. The variables include the following: 1) maxillomandibular morphology; 2) the position of the teeth within each arch; and 3) the soft-tissue envelope that overlies the skeletal structures.

Preferred *maxillomandibular morphology* is primarily dependent on the following: 1) facial height; 2) sagittal projection; and 3) A-point–to–B-point relationships when viewing the facial profile and the natural head position. Ideally, the positioning of the teeth within the alveolar bone establishes the preferred *incisor inclination,* which is an important component of perioral aesthetics. The *overlying soft-tissue envelope* will be distorted by either maxillomandibular skeletal or dental disharmony. Common patterns of soft-tissue distortions may include either lip strain with labiomental effacement (i.e., long face growth pattern) or a full flaccid lower lip with a deep labiomental fold (i.e., mandibular deficiency growth pattern).

Morphology of the chin aesthetic unit is an important aspect of facial aesthetics. The essential components of chin morphology include *vertical height, horizontal projection,* and the *surface contour of the pogonion.* When considering the chin aesthetic unit, an assessment of the individual's lateral cephalometric radiograph is useful. As stated, if the vertical height of the chin is too long, lip incompetence and an overly effaced lip–chin soft-tissue drape will result. If the vertical height of the chin is too short, there will be a flaccid lower lip, a deep labiomental curl, and a "button" appearance to the soft tissues.

Riolo's table of normative cephalometric values confirms that the mean vertical height of the chin (i.e., the mandibular incisal edge to the menton) is approximately 42 mm (±2.7 mm) for females and 49.4 mm (±2.9 mm) for males.[83A] These measurements are a useful benchmark from which to judge the preferred vertical height of the individual's chin.

The extent of preferred horizontal chin projection at the pogonion should be with reference to the B-point. When viewing an individual's lateral cephalometric radiograph taken in the natural head position and with the incisors upright (with or without the need for orthodontics) and with the maxillomandibular vertical height and horizontal projection being proportionate (with or without the need

for orthognathic surgery), the pogonion should not project more than 3 mm to 5 mm anterior to the B-point. If the pogonion projects too far in front of the B-point, the overly prominent chin is unattractive. If the pogonion does not project enough in front of the B-point, then a "weak" chin will be apparent in profile.

At times, the surface contour of the anterior bony chin will be unattractive. Two common patterns of chin-surface dysmorphology include either an *overly pointed pogonion* or *bifid bony morphology* (i.e., prominent lateral tubercles). Direct recontouring of the exposed chin surface with a bur on a rotary drill can be carried out to improve either of these surface irregularities. When indicated, this is accomplished simultaneously with an inferior border osteotomy to also alter the vertical height and horizontal projection of the chin.

In review, errors concerning chin aesthetics are more likely when maxillomandibular morphology and incisor inclination are not fully considered. Attempting to compensate for the unfavorable positioning of either the maxillomandibular complex or the teeth is responsible for much of the observed patient dissatisfaction after a chin procedure. The common error in these circumstances is to either use an osseous genioplasty to project the pogonion too far in front of the B-point or to place a "too-large" chin implant with a resulting unnatural look. Either of these techniques is likely to be more unattractive than the presenting "weak" chin.

Avoiding Pitfalls during Chin Surgery

The chin is one of the most visually prominent facial features, and its role in facial appearance has been recognized since antiquity. It may seem to be an easy structure to alter, but focusing on the chin without fully considering the associated facial dysmorphology (e.g., the skeleton, the dental and soft tissues) may have unexpected and unwanted effects on overall aesthetics. The basic surgical technique of osseous genioplasty is reviewed earlier in this chapter. It is also illustrated in Figure 37-20 and can be seen on (**Videos 11 and 12**). Avoiding pitfalls during the aesthetic evaluation of the patient's chin and during the surgical execution of an osseous genioplasty is essential to the achievement of a favorable outcome.*

1. When assessing a chin that is considered to be either retrusive or protrusive, place the patient in the natural head position and the mandible in the rest position (i.e., with the condyles seated and with normal freeway space between the teeth in each arch). The lips should be relaxed and separated. By assessing the face in this way, you are likely to unmask any baseline dentofacial deformity (e.g., short or long face growth pattern) and to gain a more accurate impression of any true chin disproportion.
2. The experienced surgeon will rely heavily on the direct visual examination to evaluate overall facial proportions and any notable mirror-image asymmetries that involve (but are not limited to) the chin region. Without protractors or rulers, the absence of Euclidean proportions can usually be discerned. Confirmation of the clinician's aesthetic impressions may then be clarified through either anthropometric surface measurements or cephalometric radiographic analysis, as indicated.
3. The theoretic advantage of an alloplastic (chin implant) genioplasty is that it is a procedure that is seemingly easier to execute and often considered less invasive by the patient. It has the disadvantage of being primarily effective only for a mild anteroposterior deficiency of the chin, and it generally requires an external facial scar. It will have a higher incidence of infection than a chin osteotomy, and such an infection may even occur years later.[34,35,41,49,82,121,125] In addition, there may be underlying bony erosion from the implant over time, and the individual can never be without at least some concern about trauma to the chin. Alternatively, the osseous genioplasty is versatile in all three planes and has proven and long-lasting results without concerns about infection, bony erosion, a shift in position, or the need for limited physical activities after initial satisfactory healing has occurred.
4. When completing the intraoral incision and the soft-tissue dissection to expose the surface of the bony chin, avoid subperiosteal dissection above the mental neurovascular bundle to limit the extent of nerve contusion and stretching and the chance of avulsion from the mental foramen. The mucosal incision should be deep in the vestibule to leave a full cuff of mucosa and muscle for ease of two-layer closure and to limit periodontal sequelae.
5. When lengthening the chin, an oblique osteotomy with the interpositional placement of a graft (e.g., allogenic or autogenous bone, porous bloc hydroxyapatite) is generally preferable to the placement of a chin implant (as mentioned in point 3; see Fig. 37-20, *G*).
6. When vertical reduction and posterior set-back are indicated, the vector of reduction and set-back should generally follow the long axis of the chin prominence. This will avoid step-offs at the junction of the chin osteotomy and the inferior border of the mandible and limit suboptimal aesthetics as a result of the "bunching" of the soft tissues (see Fig. 37-18).
7. When using a reciprocating saw to complete the posterior (lateral) aspect of the oblique chin osteotomy, the inferior border may unknowingly remain partially intact. When this happens, there is a tendency for the uncontrolled fracture of the chin to be more posterior than desired. This can result in an

*References 5, 12, 15, 19, 35, 38-40, 42, 47, 48, 55, 58, 62, 63, 71, 74, 76, 78, 81, 93, 94, 96, 102, 103, 110, 111, 119, 122, 134

uneven aesthetic result, and it is avoided by carefully completing the lateral and posterior osteotomy before the "splitting" of the chin (see Fig. 37-20, *E*).

8. When completing a vertical reduction and advancement genioplasty, limit significant step-offs at the junction of the chin osteotomy and the inferior border of the mandible to avoid a palpable ridge or an unattractive "hourglass" appearance to the inferior border of the mandible. A continuous inferior border of the mandible is less likely to accentuate jowling. A significant ridge at the osteotomy site may be prevented by extending the chin osteotomy more posterior (i.e., as far back as first molar); by not "overadvancing" the chin (i.e., in profile, the soft tissue of the chin should not be anterior to the lower lip); and by recontouring any significant step-offs using a rotary drill with a watermelon bur before securing the chin in its new location.
9. When an alloplastic chin implant has failed and requires removal (i.e., as a result of suboptimal aesthetics, infection, bony erosion, or a shift in implant position), chin morphology is generally best restored with a vascularized osseous genioplasty rather than another avascular implant. Unfortunately, the implant will have stretched the overlying soft tissues, resulting in ptosis and possible muscle dysfunction when it is removed. If the chin implant was composed of non-porous material (e.g., Silastic), then a fibrous capsule will have formed, which adds to the residual soft-tissue deformity. Reassessing the overall maxillofacial morphology is essential before proceeding with further chin surgery. A baseline maxillo-mandibular dysharmony may be present and require reconstruction to best achieve patient satisfaction (see Figs. 37-1, 37-7, 37-9, and 37-10).
10. When the surface contour of the chin is severely dysmorphic (i.e., overly pointed or bifid), direct bur recontouring using a rotary drill with a watermelon bur is the preferred approach. This does not negate the simultaneous completion of an oblique osteotomy for the three-dimensional repositioning of the remaining chin unit. Adequate circulation to the distal chin is maintained through the genioglossus and geniohyoid muscle attachments to the genial tubercle.
11. When completing an oblique osteotomy of the chin, the temporary placement of a screw in the distal chin (i.e., one that is only halfway screwed in) that is then clamped with a wire twister (i.e., to be use used as a handle or retractor) is helpful for ease of positioning of the chin before osteotomy fixation (see Fig. 37-20, *C* and *F*).
12. Marking the midline of the bony chin with a small vertical notch perpendicular to the proposed horizontal osteotomy helps with the maintenance of chin symmetry before the placement of plate and screw fixation (see Fig. 37-20, *C*).
13. Adequate fixation of the distal chin across the osteotomy site is reliably carried out with a limited volume of small titanium plates and screws. The type and extent of screws and plates used will vary in accordance with patient-specific fixation needs and surgeon preferences (see Fig. 37-20, *F* and *G*).

Complications and Unfavorable Results

Although the majority of patients who have undergone an osseous genioplasty state that they would do it again, complications and suboptimal results will always occur in a minority of cases.*

Infection

Infection occurs among less than 1% of patients who have undergone an osseous genioplasty. The routine use of prophylactic antibiotics to cover both oral flora and skin organisms is useful to limit this complication. When an avascular implant is placed to augment the chin (rather than a chin osteotomy being performed), a higher rate of infection would be expected. This is especially true for non-porous implants that are not stably fixed to the underlying bone.

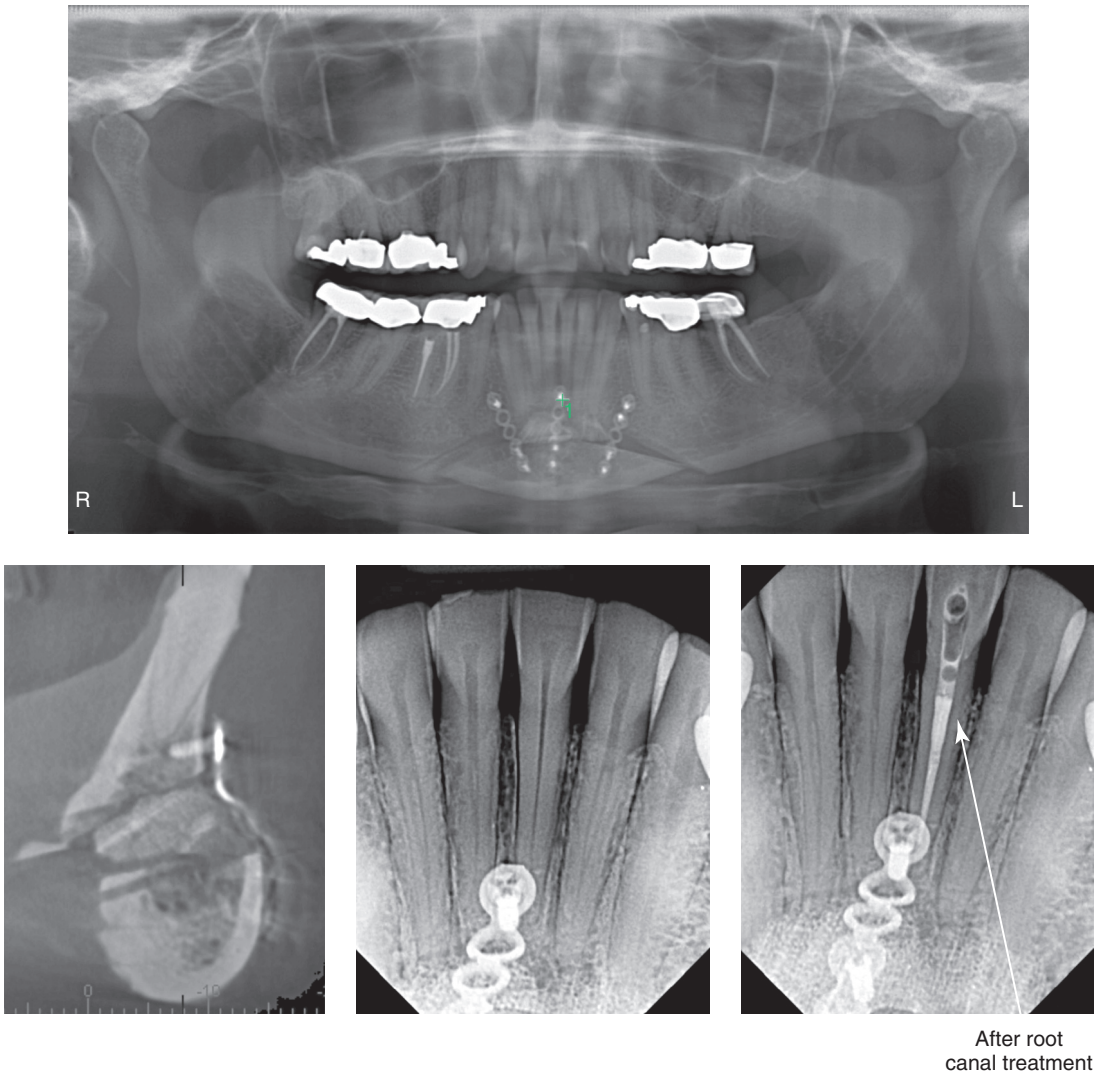
Altered Sensibility

The mental nerve is at risk of injury in association with an osseous genioplasty. The mental nerves will always be stretched when the soft-tissue dissection and retraction is carried out for exposure of the chin. The observed loss of sensibility to the lip–chin region early after surgery is usually temporary. The potential for cutting the mental nerve during the soft-tissue vestibular incision is limited by taking care during the lateral aspect of the incision. The nerve can also be avulsed from the mental foramen during retraction at the time of osteotomy. When determining a location for the osteotomy, remember that the nerve dips below the mental foramen posteriorly. According to the literature, as many as 10% of patients may experience a degree of permanent loss of sensibility in the distribution of the mental nerve on one or both sides as a result of genioplasty.

Wound Complications

The osseous genioplasty is completed through an intraoral vestibular incision. To limit wound-healing complications, the incision is made in the depth of the vestibule. Maintenance of the attached gingiva and a full cuff of mucosa and muscle adjacent to the cervical margins of the teeth best ensures a relaxed wound closure in two layers (i.e., the muscle and the mucosa). This will minimize wound dehiscence, muscle dysfunction, and chin ptosis and limit mucogingival problems. Placing the soft-tissue incision too close

*References 7, 10, 11, 17, 18, 20, 24, 25, 29, 32, 34, 49, 53, 54, 65, 72, 73, 75, 80, 82, 85, 98, 100, 113, 114, 117, 121, 133, 136, 137



• **Figure 37-21** An oblique osteotomy of the chin was carried out (vertical lengthening and horizontal advancement) with interpositional hydroxyapatite grafting. Postoperative radiographs indicate the closeness of the screw fixation to the lateral incisor (1.7-mm diameter and a 4-mm length). Darkening of the crown indicates pulpal injury. Root canal therapy and internal bleaching of the involved tooth were carried out.

to the teeth can negatively affect wound healing and result in scar bands and periodontal sequelae (e.g., mucogingival pull).

Injury to the Teeth

The location of the chin osteotomy should be based on the patient's chin morphology. The review of the patient's radiographs (e.g., lateral cephalometric, Panorex) will confirm the anatomy of the anterior teeth and the location of the mental foramen. Injury to the teeth at the time of osteotomy is not common, but it can occur. Dental injury may also occur as a result of the placement of the fixation screws, but this is also not common. If dental injury is suspected, evaluation by the patient's dentist or referral to an endodontist is recommended. Discussions among the surgeon, the dentist, and the patient are essential, because root canal therapy or other forms of treatment may be indicated (Fig. 37-21).

Patient Dissatisfaction

Patient dissatisfaction after an osseous genioplasty will generally revolve around facial aesthetics. This is best avoided via the following means:

1. Accurate preoperative facial and dental assessment by the surgeon and then discussion with the patient of the following: 1) the surgeon's findings; 2) the patient's goals; 3) any patient-specific biologic limitations (e.g., soft tissues, skeletal, dental); 4) any baseline head and neck dysfunction that may be related to the

chin (e.g., breathing, lip competence); and 5) the surgeon's overall recommendations
2. A clear understanding among all parties of the operative objectives and any compromises in treatment that have been jointly agreed upon
3. The meticulous execution of the procedures by the surgeon with the patient under controlled anesthesia and with proper instrumentation and assistance
4. At set time intervals after surgery, a discussion with the patient about the results achieved and any shortcomings perceived

Alloplastic Chin Augmentation

Successful chin implants are primarily placed to address mild deficiencies in the sagittal dimension at the pogonion and any limited hypoplasia immediately lateral to the symphysis.[128,129] Depending on the philosophy and skill of the surgeon, some vertical lengthening may also be possible.[130] Complaints after alloplastic chin augmentation are not infrequent, which may explain their decreased use by board-certified surgeons over a recent 10-year time frame.* Available statistics from the American Society of Aesthetic Plastic Surgeons confirm an 85% drop in the number of chin implants that were placed between 1997 and 2009 (i.e., 11,000 implants were placed in 1997, whereas 1700 were placed in 2009).[101] Although individuals often present with the specific complaint of a "weak chin" and with a seemingly simple request for a "chin implant," they frequently have a combination of microgenia, mandibular deficiency, and additional associated maxillofacial dysmorphology. A complete systematic soft-tissue and skeletal facial evaluation is required. The surgeon may choose not to discuss associated baseline maxillomandibular skeletal deformities either as a result of a lack of knowledge (i.e., an incomplete diagnosis) or because the patient or family "only requested a chin implant." In either case, after sagittal chin augmentation, a dissatisfied patient may express bewilderment with the result and at a minimum demand implant removal (see Figs. 37-1, 37-7, 37-9, and 37-10). When indicated, counseling the patient about the preferred orthodontic and orthognathic approach versus camouflage genioplasty alone is advised.

When the facial aesthetic analysis indicates that an implant is an acceptable alternative, a customized porous polyethylene chin augmentation with screw fixation is generally preferred.[104,128-130] Silicone (i.e., Silastic) implants heal with a surrounding fibrous capsule. Porous polyethylene implants heal with fibrous tissue in-growth into the open pores. The placement of an appropriately shaped implant with stable fixation will limit the risk of displacement, infection, and erosion of the underlying bone. The obliteration of dead-space gaps (i.e., between the implant and the skeleton) by custom contouring the implant and when applying compressive fixation screws before wound closure is essential. To review, experienced surgeons generally recommend the following: 1) placing a chin implant through a submental incision without intraoral exposure; 2) using an implant of porous material that is customized; and 3) applying stable screw fixation to increase success and limit complications.[128-130]

Despite the apparent technical ease of placing an alloplastic implant, a disproportionate number of repetitive problems seem to occur after a chin implant.[11,24,57,97] A common clinical scenario involves sagittal augmentation with an implant that is excessive in size and that does not address vertical aspects or associated jaw deformity. In these cases, it is generally not just the patient who perceives that the implant is too big; it is hard for the surgeon and other observers at conversational distance to deny the unfavorable aesthetic result, and thus surgeons are forced to honor their patients' request for implant removal. Unfortunately, depending on the material used, the implant may have caused capsule formation, splaying of the mentalis muscles, and stretching of the overlying soft-tissue envelope. After removal of the "too-large" implant, soft-tissue contracture may occur, which looks even worse with dynamic function (i.e., the soft-tissue "balling up" effect). If mentalis muscle dysfunction remains, there may be unattractive sagging (ptosis) of the soft tissues. Removing the implant and simply placing a smaller one has a low probability of satisfying the patient's concerns. Facial reassessment may clarify other skeletal deformities (e.g., primary mandibular deficiency, maxillomandibular deficiency, long face growth pattern) that account for the suboptimal result. Many times, after implant removal, a chin osteotomy with three-dimensional repositioning with or without combined orthognathic procedures will give the best result.

Frequent sequelae include improper initial positioning of the chin implant (i.e., not directly over pogonion) and inadequate fixation with lateral or superior shifting. These conditions may result in suboptimal aesthetics, erosion of the underlying bone, or compression of the mental nerve with dysesthesia of the lip–chin region. A late trauma event involving an otherwise initial satisfactory implant may lead to swelling and then infection. Late infection as a result of either bacteremia with secondary implant seeding or an adjacent dental infection can also occur. Management generally requires drainage, the removal of the implant, and the use of antibiotics. After resolution, further reconstructive options can be considered.

Conclusions

An osseous genioplasty can be a useful technique for the enhancement of facial aesthetics. Before an isolated genioplasty procedure can be considered, a thorough facial aesthetic and head and neck functional assessment—followed by a frank discussion with the patient and the family about

*References 8, 11, 14, 21, 24, 26, 27, 37, 41, 46, 50, 56, 57, 67, 68, 70, 72, 84, 86, 87, 97, 107-109, 115, 132

the ideal treatment and any compromised treatment agreed to—is essential. A review of the risks, possible complications, and hoped-for benefits of a chin reshaping procedure should result in a high degree of patient satisfaction. To limit the occurrence of a dissatisfied patient outcome, the setting of realistic and agreed-upon objectives is essential. The meticulous execution of the procedure by the surgeon with the patient under controlled anesthesia and with proper instrumentation and assistance further ensures success.

Patient Education Materials

Osseous Genioplasty (Chin Reshaping) Surgery

Many people are motivated to alter their chins when they see themselves in profile. For example, when the chin is small, it may undermine the entire face and make the nose look larger and more conspicuous. An osseous genioplasty is a procedure that is often carried out to reshape or change the size, projection, or appearance of the chin. The chin region may be surgically changed with regard to its length, projection, or symmetry.

An osseous genioplasty may balance your profile by enhancing or diminishing chin prominence in relationship to the nose, jaws, and lips. Strengthening the chin will tend to tighten the skin of the neck and generally maintain or restore a more youthful appearance. The surgical reshaping of the chin is carried out either as an isolated procedure or as part of other facial surgery (e.g., orthognathic procedures, rhinoplasty, neck lift). The procedure usually requires 1 hour and is carried out with the patient under anesthesia. The incision that is used to gain access to the chin is made on the inside of the lower lip. Depending on other procedures that are carried out simultaneously, most people return to work after 1 week.

INSTRUCTIONS FOR OSSEOUS GENIOPLASTY SURGERY

The purpose of these instructions is to help you prepare for and recover from your operation with as little discomfort and inconvenience as possible.

Preoperative Instructions

1. Do not take any aspirin, aspirin-containing, or aspirin-like products (e.g., Advil) for at least 2 weeks before surgery. Aspirin and aspirin-like products tend to increase bleeding during surgery and bruising postoperatively.
2. Be sure that you do not smoke and that there are no nicotine products in your bloodstream for at least 3 weeks before and 2 weeks after surgery. Nicotine in the bloodstream jeopardizes wound healing. Smoke that is chronically ingested by the lungs may also result in pulmonary complications that will hinder optimal healing.
3. Do not eat or drink anything after midnight the night before surgery, not even a sip of water. You may brush your teeth, but do not swallow the water.
4. Starting 2 days before surgery, we request that you shampoo your hair and shower at least once per day. The morning of surgery, you may also shower and shampoo your hair again.
5. We will give you a prescription for pain control and another for antibiotics. You may wish to fill them before the date of surgery.
6. Be sure to make arrangements for someone to accompany you home after surgery, because you will not be allowed to drive.

Postoperative Instructions

1. A cold compress applied in the neck and chin region should reduce swelling and discomfort for the first 24 to 36 hours after surgery. However, this is of little value after that time.
2. Keep the head of your bed elevated when resting and sleeping for the first week after surgery.
3. You may shower and shampoo your hair the day after surgery. Do not be afraid to wash your face and neck.
4. After discharge from the hospital, eat lightly (mostly liquids) and advance to a soft diet during the first week until further instructions are given.
5. You should begin brushing your teeth the day of surgery. Brush your upper teeth in the regular way and your lower teeth (including the anterior teeth) carefully and slowly. Do not brush the incision.

Continued

6. Keep your level of physical activity to a minimum, get lots of rest, and do not do anything that requires more than limited physical exertion during the first week after surgery. You will gradually increase your activity level, but no sports or exertional exercise are allowed for 5 (five) weeks from the date of surgery. Ask your surgeon for specific instructions with regard to physical activity.
7. Do not consume beverages that contain alcohol or drive a motorized vehicle while taking prescription pain medications.
8. When you are at home, you should take the prescribed pain medication as needed. Tylenol may be substituted when your pain has diminished. Avoid taking aspirin or aspirin-like products for an additional 2 weeks after surgery. Take the antibiotic medication as directed until the supply is exhausted.
9. Call the office for an appointment to see your surgeon on the date specified on your discharge instructions. If there are any concerns, contact your surgeon's office at any time, day or night. After hours, the answering machine will advise you about how to proceed.

Jeffrey C. Posnick, DMD, MD
Director, Posnick Center for Facial Plastic Surgery

Consent for Osseous Genioplasty Surgery

_____ **INSTRUCTIONS**

Initial here This is a document that has been prepared to help your surgeon educate and inform you about your chin reshaping (genioplasty) surgery and its risks as well as alternative treatment options. It is important that you read this document carefully and completely. Please initial each section on each page to indicate that you have read and understand it. In addition, please sign the consent for surgery on the last page as proposed by your surgeon.

_____ **INTRODUCTION**

Initial here Many people are motivated to alter their chins when they see themselves in profile. For example, when a chin is small, it may undermine the entire face and make the nose look larger and more conspicuous. Genioplasty is a surgical procedure that is often carried out to reshape or change the size, projection, or appearance of the chin.

Under anesthesia, in an operating room, a small incision is made on the inside of the lower lip. Then, with the use of specialized instruments, the bone of the chin is cut and then repositioned or reshaped in accordance with your specific needs as discussed with your surgeon.

Genioplasty can balance your profile by enhancing or diminishing chin prominence in relationship to the nose, jaws, and lips. Strengthening the chin may tighten the skin of the neck and generally result in a more attractive appearance. The surgical reshaping of the chin is carried out either as an isolated procedure or as part of other facial surgery (e.g., orthognathic procedures, rhinoplasty, neck lift). The surgery usually requires about 1 hour to complete. Most people return to work 1 week after surgery.

_____ **ALTERNATIVE TREATMENT**

Initial here Alternative forms of management include not doing anything to your chin at all. Some individuals may consider a chin implant option; this involves the placement of artificial material (i.e., a chin implant) directly over the surface of your bone. Risks and potential complications are also associated with this and other alternative forms of treatment. These are all issues to consider in detail before you select the approach that best suits your overall needs.

_____ **RISKS OF SURGERY**

Initial here Every surgical procedure involves a certain amount of risk. An individual's choice to undergo a specific procedure is based on the comparison of the risk to potential benefit. Although the majority of patients do not experience the complications detailed below, you should discuss each of these items with your surgeon to be sure that you understand the risks, potential complications, and consequences of the chin reshaping surgery planned for you.

_____ **SURGICAL ANESTHESIA**

Initial here The use of both local and general anesthesia involve risk. Complications and injuries are possible with all forms of anesthesia and sedation.

_____ **INFECTION**

Initial here Infection is not common after chin reshaping surgery. Treatment with antibiotics will begin just before surgery and is continued afterward surgery. If an infection does occur, additional treatment (e.g., a change in antibiotics, drainage, other procedures) may be necessary.

_____ **SCARRING**

Initial here Although good wound healing after the procedure is expected, abnormal healing or unattractive scars may occur within the skin, the deeper structures, and the mucous membranes. Additional treatment or procedures may be helpful to treat scarring or gingival (gum) problems.

_____ **ALTERATION OF FACIAL SENSIBILITY**

Initial here With a genioplasty procedure, the mental nerve is at risk for injury. Such an injury may result in diminished sensation of the lower lip, the skin of the chin, the gums around the anterior teeth, and the anterior teeth themselves. Gradually over the course of 6 to 12 months, the feeling improves and generally returns to close to normal. Approximately 10% of the time, a degree of permanent sensory loss in the lower lip, chin, gums, and teeth remains.

_____ **INJURY TO ADJACENT TEETH**

Initial here During the genioplasty procedure, injury to the roots of the anterior teeth or interference with the circulation (blood flow) to the dental and bone segments can occur. Devitalization (nerve damage) of the teeth, which may then require a root canal procedure, can occur. Infrequently, the teeth may be injured by the fixation screws. It is possible (but unlikely) that the loss of teeth may occur and later require additional treatment. These are not common events, but they must be mentioned as possible occurrences.

_____ **DISSATISFACTION WITH FACIAL APPEARANCE**

Initial here An individual's dissatisfaction with his or her facial appearance after genioplasty surgery is not common, but it can occur. Despite your surgeon's best efforts, on occasion, this type of surgery may result in visible deformities, asymmetries, or structural malposition. You may be disappointed if the results of the chin surgery do not meet your expectations. Additional treatment and procedures may be possible should the results be considered unsatisfactory.

Continued

_____ **FACIAL ASYMMETRY**

Initial here The human face is normally asymmetric. There can also be variation from one side of the face to the other with regard to the results achieved with a chin reshaping procedure. Additional surgery may be possible should the extent of asymmetry be of concern.

_____ **MEDICATIONS**

Initial here Medications, prescription drugs, and anesthetics, may cause drowsiness and a lack of awareness and coordination, which can be increased by the use of alcohol or non-prescription drugs. Your surgeon advises you not to operate any vehicles, automobiles, or other potentially hazardous devices while taking such medications and until you have achieved full recovery from the effects of these substances. We ask that you not drive yourself home after discharge from the hospital or to and from office appointments initially after surgery.

_____ **HEALTH INSURANCE**

Initial here Most health insurance companies exclude coverage for changing the shape of the chin (genioplasty) or for any complications that may arise from this surgery. Please carefully review your health insurance subscriber-information to clarify these issues.

_____ **FINANCIAL RESPONSIBILITIES**

Initial here The cost of surgery involves several charges for the services provided, including the fees charged by your surgeon (i.e., surgeon's fees); the cost of surgical supplies, laboratory tests, and the use of the operating room (i.e., hospital charges); and the cost of anesthesia (i.e., anesthesiologist's fees). Depending on whether the cost of surgery is covered by your medical insurance plan, you will be responsible for necessary copayments, deductibles, and all other charges that are not covered. Additional costs may occur should complications or an unfavorable result develop from the surgery. Any secondary surgeries and all charges involved with revision procedures (i.e., surgeon's fees, hospital charges, and anesthesiologist's fees) would also be your responsibility.

_____ **DISCLAIMER**

Initial here Informed-consent documents are used to communicate information about the proposed surgery and to disclose risks and alternative forms of treatment. The informed-consent process attempts to define principles of risk disclosure that should generally meet the needs of most patients in most circumstances.

Since every patient is unique, this informed-consent document should not be considered all-inclusive with regard to defining methods of treatment and potential risks encountered. Your surgeon will provide you with additional written and verbal information that is based on all of the facts of your particular case and the current state of medical knowledge.

Informed-consent documents are not intended to define or serve as the standard of medical care. Standards of medical care are determined on the basis of all of the facts involved in an individual case and are subject to change as scientific knowledge and technologic advances and practice patterns evolve.

It is important that you read the above information carefully and have all of your questions answered by your surgeon before signing the consent form on the next page.

CONSENT FOR OSSEOUS GENIOPLASTY SURGERY

1. I hereby authorize my surgeon, _____, and such assistants as may be selected by my surgeon to perform the following procedure(s):

   _____

   _____

2. I have received, read, and understand the following information sheets:
   - Osseous Genioplasty Surgery
   - Book Chapter about Genioplasty
   - Consent for Osseous Genioplasty Surgery
3. I recognize that, during the course of the operation, unforeseen conditions may necessitate different procedures then those listed above. I therefore authorize my surgeon, _____, and my surgeon's assistants or designees to perform such other procedures that are in the exercise of professional judgment necessary and desirable. The authority granted under this paragraph shall include all conditions that require treatment and that are not known to my surgeon at the time the procedure is begun.
4. I acknowledge that no guarantee has been given by anyone with regard to the results that may be achieved by the procedures to be carried out.
5. I agree not to use alcoholic beverages and unprescribed drugs and to avoid all sports and water activities for at least 5 (five) weeks and then to resume them only after approval by my surgeon.
6. I consent to the photographing of me (head and neck region) before, during, and after surgery. These photographs may be used by my surgeon for medical, scientific, or educational purposes now and in the future.
7. I agree to cooperate completely with the recommendations of my surgeon and to provide all medical and dental information about myself while I am under my surgeon's care. I realize that a lack of same could result in less than optimal care.
8. I consent to the disposal of any body tissues or medical devices that may be removed.
9. **THE FOLLOWING INFORMATION HAS BEEN EXPLAINED TO ME IN A WAY THAT I UNDERSTAND:**
   - THE PREVIOUSLY DESCRIBED PROCEDURES TO BE UNDERTAKEN
   - ALTERNATIVE PROCEDURES AND METHODS OF TREATMENT THAT I HAVE DECIDED AGAINST
   - THE SPECIFIC RISKS AND POTENTIAL COMPLICATIONS OF THE PROCEDURES THAT I PLAN TO UNDERGO

I CONSENT TO THE PROCEDURES AND THE ABOVE LISTED ITEMS (1 THROUGH 9). I AM SATISFIED WITH THE EXPLANATION GIVEN TO ME.

_____	_____
Patient (or Person Authorized to Sign for Patient)	Date
_____	_____
Witness	Date

References

1. Aufricht G: Combined nasal plastic and chin plastic: Correction of microgenia by osteocartilaginous transplant from large hump of nose. *Am J Surg* 25:292, 1934.
2. Aufricht G: Combined plastic surgery of the nose and chin. *Am J Oral Surg* 95:231, 1958.
3. Becking AG, Tuinzing DB, Hage JJ, Gooren LJG: Facial corrections in male to female transsexuals: A preliminary report on 16 patients. *J Oral Maxillofac Surg* 54:413, 1996.
4. Bell WH: Correction of the contour-deficient chin. *J Oral Surg* 27:110, 1969.
5. Bell WH, Brammer JA, McBride KL, Finn RA: Reduction genioplasty: Surgical techniques and soft-tissue changes. *Oral Surg Oral Med Oral Pathol* 51:471, 1981.
6. Bell WH, Dann JJ, III: Correction of dentofacial deformities by surgery in the anterior part of the jaws. *Am J Orthod Dentofacial Orthop* 64:162, 1973.
7. Bell WH, Gallagher DM: The versatility of genioplasty using a broad pedicle. *J Oral Maxillofac Surg* 41:763, 1983.
8. Brennan HG, Giammanco PF: The ptotic chin syndrome corrected by mentopexy. *Ann Plast Surg* 18:200, 1987.
9. Burstone CJ: Lip posture and its significance in treatment planning. *Am J Orthod* 53:262, 1967.
10. Clark CL, Baur DA: Management of mentalis muscle dysfunction after advancement genioplasty: A case report. *J Oral Maxillofac Surg* 62:611, 2004.
11. Cohen SR, Mardach OL, Kawamoto HK, Jr: Chin disfigurement following removal of alloplastic chin implants. *Plast Reconstr Surg* 88:62, 1991.
12. Collins P, Epker BN: Improvement in the augmentation genioplasty via suprahyoid muscle reposition. *J Maxillofac Surg* 11:116, 1983.
13. Connor AM, Moshiri F: Advancement genioplasty: An important part of combination surgery in Black American patients. *Am J Orthod Dentofacial Orthop* 93:92–99, 1988.
14. Dann JJ, Epker BN: Proplast genioplasty: A retrospective study with treatment recommendations. *Angle Orthod* 47:173, 1977.
15. Ellis E, III, Dechow P, McNamara J, et al: Advancement genioplasty with and without soft tissue pedicle. *J Oral Maxillofac Surg* 42:637, 1984.
16. Farkas LG, Sohm P, Kolar JC, et al: Inclinations of the facial profile: Art versus reality. *Plast Reconstr Surg* 75:509, 1985.
17. Feldman JJ: The ptotic (witch's) chin deformity: An excisional approach. *Plast Reconstr Surg* 90:207, 1992.
18. Field LM: Correction by a flap of suprahyoid fat of a "witch's chin" caused by a submental retracting scar. *J Dermatol Surg* 7:719, 1981.
19. Finn RA, Bell WH, Brammer JA: Interpositioning grafting with autogenous bone and coralline hydroxyapatite. *J Maxillofac Surg* 8:217, 1980.
20. Fitzpatrick BN: Genioplasty with reference to resorption and the hinge sliding genioplasty. *Int J Oral Surg* 3:247, 1974.
21. Flowers RS: Alloplastic augmentation of the anterior mandible. *Clin Plast Surg* 18:107, 1991.
22. Fogel ML, Stranc MF: Lip function: A study of normal lip parameters. *Br J Plast Surg* 37:542, 1984.
23. Freihofer HPM: Surgical treatment of the short face syndrome. *J Oral Surg* 39:907, 1981.
24. Friedland JA, Coccaro PJ, Converse JM: Retrospective cephalometric analysis of mandibular bone absorption under silicone rubber chin implants. *Plast Reconstr Surg* 57:144, 1976.
25. Gianni AB, D'Orto O, Biglioli F, et al: Neurosensory alterations of the inferior alveolar and mental nerve after genioplasty alone or associated with sagittal osteotomy of the mandibular ramus. *J Craniomaxillofac Surg* 30:295, 2002.
26. Gillies H, Kristensen HK: Ox cartilage in plastic surgery. *Br J Plast Surg* 4:63, 1951.
27. Godin M, Costa L, Romo T, et al: Gore-Tex chin implants: A review of 324 cases. *Arch Facial Plast Surg* 5:224, 2003.
28. Gonzalez-Ulloa M: Quantitative principles in cosmetic surgery of the face (profile plasty). *Plast Reconstr Surg* 29:186, 1962.
29. Gonzales-Ulloa M: Ptosis of the chin: The witch's chin. *Plast Reconstr Surg* 50:54, 1972.
30. Gonzales-Ulloa M, Stevens E: The role of chin correction in profile plasty. *Plast Reconstr Surg* 36:364–373, 1961.
31. Gonzalez-Ulloa M, Stevens E: The role of chin correction in profile plasty. *Plast Reconstr Surg* 41:477, 1968.
32. Gross BD, Moss RA: Cutaneous necrosis of the chin following mandibular advancement and genioplasty. (Case report). *J Maxillofac Surg* 6:140, 1978.
33. Guyuron B, Michelow BJ, Willis L: A practical classification of chin deformities. *Aesthetic Plast Surg* 19:257, 1995.
34. Guyuron B, Kadi JS: Problems following genioplasty. *Clin Plast Surg* 24:507, 1997.
35. Guyuron B, Raszewski RA: A critical comparison of osteoplastic and alloplastic augmentation genioplasty. *Aesthetic Plast Surg* 14:199, 1990.
36. Hambleton RS: Tissue covering of the skeletal face as related to orthodontic problems. *Am J Orthod* 50:405, 1964.
37. Heidingfield M: Histopathology of paraffin prosthesis. *J Cutan Dis* 24:513–521, 1906.
38. Hinds EC, Kent JN: Genioplastic: The versatility of the horizontal osteotomy. *J Oral Surg* 27:690, 1969.
39. Hofer O: Die operative behandlung der alveolare retraktion des unterkiefers und ihre anwendungsmoglichkeit fur prognathie und mikrogenie. *Dtsch Zahn Mund Kieferheilkd* 9:130, 1942.
40. Hofer O: Die osteoplastiche verlagerung des unterkiefers nach von Eiselberg bei mikrogenie. *Dtsch Zahn Mund Kieferheilkd* 27:81, 1957.
41. Hoffman S: Loss of a Silastic chin implant following a dental infection. *Ann Plast Surg* 7:484, 1981.
42. Hohl TH, Epker BN: Macrogenia: A study of treatment results, with surgical recommendations. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 41:545, 1976.
43. Holdaway RA: A soft tissue cephalometric analysis and its use in orthodontic treatment planning: Part I. *Am J Orthod Dentofacial Orthop* 84:1, 1983.
44. Holdaway RA: A soft-tissue cephalometric analysis and its use in orthodontic treatment planning. Part II. *Am J Orthod* 85:279–293, 1984.
45. Holmes RE, Wardrop RW, Wolford LM: Hydroxyapatite as a bone graft substitute in orthognathic surgery: Histologic and histometric findings. *J Oral Maxillofac Surg* 46:661, 1988.
46. Jobe R, Iverson R, Vistnes L: Bone deformation beneath alloplastic implants. *Plast Reconstr Surg* 51:169, 1972.
47. Joseph J: Verbesserung meiner hangewangenplastik (melomioplastik). *Dtsch Med Wochenschr* 54:567, 1928.
48. Kawamoto HK: Reduction mentoplasty [discussion]. *Plast Reconstr Surg* 70:151, 1982.
49. Kim SG, Lee JG, Lee YC, et al: Unusual complication after genioplasty. *Plast Reconstr Surg* 109:2612, 2002.
50. Klawitter JJ, Bagwell JG, Weinstein AM, Sauer BW: An evaluation of bone ingrowth into porous high density polyethylene. *J Biomed Mater Res* 10:311, 1976.
51. Krekmanov L, Kahnberg KE: Soft tissue response to genioplasty procedures. *Br J Oral Maxillofac Surg* 30:87, 1992.
52. Legan HL, Burstone CJ: Soft tissue cephalometric analysis for orthognathic surgery. *J Oral Surg* 38:744, 1980.
53. Lesavoy MA, Creasman C, Schwartz RJ: A technique for correcting witch's chin deformity. *Plast Reconstr Surg* 97:842, 1996.
54. Lindquist CC, Obeid G: Complications of genioplasty done alone or in combination with sagittal split-ramus osteotomy. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 66:13, 1988.
55. Loeb R: Surgical elimination of the retracted submental fold during double chin surgery. *J Aesthetic Surg* 2:31, 1978.
56. Matarasso A, Elias AC, Elias RL: Labial incompetence: A marker for progressive bone

resorption in Silastic chin augmentation. *Plast Reconstr Surg* 98:1007, 1996.
57. Matarasso A, Elias AC, Elias RL: Labial incompetence: A marker for progressive bone resorption in Silastic chin augmentation: An update. *Plast Reconstr Surg* 112:676, 2003.
58. McBride KL, Bell WH: Chin surgery. In Bell WH, Proffit WR, White R, editors: *Surgical correction of dentofacial deformities*, Philadelphia, 1980, WB Saunders, p 1210.
59. McCarthy JG: Microgenia: A logical surgical approach. *Clin Plast Surg* 8:269, 1981.
60. McCarthy JG, Kawamoto HK, Grayson BH, et al: Surgery of the jaws. In McCarthy JG, editor: *Plastic surgery*, Philadelphia, 1990, Saunders, pp 1332–1333.
61. McCarthy JG, Ruff GL: The chin. *Clin Plast Surg* 15:125, 1988.
62. McCarthy JG, Ruff GL, Zide BM: A surgical system for the correction of bony chin deformity. *Clin Plast Surg* 18:139, 1991.
63. McDonnell JP, McNeill RW, West RA: Advancement genioplasty, a retrospective cephalometric analysis of osseous and soft tissue changes. *J Oral Surg* 35:640, 1977.
64. McKinney P, Rosen PB: Reduction mentoplasty. *Plast Reconstr Surg* 70:147, 1982.
65. Mercuri LG, Laskin DM: Avascular necrosis after anterior horizontal augmentation genioplasty. *J Oral Surg* 35:296, 1977.
66. Michelow BJ, Guyuron B: The chin: Skeletal and soft-tissue components. *Plast Reconstr Surg* 95:473, 1995.
67. Millard DR: Chin implants. *Plast Reconstr Surg* 13:70, 1954.
68. Millard DR: Adjuncts in augmentation mentoplasty and corrective rhinoplasty. *Plast Reconstr Surg* 36:48, 1965.
69. Millard DR, Pigott R, Hedo A: Submental lipectomy. *Plast Reconstr Surg* 41:513, 1968.
70. Mole B: The use of Gore-Tex implants in aesthetic surgery of the face. *Plast Reconstr Surg* 90:200–206, 1992.
71. Mommaerts MY, Van Hemelen G, Sanders K, et al: High labial incisions for genioplasty. *Br J Oral Maxillofac Surg* 35:398–400, 1997.
72. Newman J, Dolsky RL, Mai ST: Submental liposuction extraction with hard chin augmentation. *Arch Otolaryngol* 11:454, 1984.
73. Nishioka GJ, Mason M, Van Sickles JE: Neurosensory disturbance associated with the anterior mandibular horizontal osteotomy. *J Oral Maxillofac Surg* 46:107, 1988.
74. Obwegeser H: The surgical correction of mandibular prognathism and retrognathia with consideration of genioplasty. *J Oral Surg* 10:677–689, 1957.
75. Omnell ML, Tong DC, Thomas T: Periodontal complications following orthognathic surgery and genioplasty in 19-year-old: A case report. *Int J Adult Orthodon Orthognath Surg* 9:133, 1994.
76. Ousterhout DK: Sliding genioplasty, avoiding mental nerve injuries. *J Craniofac Surg* 4:297, 1996.
77. Peterson RA: Correction of the senile chin deformity in face lift. *Clin Plast Surg* 19:433, 1992.
78. Polido WD, de Clairfont RL, Bell WH: Bone resorption, stability, and soft-tissue changes following large chin advancements. *J Oral Maxillofac Surg* 49:251, 1991.
79. Posnick JC: Aesthetic alteration of the chin: Evaluation and surgery. In Posnick JC, editor: *Craniofacial and maxillofacial surgery in children and young adults,* vol 43, Philadelphia, 2000, WB Saunders Co, pp 1113–1124.
80. Posnick JC, Al-Qattan MM, Stepner NM: Alteration in facial sensibility in adolescents following sagittal split and chin osteotomies of the mandible. *Plast Reconstr Surg* 97:920, 1996.
81. Putnam JM, Donovan MG: Modified reduction genioplasty. *J Oral Maxillofac Surg* 47:203, 1989.
82. Richard O, Ferrara JJ, Cheynet F, et al: Complications of genioplasty. *Rev Stomatol Chir Maxillofac* 102:34, 2001.
83. Ricketts RM: Esthetics, environment, and the law of lip relation. *Am J Orthod* 54:272, 1968.
83A. Riolo ML, Moyers RE, McNamara JA, Hunter WS: *An Atlas of Craniofacial Growth. In Monograph No. 2, Craniofacial Growth Series*, Ann Arbor, Mich, 1974, Center for Human Growth and Development, University of Michigan.
84. Rish BB: Profile-plasty. Report on plastic chin implants. *Laryngoscope* 74:144, 1964.
85. Ritter EF, Moelleken BRW, Mathes SJ, Ousterhout DK: The course of the inferior alveolar neurovascular canal in relation to sliding genioplasty. *J Craniofac Surg* 3:20, 1992.
86. Robinson M: Bone resorption under plastic chin implants: Follow-up of a preliminary report. *Arch Otolaryngol* 95:301, 1972.
87. Robinson M, Schuken R: Bone resorption under plastic chin implants. *J Oral Surg* 27:116, 1969.
88. Rosen HM: Surgical correction of the vertically deficient chin. *Plast Reconstr Surg* 82:247, 1988.
89. Rosen HM: Porous, block hydroxyapatite as an interpositional bone graft substitute in orthognathic surgery. *Plast Reconstr Surg* 83:985, 1989.
90. Rosen HM: Aesthetic refinements in genioplasty: The role of the labiomental fold. *Plast Reconstr Surg* 88:760, 1991.
91. Rosen HM: Aesthetics in facial skeletal surgery. *Perspect Plast Surg* 6:1, 1992.
92. Rosen HM: Facial skeletal expansion: Treatment strategies and rationale. *Plast Reconstr Surg* 89:798, 1992.
93. Rosen HM: Aesthetic guidelines in genioplasty: The role of facial disproportion. *Plast Reconstr Surg* 95:463, 1995.
94. Rosen HM: Autogenous chin augmentation. *J Plast Surg Tech* 115:22, 1997
95. Safian J: Progress in nasal and chin augmentation. *Plast Reconstr Surg* 37:446, 1966.
96. Satoh K, Tsukagoshi T, Shimizu Y: Surgical refinement of the operative procedure for a minor degree of mandibular prognathism. *Plast Reconstr Surg* 98:740, 1996.
97. Sclaroff A, Williams C: Augmentation genioplasty: When bone is not enough. *Am J Otolaryngol* 13:105, 1992.
98. Shaughnessy S, Mobarak KA, Høgevold HE, Espeland L: Long-term skeletal and soft-tissue responses after advancement genioplasty. *Am J Orthod Dentofacial Orthop* 130:8–17, 2006.
99. Simons RL, Lawson W: Chin reduction in profile plasty. *Arch Otolaryngol* 101:207, 1975.
100. Smith HW: Surgical correction of chin ptosis. *Am J Cosmetic Surg* 2:6–15, 1985.
101. Society for Aesthetic Plastic Surgery: 2009 Cosmetic Surgery National Data Bank American Statistics. New York.
102. Spear SL, Kassan M: Genioplasty. *Clin Plast Surg* 16:695, 1989.
103. Spear SL, Mausner M, Kawamoto HK: Sliding genioplasty as a local anesthetic outpatient procedure: A prospective two-center trial. *Plast Reconstr Surg* 80:55, 1987.
104. Spector M, Flemming WR, Sauer BW: Early tissue infiltrate in porous polyethylene implants into bone: A scanning electron microscope study. *J Biomed Mater Res* 9:537, 1975.
105. Steiner CC: Cephalometrics in clinical practice. *Angio Orthod* 29:8, 1959.
106. Storum KA, Bell WH, Nagura J: Microangiographic and histologic evaluation of revascularization and healing after genioplasty by osteotomy of the inferior border of the mandible, *J Oral Maxillofac Surg* 48:210–216, 1988.
107. Terino EO: Complications of chin and malar augmentation. In Peck G, editor: *Complications and problems in aesthetic plastic surgery*, New York, 1991, Gower Medical.
108. Terino EO: Alloplastic facial contouring by zonal principles of skeletal anatomy. *Clin Plast Surg* 19:487, 1992.
109. Terino EO: Versatile "chin implant" to alter the lower 1/3 facial segment. Presented at the Symposium, Advances in Aesthetic Plastic Surgery: The Cutting Edge II. New York, NY, November 12, 1998.
110. Trauner R, Obwegeser H: The surgical correction of mandibular prognathism and retrognathia with consideration of genioplasty: I. Surgical procedures to correct mandibular prognathism and reshaping of the chin. *Oral Surg* 10:677, 1957.
111. Trauner R, Obwegeser H: The surgical correction of mandibular prognathism and retrognathia with consideration of genioplasty. II. Operating methods for microgenia and distoocclusion. *Oral Surg Oral Med Oral Pathol* 10:787–792, 1957.

112. Tweed C: Frankfort-mandibular incisor angle (FMIA) in orthodontic treatment planning and prognosis. *Angle Orthod* 24:121, 1954.
113. Van Sickels JE, Hatch JP, Dolce C, et al: Effect of age, amount of advancement and genioplasty on neurosensory disturbance after a bilateral sagittal split osteotomy. *J Oral Maxillofac Surg* 60:1012, 2002.
114. Vedtofte P, Nattestad A, Hjirting-Hansen E, et al: Bone resorption after advancement genioplasty: Pedicled and non-pedicled grafts, *J Craniomaxillofac Surg* 19:102, 1991.
115. Wellisz T, Kanel G, Anooshian RV: Characteristics of the tissues response to Medpor porous polyethylene implants in the human facial skeleton. *J Long Term Eff Med Implants* 3:223, 1993.
116. Wessberg GA, Wolford LM, Epker BN: Interpositional genioplasty for the short face syndrome. *J Oral Surg* 38:584, 1980.
117. Westermark A, Bystedt H, Von Konow L: Inferior alveolar nerve function after mandibular osteotomies. *Br J Oral Maxillofac Surg* 36:425, 1998.
118. Wider TM, Spiro SA, Wolfe SA: Simultaneous osseous genioplasty and meloplasty. *Plast Reconstr Surg* 99:1273, 1997.
119. Wilcox JW, Hickory JE: Percutaneous total genioplasty. *J Oral Surg* 36:706, 1978.
120. Wolfe SA: Chin advancement as an aid in correction of deformities of the mental and submental regions. *Plast Reconstr Surg* 67:624, 1981.
121. Wolfe SA: (Discussion of) Chin surgery: I. Augmentation—The allures and the alerts, and Chin Surgery: II. Submental ostectomy and soft-tissue excision. *Plast Reconstr Surg* 104:1861, 1999.
122. Wolfe SA: (Discussion of) Sliding genioplasty as a local anesthetic outpatient procedure: A prospective two-center trial. *Plast Reconstr Surg* 80:67, 1987.
123. Wolfe SA: Shortening and lengthening the chin. *J Craniomaxillofac Surg* 15:223, 1987.
124. Wolfe SA: The genioplasty: An essential tool in the correction of chin deformities. In Ousterhout DK, editor: *Aesthetic contouring of the craniofacial skeleton*, Boston, 1991, Little, Brown, pp 409–430.
125. Wolfe SA, Berkowitz S: The chin. In Wolfe SA, Berkowitz S, editors: *Plastic surgery of the facial skeleton*, Boston, 1989, Little, Brown, pp 111–148.
126. Wolfe SA, Posnick JC, Yaremchuk MJ, Zide BM: Panel discussion: Chin augmentation. *Aesthet Surg J* 24(3):247–256, 2004.
127. Wolford LM: Porous, block hydroxyapatite as an interpositional bone graft substitute in orthognathic surgery. *Plast Reconstr Surg* 83:6, 1989.
128. Yaremchuk MJ: Mandibular augmentation. *Plast Reconstr Surg* 106:697, 2000.
129. Yaremchuk MJ: Facial skeletal reconstruction using porous polyethylene implants. *Plast Reconstr Surg* 111:1818, 2003.
130. Yaremchuk MJ: Improving aesthetic outcomes after alloplastic chin augmentation. *J Plast Reconstr Surg* 112:1422–1432, 2003.
131. Zeller SD, Hiatt WR, Moore DL, et al: Use of preformed hydroxyapatite blocks for grafting in genioplasty procedures. *J Oral Maxillofac Surg* 15:665, 1986.
132. Zide BM: Chin disfigurement following removal of alloplastic chin implants [discussion]. *Plast Reconstr Surg* 88:68, 1991.
133. Zide BM: The mentalis muscle: An essential component of chin and lower lip position. *Plast Reconstr Surg* 105:1213, 2000.
134. Zide BM, Boutros S: Chin surgery: III. Revelations. *Plast Reconstr Surg* 111:1542, 2003.
135. Zide B, Longaker MT: Chin surgery: II. Submental ostectomy and soft-tissue excision. *J Plast Reconstr Surg* 104:1854–1860, 1999.
136. Zide BM, McCarthy JG: The mentalis muscle: An essential component of chin and lower lip position. *Plast Reconstr Surg* 83:413, 1989.
137. Zide BM, Pfeifer TM, Longaker MT: Chin surgery: I. Augmentation—The allures and the alerts. *Plast Reconstr Surg* 104:1843, 1999.

38

Aesthetic Alteration of the Nose: Evaluation and Surgery

JEFFREY C. POSNICK, DMD, MD

- Nasal Surface Anatomy
- Classic Rhinoplasty Maneuvers
- Facial versus Nasal Proportions and Effects on the Aesthetic Perspective
- The Nasal Airway: Anatomy and Physiology
- Anatomic Locations and Causes of Nasal Obstruction
- Evaluation of the Nasal Airway
- Surgical Approach to Open the Nasal Airway
- Outcome Assessment of Surgery to Improve Nasal Breathing
- Initial Rhinoplasty Consultation
- Definitive Rhinoplasty Planning
- Open versus Closed Approach to the Nose
- Complications Associated with Rhinoplasty
- Primary Septorhinoplasty: Step-by-Step Approach
- Cleft Nasal Deformities
- Congenital and Posttraumatic Saddle-Nose Deformities
- Cocaine-Related, Autoimmune and Iatrogenic Saddle-Nose Deformities
- Conclusions
- Patient Education Materials

Nasal Surface Anatomy

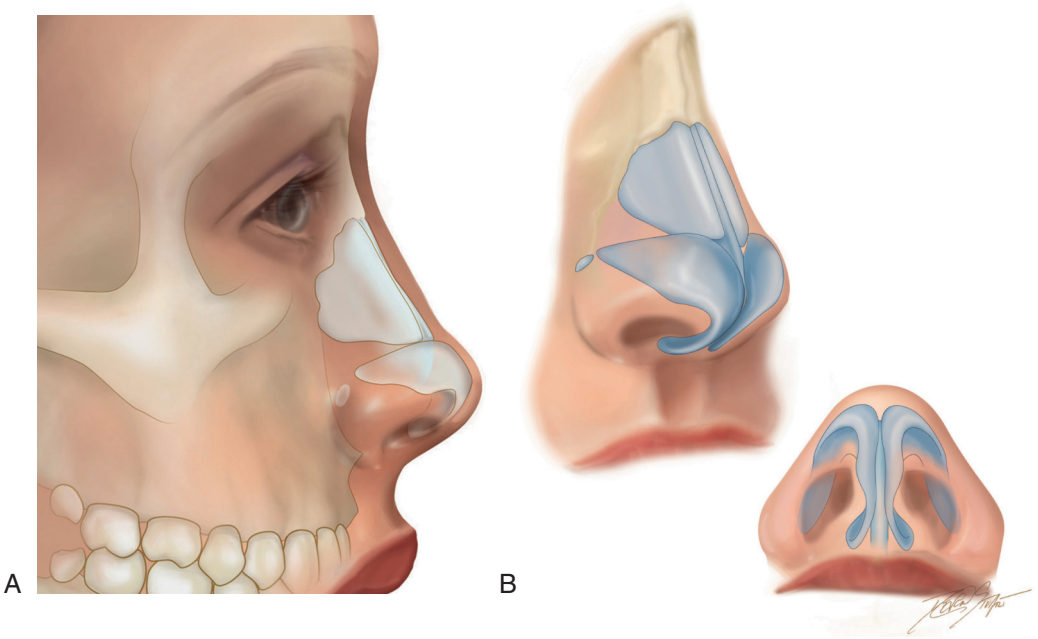
The external nose may be described as consisting of three parts: the osseocartilaginous vault, the lobule/tip, and the base (Fig. 38-1).

The *osseocartilaginous vault* consists of the bony vault (the nasal bones, the nasal process of the frontal bones, and the nasal process of the maxilla) and the cartilaginous vault (the upper lateral cartilages [ULCs] and the dorsal aspect of the cartilaginous septum). The transition from bony to cartilaginous dorsum is called the *keystone area.*[49] The caudal aspects of the nasal bones overlap the cephalic portions of the ULCs in the keystone area. The width of the nasal bones and ULCs is greater in the keystone area than at either the radix or the supratip regions.

Aesthetically, the dorsum of the nose is analyzed with regard to its height or anterior projection and its width and shape. Ideally, the dorsum of the nose, when viewed in profile, should be straight or slightly concave. The *nasofacial angle* is a soft-tissue measurement from the nasion to the tip and from the nasion to the glabella and should be approximately 34 to 36 degrees. The width of the *dorsal lines of the nose* should be roughly equal to that of the philtral columns. The maximum width of the bony vault (i.e., the dorsal width) of the nose is ideally less than that of the medial intercanthal width.

The *lobule of the nose* encompasses the entire area that overlies the lower lateral cartilages (LLCs), whereas the *intrinsic nasal tip* represents the area between the nasal-tip–defining points and the area between the infratip breakpoint and the supratip breakpoint. The ideal *tip-defining points* are approximately 6 to 8 mm apart in females and 8 to 10 mm apart in males. The LLCs are key anatomic points of reference that define the *nasal tip*. They are subdivided into the *medial crus,* the *middle crus,* and the *lateral crus.* Of aesthetic importance are the angles formed by the junction of the crura; these include the *columella infratip breakpoint* (i.e., the middle crura–medial crura angle) and the *dome tip-defining points* (i.e., the middle crura–lateral crura angle).

The *columella* is a critical region and component of the nose. It must be in balance between the adjacent alar rims and the medial crura. It should also form an attractive angle with the upper lip (i.e., the *nasolabial* or *columella–labial angle*). If the columella is either retracted or demonstrating excessive show, it will be less attractive and benefit from change (i.e., augmentation versus reduction).



• **Figure 38-1 A,** Illustration of the soft tissues and skeletal anatomy of the nose as seen in profile view. **B,** Illustration of the skeletal anatomy of the external nose and the lower lateral cartilages as seen in three-quarter and worm's-eye views.

The thickness and *quality of the soft tissues over the nasal skeleton* have a significant impact on the appearance and will also affect the final result of a rhinoplasty. In most individuals, the skin is thickest in the supratip region; it thins over the domal area.

To the experienced clinician, the appearance of the external nasal anatomy will give an indication of the anatomic causes of subjective nasal airway obstruction.[249] Middle-vault collapse, pinched nasal sidewalls, asymmetric nostrils, and a crooked dorsum are all outward indications of obstructions to nasal airflow; this is discussed later in this chapter.

Classic Rhinoplasty Maneuvers

Classic rhinoplasty maneuvers include the following 1) nasal osteotomies (i.e., to reduce the nose's width, to straighten a crooked nose, and to close an open roof) 2) dorsal reduction (bone and cartilage) 3) the selective removal of cephalic portions of the lower lateral crura (maintenance of 6 mm of caudal rim) 4) suturing of the LLCs (i.e., interdomal and transdomal areas) and 5) augmentation with cartilage grafts (i.e., caudal strut graft, alar contour grafts, lateral crural strut grafts, shield tip graft, columellar tip graft, spreader grafts, dorsal strut graft).[47,51,69,93,138,142-144,182,207,219,237]

Grafts are indicated when other lesser maneuvers to improve the projection and definition of the nasal tip are not adequate.* When indicated, a graft that will be used to alter the nasal tip can be fashioned from removed quadrangular cartilage or from another source (e.g., the conchal floor, the rib cartilage).[201] A *caudal strut graft* remains the workhorse for the addition of support to weak LLCs and for the control and maintenance of the nasal tip position. The caudal strut graft also functions as a central scaffold on which the tip structures can be unified. When a *shield tip graft* is used, it is placed over the domes with the inferior margin extending to the mid columella. A shield graft is effective for increasing infratip lobular definition and projection. *Columellar tip grafts* extend superiorly and are often secured to the ULC above and the caudal margin of the medial crura below. They are more useful in thick-skinned individuals in whom there is an additional need to improve tip definition beyond the usual rhinoplasty maneuvers. In thin-skinned individuals, tip grafts are only used cautiously, because they may result in visible irregularities.

Interdomal sutures are placed in mattress fashion in the region of the middle crura.[89,98,199] This suturing technique is effective for reducing interdomal distance and thereby increasing tip projection and refinement. *Medial crural fixation sutures* are placed to unify the medial crura beneath the domes of the LLC and to reduce flaring. These sutures are typically placed after a caudal strut graft is secured. *Transdomal sutures* are placed in a horizontal mattress fashion and then across the dome of the middle crura. All of these suture techniques can be effective to alter the tip position, to narrow the distance between the tip-defining points, and to correct domal asymmetries.

Spreader grafts may be either horizontal or vertical, and they are used to correct deficiencies of the cartilaginous vault.[24,38,46,87,88,92,95,97,208,209,255] *Vertical spreader grafts* are generally paired and extend from just deep to and then flush

*References 6, 21, 23, 36, 46, 48, 53, 70, 79, 90, 96, 99, 100, 108, 135, 156, 169, 174, 235, 239, 241

with the nasal bones. They are secured on each side of the dorsal septum and then generally to the ULCs with sutures. The caudal end of each vertical spreader graft may continue beyond the septal angle and below the LLCs to elongate the nose and to derotate the tip. They are typically 15 to 20 mm in length and approximately 2 to 4 mm in width. A *horizontal spreader graft* is a single dorsal graft that is inset between the ULCs and that forms a "T" with the dorsal septum. Spreader grafts are used as a technique to prevent or correct the narrowing of the internal nasal valve, to preserve or correct the nasal dorsum or dorsal aesthetic lines, and to correct and stabilize a "crooked" dorsal septum.

There are four aspects of the *nasal base* that may be considered for surgical modification[5]:

1. Selective reduction of the caudal aspect of the quadrangular cartilage (septum) may be completed to limit columella show in profile. Augmentation of the deficient caudal septum may be required after overaggressive rhinoplasty or to reconstruct congenital deficiencies (e.g., Binder syndrome).
2. Modification of the anterior nasal spine may be carried out to alter the nasolabial angle or to augment a congenital deficiency (e.g., Binder syndrome).
3. Deepening of a high nasal floor and widening of narrow pyriform apertures by recontouring (e.g., using a rotary drill with a bur) may be indicated to improve the airway and to enhance aesthetics (i.e., in those with a long face growth pattern). Augmentation of this region may also be carried out to reconstruct a deficiency (e.g., Binder syndrome, alveolar clefting).
4. A cautious approach to excision of the alar rims (i.e., Weir excisions) may be performed to achieve a preferred interalar width (i.e., just less than the intercanthal width). It is preferred to accept a slightly increased interalar width rather than to end up with an "operated" look from either overresection of the rims or resulting scarring.

Facial versus Nasal Proportions and Effects on the Aesthetic Perspective

The surgeon must distinguish between the *aesthetics of the nose* (i.e., nasal proportions) and the *aesthetics of the face* (i.e., overall facial proportions) (Fig. 38-2).* Establishing the *Euclidean proportions* of the full face represents a different aesthetic objective than simply establishing pleasing proportions of the nose as an isolated aesthetic unit. The individual who is seeking rhinoplasty often perceives that his or her aesthetic objectives can be met by addressing the nose only. In these circumstances, after cosmetic rhinoplasty, the surgeon may be pleased with improvements in the nasal aesthetic unit, but the patient may express displeasure with the results. This will more frequently occur in the individual with either a long face growth pattern, a short face growth pattern, or an isolated sagittal mandibular deficiency growth pattern who perceives that he or she still has a "big" nose, even after rhinoplasty (Fig. 38-3). The patient may have initially sought out a cosmetic surgeon and requested a rhinoplasty with the goal of achieving a "smaller" nose. The surgeon may create a well-proportioned nasal aesthetic unit, but the nose may still remain "big" in proportion to the rest of the face. The surgeon may then be asked to revise the rhinoplasty to create a "smaller" nose.[192,193] When the surgeon further reduces the nasal bones and cartilage beneath the soft-tissue envelope, an "operated" look may result, and the overall facial proportions may move even further out of balance.

*References 163-165, 186, 192, 203, 205, 211, 248

The Nasal Airway: Anatomy and Physiology

Symptomatic nasal obstruction is a condition in which the individual feels that he or she has inadequate nasal airflow. Although it is a subjective feeling, it should have a foundation in objective anatomic and physiologic findings (Fig. 38-4). Knowledge of both nasal anatomy and physiology is required for the accurate diagnosis and treatment of the subjective sensation of obstructed nasal breathing.*

Bony Anatomy

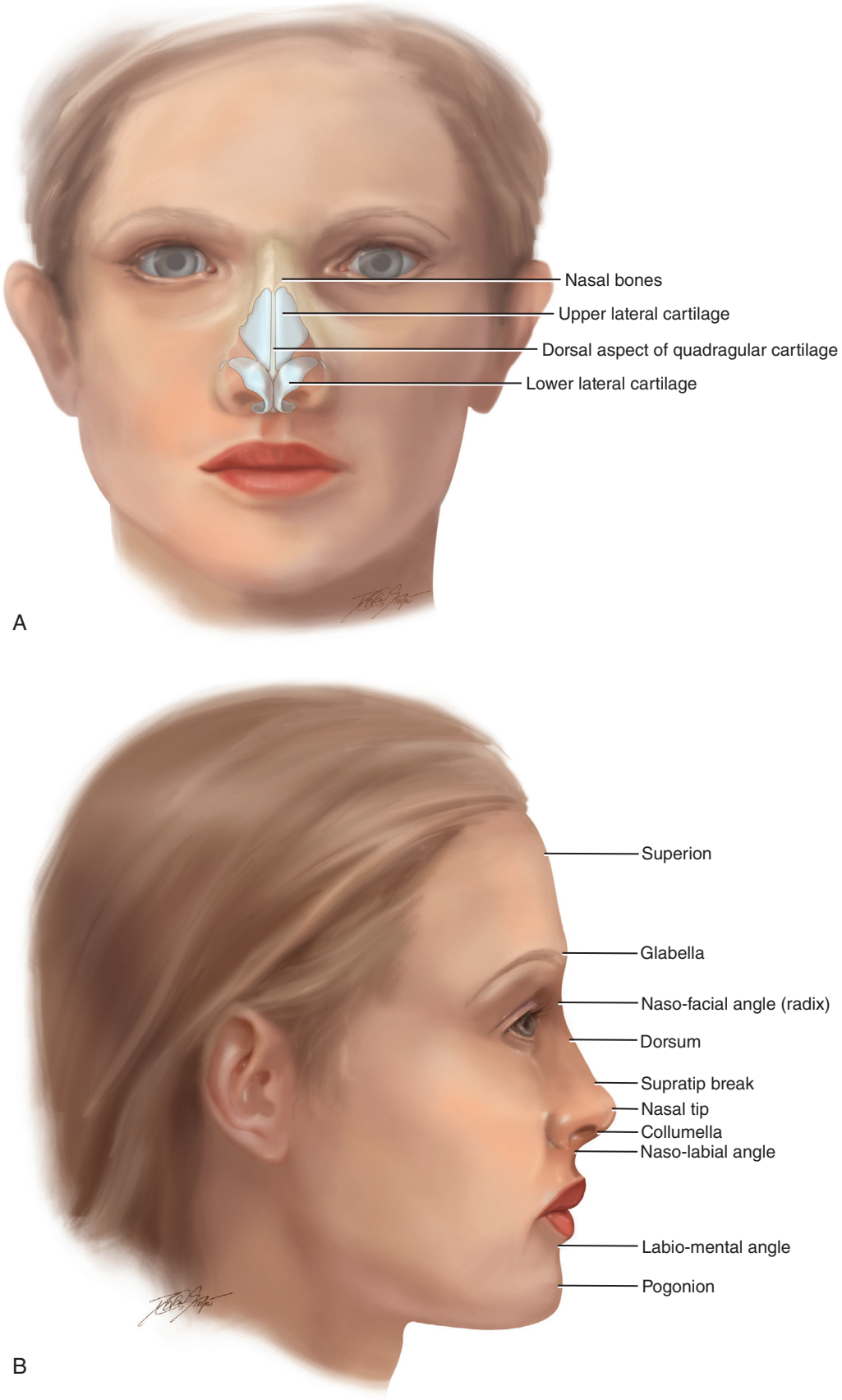
The *anterior (pyriform) aperture* generally represents the narrowest bony aspect of the nasal airway.[123] Its anatomic boundaries include the paired nasal bones (i.e., the nasal pyramid), which join to the ascending process of the maxilla laterally. The *bony floor of the nose* is formed by the superior aspect of the premaxillary alveolus anteriorly and the secondary palate posteriorly. The *anterior nasal spine* is in the anterior midline of the nasal floor, and the *posterior nasal spine* is in the posterior midline of the nasal floor. These spines provide the anterior and posterior limits of the inferior aspect of the cartilaginous and bony septum, respectively.

The *posterior (choanae) aperture* has a medial wall that is formed by the *vomer,* which is the posterior aspect of the nasal septum. The floor of the choanae is formed by the posterior aspect of the hard palate. The lateral walls of the posterior aperture are formed by each lateral maxillary process.

The *posterior inferior septum* is composed of the vomer, and it is contiguous with the *perpendicular plate of the ethmoid* superiorly. The anterior septum is composed of the

Text continued on p. 1620

*References 1, 9, 11, 15, 17, 27, 29, 30, 34, 39, 40, 44, 45, 55, 57, 62, 80-82, 85-87, 94, 101, 105, 110, 121, 124-126, 131, 139, 141, 146, 161, 162, 179, 197, 206, 213, 231

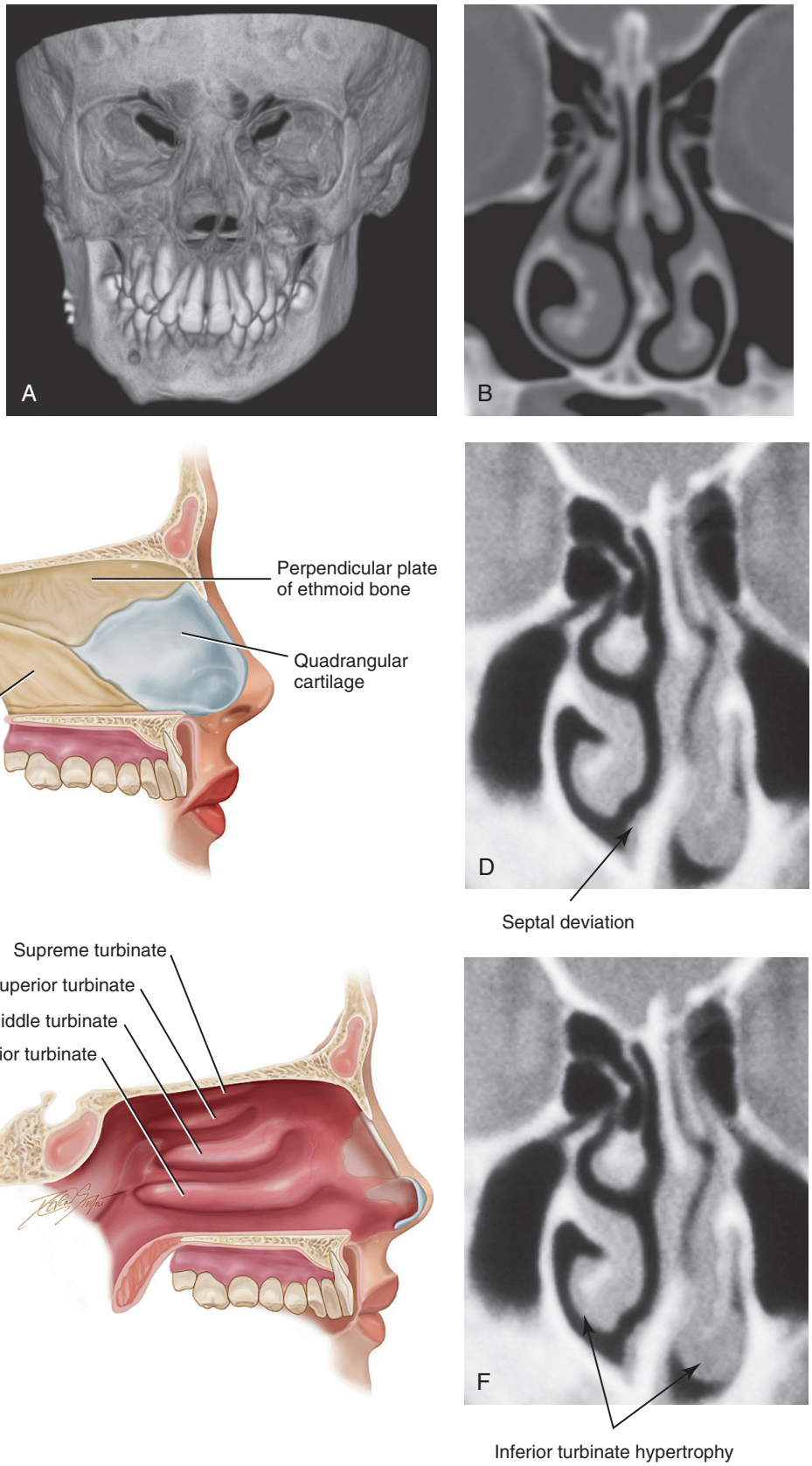


• **Figure 38-2 A,** Frontal and **B,** profile views of a well-proportioned face, including the underlying skeletal anatomy, are illustrated.

• **Figure 38-3** The clinician must distinguish between the aesthetics of the nose (i.e., the nasal aesthetic proportions) and the aesthetics of the face (i.e., the facial aesthetic proportions). Establishing the Euclidean proportions of the overall face represents a different aesthetic objective than simply establishing pleasing proportions of the nose as an isolated aesthetic unit. The individual who is seeking rhinoplasty often perceives that his or her objectives can be met by addressing the nose only. Errors in aesthetic judgment are especially frequent when viewing an individual with the following uncorrected issues: 1) a long face growth pattern (see Chapter 21); 2) a primary mandibular deficiency growth pattern (see Chapter 19); 3) a short face (maxillomandibular deficiency) growth pattern (see Chapter 23); or 4) a maxillary deficiency with a relative mandible excess growth pattern (see Chapter 20). Four case examples are shown to illustrate the importance of aesthetic perspective. **A,** A woman in her mid 20s with a long face growth pattern is shown before and after undergoing successful orthognathic surgery to correct facial disproportion. Before surgery, the nasal aesthetic unit was reasonably well proportioned but appeared "big" when viewing the face in profile. After successful orthognathic surgery, the same nose appears well proportioned with respect to the rest of the face. The procedures performed included Le Fort I osteotomy, sagittal split ramus osteotomies, and osseous genioplasty (see Fig. 21-5). **B,** A woman in her late 20s requested the evaluation of a "big" nose and a "small" chin. After successful orthognathic correction, which included sagittal split ramus osteotomies and osseous genioplasty the nose appears well proportioned with respect to the rest of the face (see Fig. 19-7).

Continued

• **Figure 38-3, cont'd C,** A teenage girl with a short face growth pattern (maxillomandibular deficiency with excess overjet) had previously undergone orthodontics to neutralize the occlusion. She complains of a "big" nose and a "small" chin. She is shown before and after successful orthognathic surgery that included Le Fort I osteotomy (horizontal advancement, vertical lengthening, and clockwise rotation), sagittal split ramus osteotomies (horizontal advancement, and clockwise rotation), and osseous genioplasty (vertical lengthening). After surgery, the nose no longer appears big and is well proportioned with respect to the rest of the face (see Fig. 23-2). **D,** A 20-year-old college student with a maxillary deficiency and a relative mandibular excess growth pattern arrived for a surgical evaluation with complaints of a "big" nose and "prominent" chin. He is shown before and after successful orthognathic surgery. The patient's procedures included Le Fort I osteotomy (arch expansion, horizontal advancement, vertical lengthening, and clockwise rotation) and sagittal split ramus osteotomies (clockwise rotation). After surgery, the nose no longer appears to be prominent, and the chin region of the lower jaw assumes improved proportions within the face (see Fig. 20-4).



• **Figure 38-4** **A,** A three-dimensional computed tomography (CT) scan view of a mixed-dentition skeleton with a deviated septum. **B,** A coronal CT view through the nasal cavity and maxilla demonstrates a deviated septum and enlarged inferior turbinates. **C,** A sagittal section through the nasal cavity and the maxilla is illustrated. The anatomy of the septum is highlighted. **D,** A CT scan through the nasal cavity (coronal cut) demonstrates a deviated septum. **E,** A sagittal section through the nasal cavity and the maxilla is illustrated. The anatomy of the turbinates is highlighted. **F,** A coronal CT view through the nasal cavity demonstrates a deviated septum and enlarged inferior turbinates.

quadrangular cartilage, which is contiguous with the bony septum.

Attached to the lateral nasal walls are three paired "scroll-like" bones that are known as the *turbinates.* The superior, middle, and inferior turbinates are composed of *conchal bones* that support the *erectile mucosal tissue* of the turbinates. Just inferior to each turbinate is a space called a *meatus.* Each of the paired sinus cavities drains into either the superior or the middle meatus region. The inferior meatus drains the *nasolacrimal duct.* The middle meatus provides the drainage pathway for the anterior ethmoid, maxillary, and frontal sinuses. The superior meatus is the drainage region for the posterior ethmoid and sphenoid sinuses. The majority of the air that passes through the nose is at a line below the middle turbinate.

Cartilaginous Anatomy

The cartilaginous septum joins to the bony portion of the septum (i.e., the vomer and perpendicular plate of the ethmoid) and the nasal crest of the maxilla inferiorly. The caudal aspect of the cartilaginous septum that joins to the maxillary crest does so through densely interwoven fibers of periosteum and perichondrium. The cartilaginous septum is called the *quadrangular cartilage* because of its geometric shape. Even small deviations or deflections of the septum can significantly alter nasal airflow. A frequent surgical procedure involves the removal of portions of the quadrangular cartilage that are obstructive to nasal airflow or for the purpose of grafting either the nasal tip or vault. During this procedure, it is important to maintain adequate dorsal and caudal struts of the quadrangular cartilage, which are responsible for the strength and support of the cartilaginous vault.

The cartilaginous anatomy of the nose also includes the *paired ULCs,* the *paired LLCs,* and the variable *sesamoid cartilages* (see Fig. 38-1). The ULCs are trapezoidal in shape and articulate with the nasal bones superiorly, with the quadrangular cartilage over the midline dorsum, with limited connections laterally to the bony pyriform aperture, and also to small sesamoid cartilages inferiorly. The LLCs connect to the ULCs superiorly via fibrous tissue. The LLCs are responsible for much of the form and shape of the nasal tip and the ala. Components of the LLC include the medial, middle, and lateral crura (see Fig. 38-4).

Intranasal Anatomy

Within the nose, sites of obstruction tend to occur at specific locations, including the *external nasal valves,* the *internal nasal valves,* the *turbinated cavities,* and the *posterior nasal (choanae) apertures* (see Fig. 38-4).

The *external nasal valve* is described as the area bounded by the caudal edge of the ULC superolaterally, the nasal ala and attachment of the lateral crus of the LLC laterally, the caudal aspect of the cartilaginous septum and the soft tissue of the columella medially, and the nasal sill and bony floor inferiorly. The size of the opening is variable and dependent on the shape, location, and strength of the bordering structures.

Generally, the site of greatest resistance to the entrance of air into the entire human airway is the *internal nasal valve.* Anatomically, the internal nasal valve is bordered superiorly by the ULC, medially by the cartilaginous nasal septum, laterally by the anterior aspect of the inferior turbinate, inferiorly by the nasal floor, and also laterally by the anteromedial aspect of the maxilla (i.e., the pyriform rim).

Mucosal Anatomy

The lining of the nasal cavity is composed primarily of epithelium. Along the superficial aspect of the external nasal valve, keratinized stratified squamous epithelium (i.e., skin) is present. Within the nose, the mucosal lining transitions from respiratory epithelium with goblet cells that secrete mucin to basal cells, columnar cells (ciliated and non-ciliated), and granule cells. Mucin glands are scattered throughout the nasal airway. The sinuses, in combination with the nasal cavity mucosa, produce between 1 and 2 liters of mucin each day. In the superior aspect of the nasal cavity are located specialized neuroepithelial cells that contain bipolar olfactory receptor neurons and mucin glands.

Nerve Supply

In addition to general sensory nerve supply through the first and second divisions of the trigeminal nerve, there is autonomic and sympathetic innervation to the nasal cavity.

Nasal Physiology and Function

The five major functions of the nose are *respiration, filtration, humidification, protection,* and *temperature regulation. Filtration* is accomplished through the vibrissae (located in the vestibule) and the mucosal cilia, which line the airway. *Humidification* and *temperature regulation* of the air are controlled by specialized vascular tissues that are regulated by sympathetic and parasympathetic nerve fibers. The capillary system is supplemented by venous sinuses that may constrict and result in enlargement, especially of each inferior turbinate.[103] *Protection* from bacteria and viruses is both mechanical and chemical. Chemical protection is provided by immunoglobulin A and muramidase, which are found in the mucus on the membranes of the nasal cavities. Mechanical clearing of trapped organisms is provided by the beating cilia, which move the mucous blanket into the nasopharynx every 20 minutes. During *respiration,* the nose resists the inspiration of air by providing almost 50% of the total airway resistance.

Airflow in the nasal cavity is very much related to resistance. The *airflow resistance* in the nasal cavity is

somewhat artificially divided into regions that include the external nasal valve, the internal nasal valve, the turbinated cavity, and the posterior nasal (choanae) aperture. The *nasal valves* comprise the major areas of resistance in the nasal cavity. The internal nasal valve is generally the "flow-limiting" segment of the nasal airway. According to Poiseuille's law, even small changes in the size of the internal nasal valve can have exponential effects on the airflow resistance. The size of the nasal airway is also governed by the physiologic alternating pattern of congestion and decongestion called the *nasal cycle.* This pattern of vascular engorgement of the capillaries involves the nasal lining in general and the vessels of the inferior turbinates specifically.

Anatomic Locations and Causes of Nasal Obstruction

The etiology of nasal airway obstruction is often multifactorial and may include both anatomic aspects and physiologic effects of the nasal and sinus mucosa.

Internal Nasal Valve

The *internal nasal valve* is typically the site of maximum resistance and frequently the cause of subjective complaints about obstructive nasal breathing. Obstruction that worsens during inspiration (i.e., dynamic obstruction) is usually caused by a weakening of the cartilaginous vault (i.e., the collapse of the ULCs and the LLCs) that is unable to withstand the inspiratory pressures. Static obstruction of the internal nasal valve occurs as a result of encroachment by the boundary structures of the valve. This is often the result of enlargement or hypertrophy of the inferior turbinates; deflection (i.e., thickening) of the nasal septum; medially located lateral nasal walls (i.e., maxillary constriction); an elevated nasal floor (i.e., long face growth pattern); or thickened and scarred nasal mucosa (i.e., as a result of allergies). During rhinoplasty, internal nasal valve collapse is prevented by avoiding the resection of the ULCs or the overresection of the LLCs. When either of these conditions is the cause, secondary reconstruction via the placement of spreader grafts is generally helpful.

External Nasal Valve

Common causes of static narrowing of the *external nasal valve* include alar malpositioning or scarring of the skin or nasal vestibule, caudal septal deflections, and nasal tip ptosis (see Fig. 38-4).[77] These may be seen after overzealous cleft lip nasal procedures. Dynamic collapse of the external nasal valve may be the result of weakened or deficient LLCs and nasal ala. During rhinoplasty, external nasal valve collapse is generally prevented by avoiding overaggressive resection of the LLCs and by preserving the soft-tissue support of the mobile alar walls. Secondary reconstruction to correct this deformity requires cartilage support grafts that are applied to the LLCs.

Posterior Nasal (Choanae) Apertures

Obstruction of the *posterior nasal apertures* may result from the skeletal structures that compose the choanae (i.e., the vomer and the palatine bones) or from the overlying soft tissues (i.e., polyps, adenoids, and turbinates).[210] Choanal atresia may also be associated with congenital anomalies such as CHARGE syndrome, Treacher Collins syndrome, and Apert syndrome.

Middle Turbinate Concha Bullosa

It is known that the majority of airflow through the nose enters the middle meatus, which is directly beneath the middle turbinate. A pneumatized *middle turbinate* (i.e., concha bullosa) is a common anatomic variation that occurs in approximately 25% of the population. If the concha bullosa is extremely large and associated with septal deviations, it may contribute to nasal obstruction.

Physiologic Causes of Nasal Obstruction

Both *benign and malignant neoplasms* may occur anywhere in the sinonasal tissues and should always be ruled out. *Inflammation* of the nasal mucosa is a common multifactorial process that results in edema and congestion of the nasal soft tissues. For example, *nasal polyposis* is thought to be triggered by a combination of factors (e.g., inflammation, asthma, allergy). If they are large enough, polyps may obstruct the nasal airway and the sinus ostium and result in blockages and sinus infections. *Allergies* are a common cause of sinoatrial inflammatory disease leading to turbinate hypertrophy, which further exacerbates ongoing complaints of nasal obstruction. *Hormonal changes* associated with pregnancy, menopause, and hypothyroidism are also known to have effects on the nasal mucosa and vasculature, with resulting obstructive nasal breathing. In an attempt to improve the symptoms of nasal obstruction, the overuse of prescription and over-the-counter *medications* (e.g., drugs from the imidazoline class, sympathomimetic amines) can also cause significant problems. The "rebound nasal congestion" phenomenon is well known and typically occurs with chronic use.

Evaluation of the Nasal Airway

Among the pertinent findings that require clarification from the patient are a history of nasal obstruction, sinusitis, or inflammatory sinonasal disease and the presence of a limited sense of smell or taste. A history of environmental or seasonal allergies, nasal surgery, or trauma should be correlated with physical findings.

With regard to the nasal airway, a complete external and internal nasal examination should be undertaken. Direct

and speculum examination of the anterior septum, the head of the inferior turbinates, and the external nasal valves should proceed. Nasoendoscopy allows for a more thorough examination of the nasal airway and should be undertaken when indicated to fully visualize the septum, the turbinates, the meatus, the nasal mucosa, the nasal valves, the posterior aperture, and other nasal structures. Decongestants may also be used to further assess the airway. For cases in which radiologic studies are needed, a coronal sinus computed tomography scan is useful.

The functional aspects of the nose that a rhinoplasty surgeon may typically alter include the nasal valve areas and the turbinated cavity. When completing a septoplasty procedure with the removal of posterior bony septal deflections, it is important to avoid injury to the cribriform plate, because cerebrospinal fluid rhinorrhea would result. As previously stated, when performing a septoplasty that includes the removal of deflected portions of quadrangular cartilage, it is important to maintain adequate caudal and dorsal struts for support of the nasal dorsum and tip.

The *internal nasal valve* is defined by both the valve angle (i.e., the relationship of the ULCs to the septum), which is typically 10 to 15 degrees, and the valve circumference. Respiratory obstruction can occur when the valve area is decreased by septal deviation, mucosal disease (i.e., engorgement of the inferior turbinates), scarring, and traumatic, neoplastic, congenital, or surgical deformities (e.g., narrow pyriform rim width, elevated nasal floor).

An example of effective reconstruction occurs when the internal nasal valves are collapsed after the surgical overresection of the ULCs, and *spreader grafts* (placed between the nasal septum and the ULC) are used to widen the internal valve angles. This will decrease nasal airway resistance and improve respiration.

Nasal airway resistance is greatly affected by the inferior turbinates, which periodically swell and decrease the internal valve area. Inferior turbinate hypertrophy may be physiologic or pathologic. For the majority of the population, a *normal "nasal cycle"* of the congestion and decongestion of the turbinates and the nasal mucosa is ongoing, with reciprocal changes on the two sides. The *total nasal airway resistance* in the healthy nose is remarkably constant, with changes occurring every 2 to 4 hours in the turbinates and the mucosal lining. When the inferior turbinates are markedly enlarged and when the process is not reversible with the use of vasoconstrictors and anti-inflammatory agents, then surgical reduction of the turbinate hypertrophy (often in combination with septoplasty) is the treatment of choice and will generally relieve the symptoms. Doing so will often open the internal nasal valve and the whole turbinated cavity. A variety of surgical techniques are used to accomplish the goal of inferior turbinate reduction (e.g., partial excision, electrocautery, submucosal resection, radiofrequency ablation, microdebridement).[157,173] The overaggressive resection of the inferior turbinates has, on occasion, resulted in rhinitis sicca or "dry nose."

Surgical Approach to Open the Nasal Airway

The clarification of any anatomic structures of the *internal nasal valve* that are causing blockage should be identified. This may include the following: 1) the septum 2) the anterior head of the inferior turbinates 3) the bony nasal floor 4) the lateral nasal walls (i.e., the tight inlet) 5) the ULCs; and 6) the nasal mucosal lining (i.e., the synechiae).

The identification of any anatomic components that interfere with the *external nasal valve* is also clarified and the areas surgically corrected, if feasible. This may require reconstruction of the LLCs, via elevation of the nasal tip or through the release of scar contracture of the nasal skin or the vestibular mucosa.

The opening of the *posterior nasal (choanae) aperture* may require the following: 1) the reduction of the adenoid pad; 2) the recontouring of the posterior floor of the nose and the lateral nasal walls; 3) the resection of the vomer and the perpendicular plate of the ethmoid; or 4) the horizontal (sagittal) advancement of the maxilla (i.e., Le Fort I osteotomy).

The partial removal (by direct excision, submucosal resection, or cauterization) of *hypertrophied middle turbinates* can, at times, significantly improve a patient's nasal airflow. If the middle turbinate is pneumatized (concha bullosa) and a cause of nasal obstruction found, it should be reduced by whatever means necessary.

Outcome Assessment of Surgery to Improve Nasal Breathing

Attempts to objectively measure alterations in nasal function after medical or surgical interventions that are carried out for the purpose of improving nasal breathing have proven to be difficult.[31,113-115,118,128,152,171,185,187,189,190,195,215,222,223,226,243] Through acoustic rhinometry and rhinomanometry, attempts at objective postoperative assessments of the nasal airway have been carried out. Consistent findings with the use of these measurement techniques are mixed at best. A spectrum of studies by different investigators has demonstrated conflicting results with similar research designs. Studies by Lindemann and colleagues confirm that nasophysiologic functions (e.g., the heating and humidifying of inspired air) were improved after successful septoplasty and appropriate inferior turbinate reductions.[126] This positive effect is believed to be derived from increased volumes and the direction of physiologic airflow to key places within the nasal passage, which increases mucosal contact with the nasal airstream. The improved flow dynamics may also improve the filtration of particulate matter more effectively.

In addition to objective instrumentation measures of nasal breathing, researchers have sought a standardized assessment of results from the patient's perspective. These

efforts have taken the form of symptom-based questionnaires. The most rigorously developed disease-specific measure for nasal obstruction was created by the *Nasal Obstruction Symptom Evaluation* (NOSE) study investigators.[233,234] The NOSE Scale generates an integral patient-specific score out of a possible 100 points and thus facilitates quantitative comparisons within subjects at different time points or among groups of subjects who are undergoing different treatments. *The NOSE Scale is now considered the reference standard for the assessment of alterations in nasal breathing.* Considering the extent of nasal obstructive treatment carried out in the United States alone, which is estimated to cost $5 billion per year, emphasis on standardization and measurable results seems to be indicated (see Chapter 10).

Initial Rhinoplasty Consultation

During the initial consultation, it is important for the patient to confirm any functional nasal difficulties and also to explain his or her facial aesthetic concerns. The individual may want a nose that is narrower (i.e., dorsal width reduction), less irregular (i.e., dorsal hump removal) and with a more attractive nasal tip (i.e., improved definition and projection). A frequently stated objective—or at least a personal desire that is expressed when an in-depth history is taken—is to have the ability to breathe better.

Aesthetic concerns often relate to an enlarged *osseocartilagenous vault* (e.g., dorsal hump, increased mid-dorsal width) or if a previous rhinoplasty was carried an over reduced dorsum and a narrow width. The *lobule and tip* may be drooped and bulbous, with a lack of definition, or they may be overreduced, pinched, and turned up as a result of a previous surgery. The *nasal base* may be overly wide or constricted, primarily as a reflection of the underlying skeletal or cartilaginous structures.

Difficulty with breathing through the nose is often chronic, with a longstanding mouth-breathing habit. If this is the case, the facial morphology may be that of a long face growth pattern (i.e., vertical maxillary excess typically with an anterior open-bite Class II malocclusion; see Chapter 21). The *obstructive nasal breathing* is often made worse in the presence of chronic allergies or with ambient changes in humidity. Examination of the *internal nose* may reveal septal deviations or perforations, external valve stenosis, internal nasal valve obstructions, posterior nasal aperture stenosis, turbinate enlargement, or an irritated nasal mucosal lining. Consideration should also be given to the *adenoids and tonsils,* which may be enlarged and obstructive. The *internal nasal valves,* the *turbinated cavities,* the *external nasal valves,* and the *posterior nasal aperture* may be obstructed by any one of a number of anatomic structures (e.g., turbinates, septum, ULC, LLC, nasal skeletal walls or floor, soft tissues).

During the initial consultation, consideration is given to the following: 1) the patient's stated objectives 2) any patient-specific anatomic limitations 3) the surgeon's concepts of the patient's facial proportions (i.e., the presence of a dentofacial deformity or isolated microgenia) and 4) the patient's nasal aesthetic unit (i.e., whether to reduce, augment, or sculpt one or more of the key regions of the nose).

It is also essential for the surgeon to understand the patient's *psychological reasons* for requesting surgery on the nose and then to assess that patient's emotional maturity and stability. Constantian has outlined specific red flags for the surgeon to be concerned about, including the patient who is seeking surgery for specific external gain (e.g., an individual who expects that nasal changes will improve his or her career); the narcissistic male; and the perfectionist.[41] He also warns against operating on an individual whom "you just don't like" (i.e., with whom you are not able to effectively communicate). Picavet and colleagues documented a high prevalence of body dysmorphic disorder symptoms among patients who are seeking rhinoplasty (see Chapter 7).[176] Individuals who request revision rhinoplasty and those with a psychiatric history are particularly at risk for ongoing dissatisfaction.

When the surgeon is satisfied that the patient has a problem or concern that can be physically and visually improved and that the patient is realistic with regard to his or her expectations and without notable psychopathology, further details of the proposed surgery can be outlined and frankly discussed.

Definitive Rhinoplasty Planning

A review of the patient's history, physical findings, facial photographs, and, if indicated, sinus computed tomography scan or other facial radiographs (e.g., lateral cephalometric, Panorex) is generally followed by a second consultation visit to finalize the operative plan. The surgical objectives are clarified after 1) hearing the patient's requests 2) analyzing the overall facial and specific nasal aesthetics and functional aspects 3) considering patient-specific anatomic limitations 4) discussing all options and 5) considering the surgeon's ideal treatment plan.

Basic questions for the surgeon to consider include the following:

- Are there any medical or psychosocial contraindications to elective nasal surgery?
- What are the functional nasal findings? Do they correlate with the patient's symptoms?
- Are there associated maxillomandibular disproportions that affect the individual's facial aesthetics?
- What aspects of the patient's nose would the surgeon like to change?

- Given the quality of the patient's overlying nasal skin and the underlying skeletal and cartilaginous structures, what are the reasonable surgical options?
- Do the patient's requests match the surgeon's thoughts when taking into account any biologic limitations of the tissues?
- Does the surgeon think that he or she can interact effectively with the patient should a complication or suboptimal result occur?

By the end of the second consultation visit, a definitive operative plan should be clear in the surgeon's mind and accurately conveyed to the patient. The patient and the surgeon should be comfortable with any compromises that are agreed to and with the expected outcomes and the potential risks. If a comfort level is not reached, it is best not to proceed.

Most primary rhinoplasties require a combination of the reduction and augmentation of various aspects of the nose. This will often include the following: 1) the reduction of the bony and cartilaginous dorsum 2) the reduction of the cephalic portions of the LLCs (lateral crura) 3) middle crural suturing 4) cartilage (caudal strut) graft augmentation and 5) nasal osteotomies with in-fracturing (to narrow the width, to accomplish the closure of an open roof, or to straighten a crooked dorsum). When indicated, the removal of any septal deviations (i.e., cartilage and bone) and the reduction of the inferior turbinates will simultaneously diminish airway resistance and improve nasal breathing.

Open versus Closed Approach to the Nose

In the hands of a limited number of experienced nasal surgeons, the closed (endonasal) approach to gain exposure of the nose allows for the predictable management of the patient's needs.[91,198,202,217-221] In this author's clinical practice, the open (columella-splitting and bilateral marginal incisions) approach provides the preferred direct exposure of the nasal structures in most patients. After elevation of the nasal soft-tissue envelope via an open approach, direct inspection of the osseocartilaginous framework and tip should confirm the preoperative clinical diagnosis. This direct exposure facilitates technical maneuvers such as the following: 1) bony and cartilaginous reduction 2) excision and suturing of the LLCs 3) the placement and stabilization of grafts and 4) septal modification. By understanding normal nasal physiology and what the osseocartilaginous framework and nasal tip should look like and then seeing the anatomy under direct vision, the intended results are achieved more often than not.

As pointed out by many authors, the open approach has advantages that include the following[3,4,33,50,72-76,84,91,101,112]:

1. Securing a cartilage (strut) graft to the caudal septum to help control or maintain the tip position
2. Suturing the middle crura to each other and then over the top of the secured caudal (strut) graft to improve tip projection and shape
3. Controlling the columellar–lobular angle by altering the axis of the caudal strut graft
4. Suturing additional nasal tip grafts in place over the top of the LLCs
5. Accurately placing and securing spreader grafts to correct nasal valve collapse, to straighten the nose, and to correct saddle deformities
6. Placing and suturing cartilage grafts to reinforce, change the shape of, or replace missing segments of the lateral crura
7. Placing and securing onlay grafts (over the bony or cartilaginous vault) to reconstruct or augment the dorsum of the nose
8. Visualizing and then reducing the bony and cartilaginous nasal dorsum
9. Visualizing and then reducing or modifying the radix, including the placing and securing of grafts

An often-stated drawback of the open approach relates to tip swelling. The amount and duration of tip swelling after open rhinoplasty is generally greater than that seen with the closed approach. Therefore, it is reasonable to consider a closed rhinoplasty for those patients who may need only nasal osteotomies, dorsal hump reduction, or minimal tip alterations.

Complications Associated with Rhinoplasty

Fortunately, in the hands of an experienced nasal surgeon, the vast majority of well-informed patients undergo an uneventful operative and postoperative course and are pleased with their initial and long-term function and their aesthetic results. However, as with any surgical procedure, perioperative problems may occur (e.g., infection, bleeding, airway compromise) in addition to problems that are specific to rhinoplasty (e.g., septal perforation, persistent nasal obstruction, suboptimal cosmetic results).* These potential problems must be considered and openly discussed in advance to limit patient dissatisfaction.

Infection

Infection occurs in approximately 1% of patients who are undergoing elective rhinoplasty.[35,37,54,83,109,129,166] The routine use of prophylactic antibiotics that are started just before

*References 10, 13, 41, 52, 102, 118, 130, 133, 148, 168, 188, 238

surgery and continued postoperatively (at least until all internal nasal packs have been removed) is recommended. If infection occurs, appropriate drainage and culturing of the fluid is carried out. The antibiotic regiment is altered, as indicated.

Bleeding

Intraoperatively, bleeding is generally more of an annoyance than a problem. Minor postoperative nosebleeds are managed with elevation of the head, compression, packing, and time. When postoperative bleeding persists, a determination is made with regard to whether the hemorrhage is anterior or posterior, and appropriate packing of the nose is carried out. Cauterization of the bleeding points and rechecking of the hemoglobin level, the clotting studies, and the platelet function may be indicated. Ongoing hemorrhage that requires blood transfusion or the proximal ligation of branches of the external carotid artery is exceedingly rare. Bleeding is more likely to follow turbinate reduction than any other septorhinoplasty maneuver.

Airway Obstruction

The assessment of nasal breathing and of the entire upper airway should be carried out and the findings discussed with the patient and the family before rhinoplasty occurs.[16,18,20,43,94,111,150,151,194,214] The importance of doing this has been confirmed by Thomson[240] and Hellings,[104] who independently reported the presence of persistent nasal airway obstruction as a frequent complaint that leads to need for revision rhinoplasty. The nasal airway may also be compromised as a result of rhinoplasty. Synechiae may form between the septum, the lateral nasal wall, or the turbinates and require release in the office or, occasionally, in the operating room. Residual septum deflections may require removal. Further turbinate reduction may also be advantageous. The clinician should not ignore the nasal airway if a favorable result and a satisfied patient are desired.

Septal Perforation

Full-thickness holes (perforations) in the septum after a septoplasty are thought to occur in less than 5% of patients. The occurrence of septal perforations is dependent on the quality of the patient's nasal mucosal lining, the surgical technique, and the complexity of the deformity. Long-term problems associated with perforations range from annoyance (e.g., crusting, occasional bleeding, whistling as a result of turbulence on inspiration) to external deformities (e.g., saddle-nose depression as a result of septal collapse) with nasal obstruction; see the discussion later in this chapter. The repair of septal perforations is extremely difficult. Currently, a reliable routine method for the correction of septal perforations has yet to be realized.

Dry Nose Syndrome

Atrophic rhinitis is uncommon, but it may be seen after turbinate reduction.[59,149] There may be gradual and progressive atrophy of the nasal mucosa with a lack of moistening secretions that leads to crusting that accumulates. The crusts harbor bacteria; when this is associated with poor aeration and drainage, an unpleasant odor (ozena) may result.

Burn Injuries

The occurrence of operating room fires and airway burns is rare. Nevertheless, there is a risk of ignition in the operating room with any application of electrocautery or lasers. This risk is increased in the setting of nasal-cannula or face-tent supplemental oxygen. It can also be a problem in the intubated patient in whom there is an "air leak." Facial burns, intraoral and intra-airway burns, and even patient demise have all been reported. Although burn injuries in the operating room are rare, they represent approximately 20% of all malpractice claims for cases performed under monitored anesthesia care, and 95% of these cases involve surgery of the head and neck.[196]

Patient Dissatisfaction

Studies indicate that, on average, 15% of patients who have undergone cosmetic rhinoplasty procedure will express at least some dissatisfaction with the result.* In general, rhinoplasty patients differ from facial aging patients with regard to their psychological complexity. Most individuals who are seeking rhinoplasty have always been dissatisfied with some aspect of their facial appearance, which they attribute to nasal dysmorphology. Alternatively (at least in theory), the adult facial aging patient is seeking rejuvenation to restore a previous "satisfactory" appearance. The rhinoplasty patient is more likely motivated by lifelong body-image undercurrents that mold his or her perception of the nasal appearance. This will influence his or her expectations for surgery in a way that different from those of the facial aging patient.

Adamson and colleagues report that patients who request primary rhinoplasty commonly cite their rationale as including a "dorsal hump" (50%); a "too large" nose (44%); a bulbous tip (41%); and an "obstructed nasal airway" (33%).[32] Because half of the patients seeking primary rhinoplasty cited a "dorsal hump" as the chief concern, the achievement of an aesthetically pleasing nasal profile is essential. This is contingent on a thorough understanding of the underlying nasal anatomy and a complete facial analysis, which includes the assessment of the true dorsal hump as well as looking for the presence of a deep radix or an underprojected tip. Care should also be taken to establish a pleasing contour and transition between the dorsum and the tip (i.e., the supratip break) and to prevent iatrogenic deformities (i.e., supratip "Polly beak"

*References 41, 52, 67, 68, 102, 116, 176, 191, 245

deformity). Other surgical causes of patient dissatisfaction include cartilage graft warping, resorption, and palpability. Another frequent reason for a primary rhinoplasty consult is a nose that is "too large" (44%). The achievement of a good nasal profile will therefore frequently depend not only on the anatomy of various nasal parts but also on the interplay of the other facial parts (i.e., the maxilla, the mandible, and the chin region). The discerning clinician must separate out these aspects (i.e., dentofacial disharmony versus nasal disharmony) if patient satisfaction and a favorable outcome are to be achieved (see Chapters 19 through 23 and Fig. 38-2).

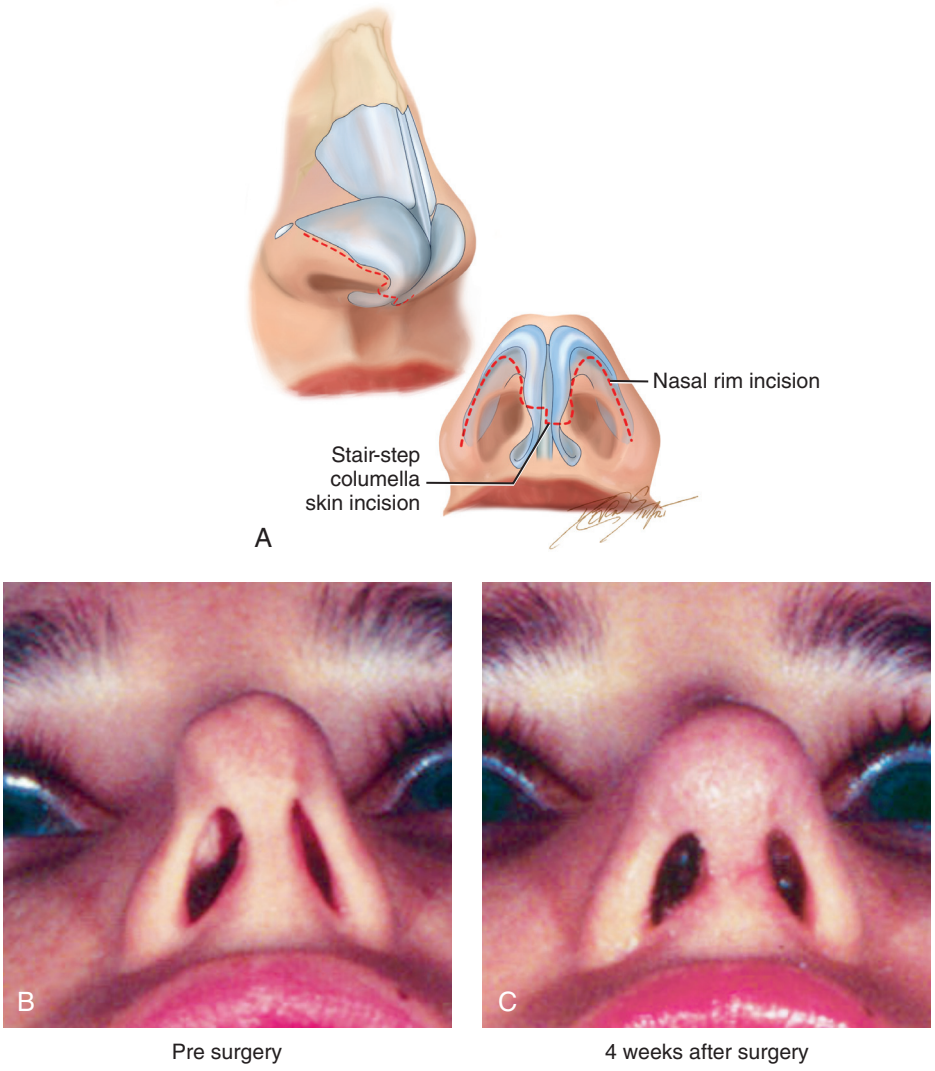
Rohrich and colleagues pointed out the importance of understanding the psyche of the male rhinoplasty patient.[200] These patients are more likely to present with nonspecific complaints, to be more demanding of the end result, and to be less attentive during the consultation visit. The clinician must painstakingly set clear and realistic expectations of the operation to achieve patient satisfaction. These authors recommend allowing 6 to 12 months of healing before agreeing to further nasal modifications. In most reported series, only 50% of patients who undergo revision rhinoplasty will move into the satisfied category.[41]

Primary Septorhinoplasty: Step-by-Step Approach (Figs. 38-5 through 38-9)

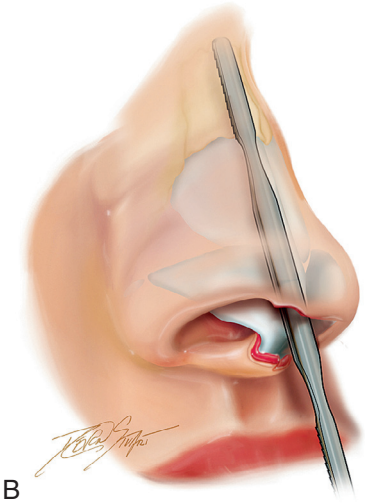
Preparation and Draping

- Complete the orotracheal (RAE) intubation, and tape the tube to the skin of the chin.
- Apply the ophthalmic ointment and place the corneal shields for eye protection.
- If the patient has long hair, tie it in a ponytail.

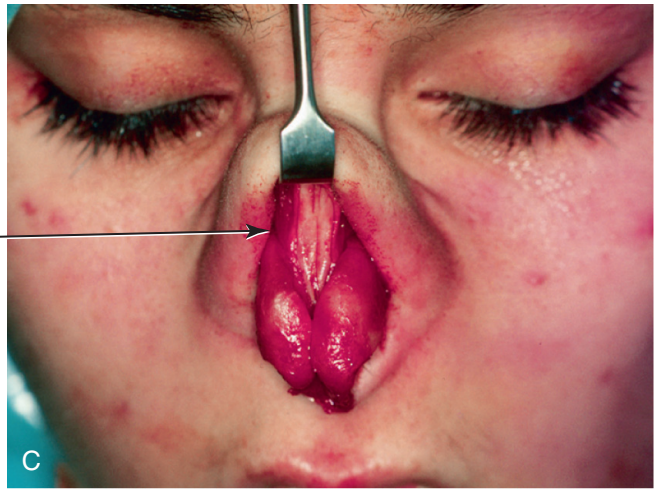
Text continued on p. 1632

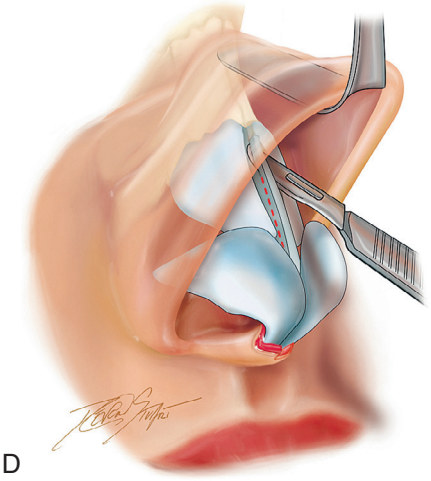


• **Figure 38-5** Incisions that are used for an open technique rhinoplasty are demonstrated. **A,** Stair-step columella (skin) and bilateral (intranasal) rim incisions are illustrated in two views. **B,** Worm's-eye views of the nose before and **C,** 4 weeks after an open-technique secondary rhinoplasty. Four weeks after surgery, the skin incision is immature (i.e., red and raised), and the soft tissues remain swollen.

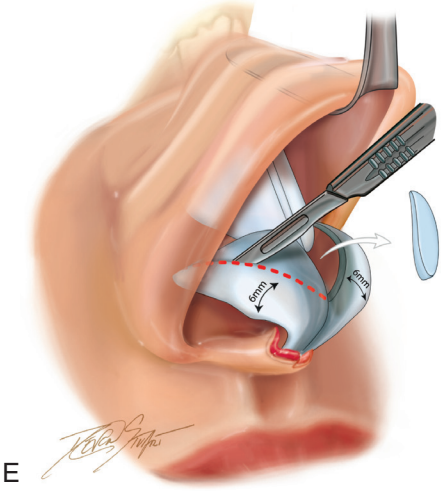


Removal of dorsal bony "hump" under direct vision

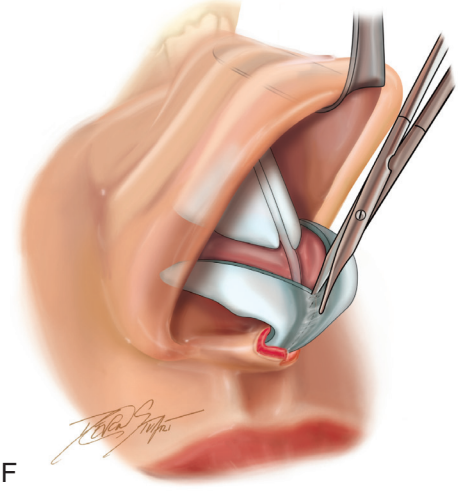




Removal of dorsal cartilage "hump" under direct vision



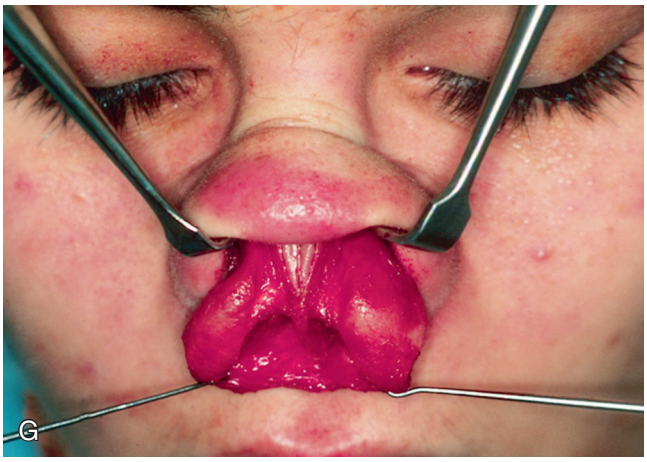
Knife used to reduce cephalic excess of lower lateral cartilages

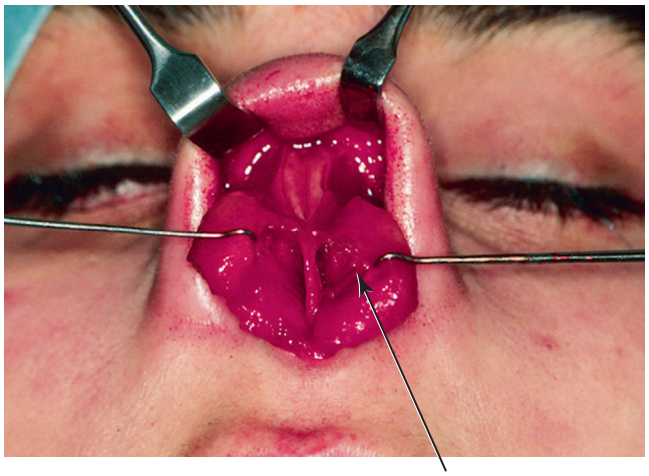


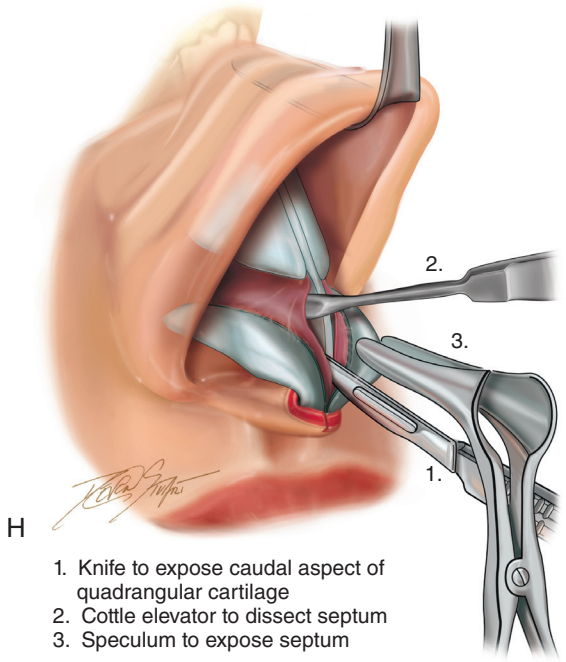
Scissors used to separate lower lateral cartilages and expose caudal aspect of septum

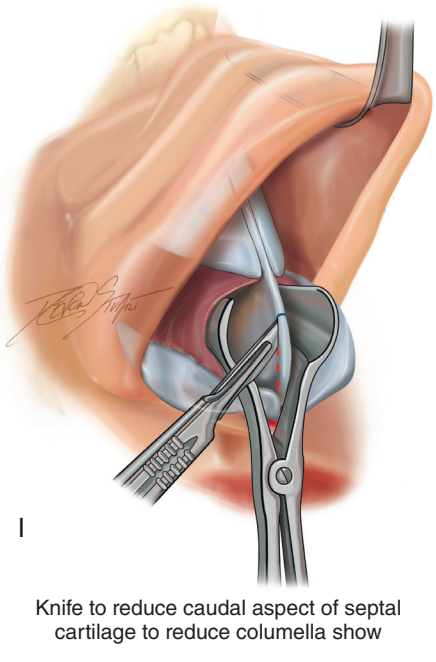
• **Figure 38-6 A,** Exposure of the deep structures, including the lower lateral cartilages (LLCs), as viewed through an open approach after the elevation of the soft-tissue envelope of the nose. **B,** A rasp is used to reduce the bony dorsum of the nose. **C,** The upper lateral cartilages and the dorsal aspect of the quadrangular cartilage are also directly visualized through the open approach. **D,** Dorsal bone reduction (as described in **B**) is followed by the sharp knife excision of the cartilaginous dorsal hump. The illustration shows the Aufricht retractor in place, with a knife (no. 15 blade) used to excise excess dorsal septal cartilage. **E,** Measurement of the LLCs is carried out with a caliper. In general, a 6-mm caudal rim of LLC is maintained. The cephalic excess is incised with a knife (no. 15 blade) and then removed with a Stevens scissors. **F,** An illustration that shows the scissors dissection to separate the LLCs and to expose the caudal aspect of the quadrangular cartilage.

Continued

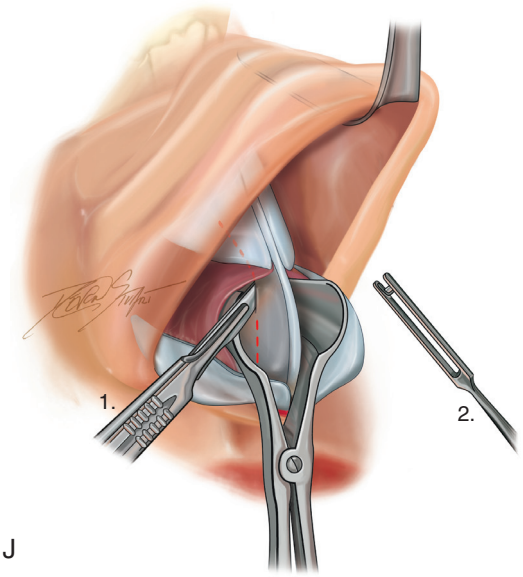






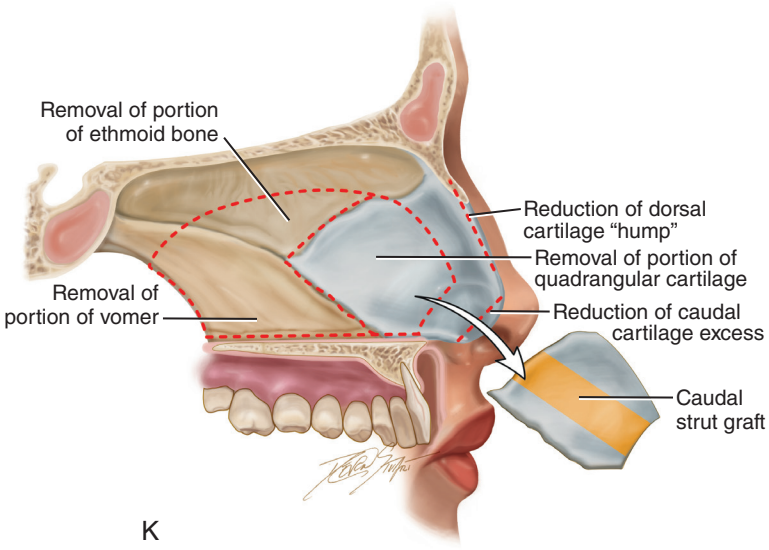


• **Figure 38-6, cont'd** **G,** Two intraoperative views show the LLCs separated to expose the caudal aspect of septum. **H,** An illustration that shows the following: 1) a knife (no. 15 blade) that is used to remove the final layer of perichondrium from the caudal edge of the septal cartilage before subperichondrial dissection; 2) a Cottle elevator that is used for exposure of the quadrangular cartilage and bony septum (i.e., vomer and ethmoid); and 3) a speculum that is inserted with a blade placed on each side of the septum. **I,** If there is excess columella show, then the caudal aspect of the quadrangular cartilage is reduced (excised) to improve aesthetics. The illustration demonstrates the planned caudal reduction.

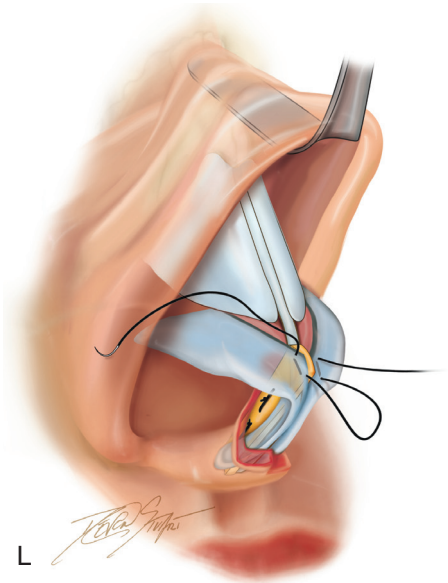


J

1. Knife to initiate incision of septal cartilage
2. Swivel knife to complete the excision of septal cartilage



K

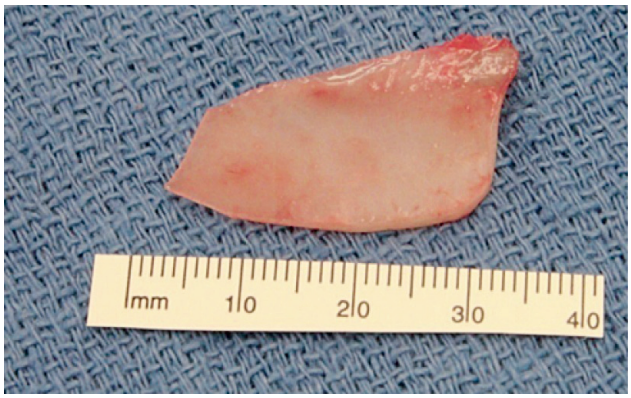


L

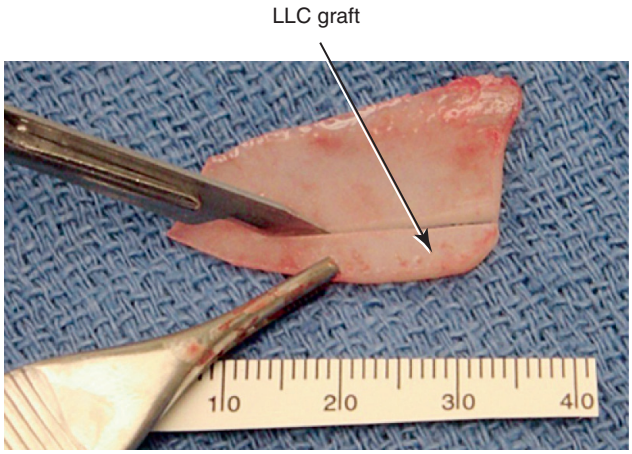
Caudal strut graft sutured in place, lower lateral cartilages are then sutured together and over the top of the graft

• **Figure 38-6, cont'd J,** An illustration showing an incision through the quadrangular cartilage to initiate submucous resection. It is important to maintain an uninjured and adequate dorsal and caudal strut of cartilage to structurally support the nose. *(1)* A knife (no. 15 blade) is used to initiate the incision in the septal cartilage. *(2)* A swivel knife will then be introduced to complete the actual submucous resection of the septal cartilage. The cartilage excision corresponds with needed graft for augmentation or with the removal of septum that is interfering with breathing. **K,** An illustration of the extent of maximum septum (bone and cartilage) that may be removed to improve the airway. This includes much of the quadrangular cartilage and bone (i.e., vomer and perpendicular plate of ethmoid). The removed quadrangular cartilage available graft is shown. The yellow-colored aspect of the removed quadrangular cartilage indicates the planned caudal strut graft. **L,** An illustration of the crafted cartilage (strut) graft sutured to the caudal aspect of the remaining septal cartilage. The LLCs are then sutured together and over the top of the strut graft. This type of suture has been called a *medial crural fixation suture*. The sutures are placed to fix one medial crus to the other, thereby unifying the medial crura beneath the domes and then above the strut graft. This combination of techniques allows for the preferred nasal tip projection, shape, and orientation.

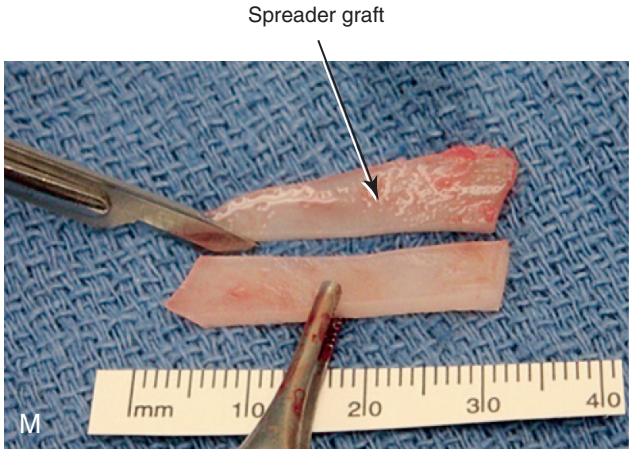
Continued



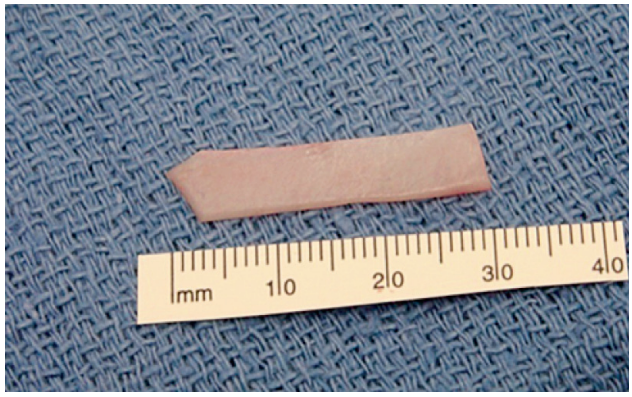
Removed septal cartilage



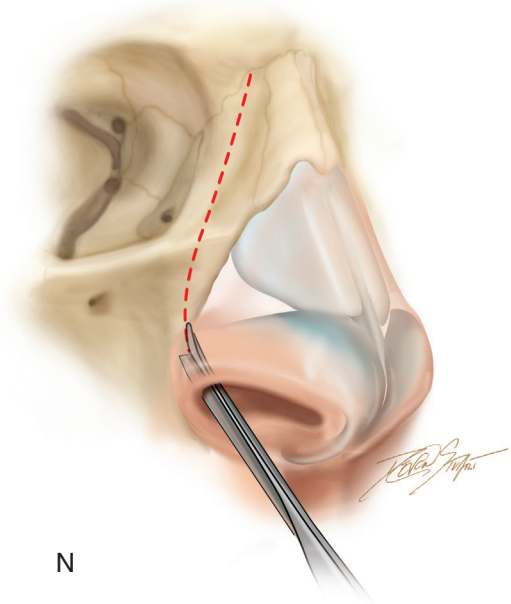
Inferior excision



Superior excision

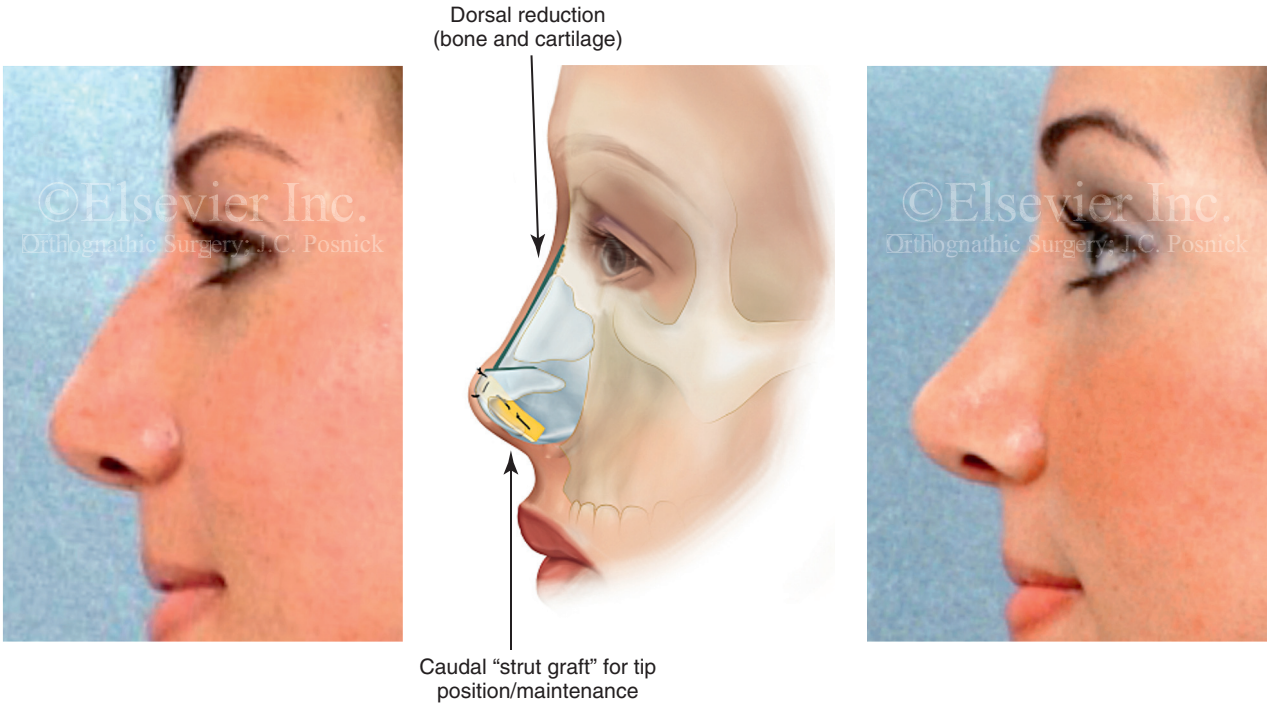


Caudal “strut” graft



Guarded ostetomy used to complete nasal
osteotomy through intranasal pyriform rim incision

• **Figure 38-6, cont’d M,** Intraoperative views of removed septal cartilage, which can be used as graft material to reconstruct the nose. The cartilage can be crafted as a caudal strut, spreader grafts, LLC augmentation grafts, or tip onlay grafts. **N,** Nasal osteotomies are completed by first identifying the piriform rim intranasally with a speculum; the speculum blades straddle the bony rim. An incision is completed through the nasal mucosa on the side of the anterior maxilla to expose the piriform rim. The speculum is removed, and a Joseph elevator is inserted. Subperiosteal tunneling in the location of the proposed osteotomy is carried out. A curved and guarded osteotome is introduced through the intranasal incision, and the osteotomy is completed. The osteotomy is performed through the pyriform rim (maxilla), lateral to the nasomaxillary suture, and then up toward the nasofrontal suture on each side. The nasal bones are then in-fractured with finger pressure and positioned as desired.



• **Figure 38-7** Profile views of the nose highlighting a young woman before and after rhinoplasty with the use of the techniques described in the legend for Figs. 38-5 and 38-6. An illustration highlights the combination of dorsal reduction (bone and cartilage) and nasal tip alteration (i.e., caudal strut cartilage grafting and lower lateral cartilage reshaping) typically carried out to achieve the objectives.

• **Figure 38-8** A woman in her 20s underwent a septorhinoplasty procedure as described previously. She is shown at 1 week and 4 weeks after surgery. At her 1-week visit, the custom-formed Aquaplast splint and skin sutures were removed.

- Use Betadine solution (do not use soap, alcohol, Hibiclens and so on) to prepare the scalp, forehead, external ears, face, and neck.
- Complete draping and leave the forehead, external ears, face, and neck exposed in the operative field.

Surgical Marking

- Locate and use a surgical pen to mark the columella (stair-step) incision and the bilateral rim (marginal) intranasal incisions before prepping and draping.

Local Anesthesia

- Inject Xylocaine (1% with 1:100,000 epinephrine) within the soft-tissue envelope of nose as usual for rhinoplasty.
- Inject Xylocaine (1% with 1:100,000 epinephrine) on each side of the septum, deep to the mucosa and superficial to the cartilage.
- Place either cocaine-soaked (5 mL of 4% solution) or Afrin-soaked pledgets in each nostril cavity.

Skin Incisions and Flap Elevation

- Incise the columella skin and the intranasal rims with a knife (#15 blade).
- Elevate the nasal soft-tissue envelope with Stevens scissors as required for an open rhinoplasty procedure.
- Elevate the overlying cutaneous soft-tissue envelope up the columella, superficial to the LLC and ULC, subperiosteal over the nasal bones (with a Joseph elevator) to the radix, and lateral toward the maxillary process on each side.
- Clean the subcutaneous tissue off of the superficial surface of each LLC with the use of Stevens scissors.

Dorsal Reduction

- If a dorsal bony hump requires reduction, it is conservatively completed with coarse and fine diamond rasps.
- If a dorsal cartilaginous hump requires reduction, it is conservatively incised with a knife (no. 15 blade) under direct visualization.

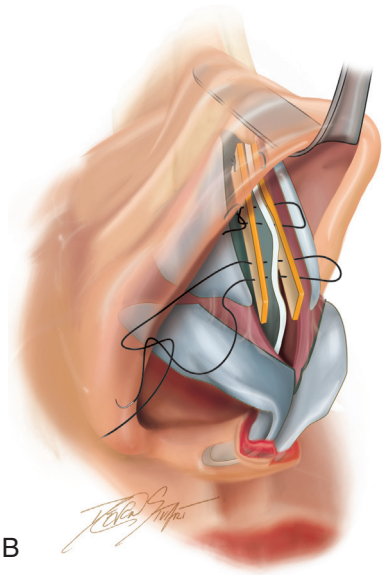
Lower Lateral Cartilage Modification

- The LLCs are separated from their midline soft-tissue connections with the use of Stevens scissors. Dissection continues down to the caudal aspect of the septum.
- The LLCs are marked with a caliper and trimmed of cephalic excess. In general, at least 6 mm of caudal LLC is retained. The redundant cephalic portions are incised with a knife (no. 15 blade) and removed with Stevens scissors.

Exposure and Resection of Portions of Septum

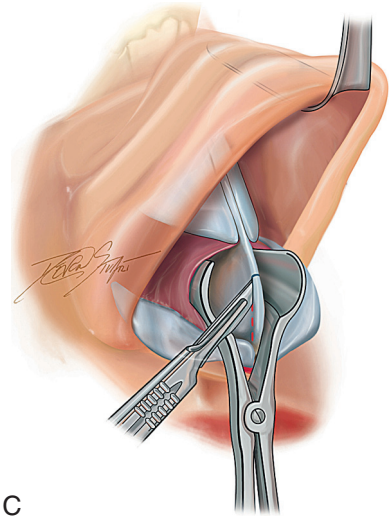
- Identify the anterior aspect of the caudal septal cartilage. (Dissection to the septum has already been completed with Stevens scissors.) A knife (no. 15 blade) is used to remove the final layer of perichondrium from the caudal

• **Figure 38-9** Three frequent maneuvers that are used to alter the cartilaginous vault and nasal tip are highlighted. For each of these three techniques, an illustration and a patient example are shown. Other rhinoplasty maneuvers were also simultaneously carried out on each patient. **A,** A frequent surgical technique to improve the position of the nasal tip is the harvesting of the septal cartilage, which is then crafted as a caudal strut. The caudal strut is sutured to the native septum and vectored to improve tip position and for maintenance. The lower lateral cartilages are then sutured together and over the top of the graft.

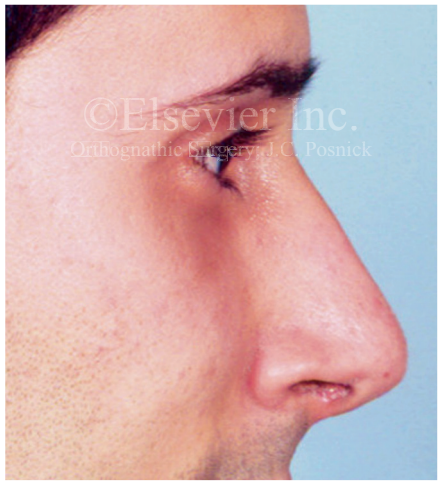


B

Spreader grafts from septal cartilage are sutured in place to straighten a "crooked" cartilagenous vault



C



• **Figure 38-9, cont'd B,** Spreader grafts may be crafted from harvested septal cartilage. They are placed on each side of the dorsal aspect of the quadrangular cartilage and sutured together. Spreader grafts can be helpful to open the internal nasal valves and to straighten a crooked cartilaginous vault. **C,** Altering the caudal aspect of the quadrangular cartilage to achieve improved aesthetics in the columella region is often a necessary aspect of rhinoplasty. When there is excessive "columella show" seen in profile, the excision of the redundant caudal septal cartilage will be helpful. A patient example and an illustration are shown. In other circumstances, when there is inadequate "columella show" (i.e., retracted columella), augmenting the caudal aspect of the septum with a cartilage graft will be a useful approach (see Chapter 29).

edge of the septal cartilage before dissection. Next, use a sharp Cottle elevator to achieve submucosal exposure on each side of the septal cartilage. Extend the submucosal dissection inferiorly to the maxillary ridge and posterior to the vomer. Continue the subperiosteal dissection as required for the exposure and resection of the deviated bony septum (i.e., to the vomer and the perpendicular plate of the ethmoid).

- If the caudal aspect of the quadrangular cartilage requires reduction (i.e., to limit columella show), it is sharply incised with a knife (no. 15 blade), and the redundant portion is removed under direct visualization.
- Submucosal resection of the cartilaginous septum is initiated with the use of a knife (no. 15 blade) to control the caudal and dorsal aspects of the excision. The knife incisions are located to maintain approximately 10 mm of caudal and 10 mm of dorsal septal cartilage support for the nose. This is followed by the insertion of a swivel knife.
- The swivel knife is used to excise the deviated, buckled, or thickened septal cartilage (i.e., submucosal resection) and to harvest a graft, as indicated.
- A rongeur (either a double action or a Takahashi) is used to remove any buckled, deviated, or thickened portions

of the vomer and the perpendicular plate of the ethmoid that are obstructing nasal airflow (i.e., submucosal resection).

Reduction of Inferior Turbinates

- If the inferior turbinates are hypertrophic and require partial reduction, this is accomplished by one of several methods, including scissors resection of the inferior aspect of each turbinate; out-fracture; submucosal (intramural) removal; or submucosal cauterization.

Completion of Nasal Osteotomies

- A short nasal speculum is inserted intranasally with one forceps blade placed on either side of the pyriform rim.
- An incision is made with a knife with a no. 15 blade piercing the nasal mucosa down to the bone along the piriform rim of the maxilla.
- A Joseph elevator is used to dissect (tunnel) subperiosteally up toward the nasofrontal suture where the osteotomy is to be completed.
- A curved, guarded osteotome is inserted through the intranasal incision that saddles the pyriform rim. A mallet is used to drive the osteotome and to complete the osteotomy.
- The same procedure is completed on the other side.
- The nasal bones are in-fractured, disimpacted for adequate mobility, and then reoriented in accordance with aesthetic preference.

Reconstruction of Nasal Tip

- Craft the required grafts (e.g., caudal strut, LLC grafts, spreader grafts, tip graft) with a knife (no. 15 blade) from the full piece of harvested septal cartilage
- If a caudal strut graft is required, it is crafted and then positioned near (abutted to) the caudal aspect of septal cartilage to achieve improved nasal tip position. It is held in the preferred location with pinching forceps or a needle and then sutured to the side of the septal cartilage with the use of interrupted stitches (5-0 Vicryl).
- The LLCs, having been trimmed of cephalic excess, are sutured together and then over the caudal strut graft to form the most superficial aspect of the nasal tip (5-0 Vicryl). This type of suture has been called a *medial crural fixation suture*; it is used to fix one medial crus to the other, thereby unifying the medial crura beneath the domes and then above the caudal strut graft.
- The soft-tissue envelope is redraped and the nose is inspected to confirm adequate dorsal reduction, proper tip projection and position, satisfactory columella show, and straightness of the nose. Further refinements are made, if needed. (They usually are!)

Wound Closure

- After the completion of the septorhinoplasty procedure, closure of the wound is accomplished.
- To approximate the columella skin (stair-step incision), a single buried dermal suture is placed (5-0 Vicryl).
- The columella skin closure is with interrupted suture ties (6-0 nylon).
- The intranasal rim incisions are closed with interrupted ties (5-0 chromic).

Placement of Dressing

- Routine dressing is placed as they are after a septorhinoplasty procedure.
- Flexible splints (Reuter) are generally placed (one on each side of the septum) and secured together with single suture tie (3-0 nylon).
- Antibiotic ointment (Bactroban) is generally injected into each nostril cavity.
- When nasal osteotomies are carried out, nasal packing is helpful to maintain the preferred nasal bone position. Packing (folded Telfa) is generally inserted equally into each nostril cavity.
- Tape is placed over the dorsum of the nose (Steri-Strips) as it is after rhinoplasty.
- A custom splint (Aquaplast) is molded over the dorsum of the nose as it is after rhinoplasty.

Images of a series of individuals are shown to demonstrate the spectrum of presentations and the typical results achieved with the use of the techniques just described (Figs. 38-10 through 38-18).

Cleft Nasal Deformities

Background

Through the dissection of a stillborn fetus with unilateral cleft lip and palate (UCLP), McComb documented that tissue distortions and malpositions are primary factors in the cleft nasal deformity.[37,136] Byrd and colleagues also completed the dissection of a stillborn fetus with UCLP and confirmed hypoplasia of the LLC complex in addition to malpositions.[25] They also found an abnormal fixation of the lateral crus to the periosteum of the pyriform rim.

The primary (unrepaired) cleft nasal malformation in an individual with UCLP is characterized by features that include the following: 1) malposition and hypoplasia of the LLC with fibrous attachment of the lateral crus to the pyriform rim 2) interruption of the muscle ring (orbicularis oris) across the clefted nasal sill with abnormal muscle insertions at the alar base to the cheek and to the lip and 3) soft-tissue clefting through the nasal floor (Figs. 38-19 and 38-20). In the unilateral complete cleft lip nasal

Text continued on p. 1645

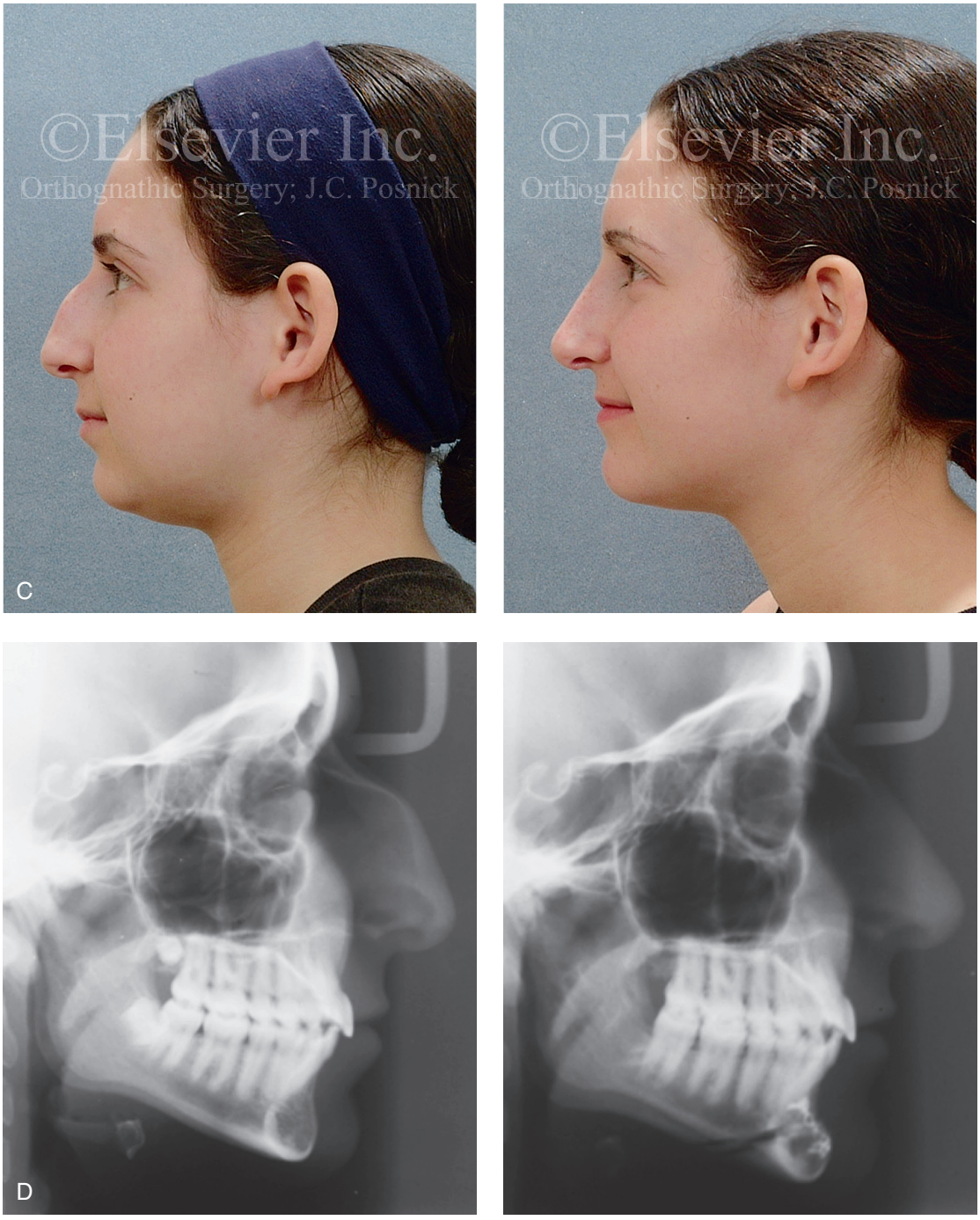
• **Figure 38-10** A teenage girl arrived with her parents and requested a rhinoplasty to remove the "hump" that was seen in profile. She also has a lifelong history of difficulty breathing through the nose. She underwent an open rhinoplasty that included nasal osteotomies (infracture); dorsal reduction (bone and cartilage); nasal tip maneuvers (lower lateral cartilage modification and septal cartilage caudal strut grafting); septoplasty; and inferior turbinate reduction. **A,** Frontal views before and after reconstruction. **B,** Oblique facial views before and after reconstruction. **C,** Profile views before and after reconstruction.

• **Figure 38-11** A teenage girl arrived with her parents and requested a reduction rhinoplasty. She also had difficulty breathing through the nose. She underwent an open rhinoplasty that included nasal osteotomies (in-fracture); dorsal reduction (bone and cartilage); nasal tip refinement (reshaping of the lower lateral cartilages and septal cartilage caudal strut grafting); septoplasty; and inferior turbinate reduction. **A,** Facial views before and after reconstruction. **B,** Oblique facial views before and after reconstruction. **C,** Profile views before and after reconstruction.

©Elsevier Inc.
Orthognathic Surgery; J.C. Posnick

• **Figure 38-12** A teenage girl arrived with her parents and requested cosmetic rhinoplasty and the improvement of her nasal breathing. She underwent an open rhinoplasty that included nasal osteotomies (in-fracture); dorsal reduction (bone and cartilage); tip refinement (reshaping of the lower lateral cartilages and septal cartilage caudal strut grafting); septoplasty; and inferior turbinate reduction. **A,** Frontal views before and after reconstruction. **B,** Oblique facial views before and after reconstruction. **C,** Profile views before and after reconstruction.

• **Figure 38-13** A teenage girl arrived with her parents and requested a "smaller and more attractive" nose. Rhinoplasty to reduce the dorsum and elevate the tip in combination with genioplasty to improve the profile was recommended. She underwent osseous genioplasty (vertical lengthening and horizontal advancement) and open rhinoplasty that included nasal osteotomies (in-fracture); dorsal reduction (bone and cartilage); tip refinement (reshaping of the lower lateral cartilages and septal cartilage caudal strut grafting); septoplasty; and inferior turbinate reduction. **A,** Frontal views before and after reconstruction. **B,** Oblique facial views before and after reconstruction.



• **Figure 38-13, cont'd C,** Profile views before and after reconstruction. **D,** Lateral cephalometric radiographs before and after reconstruction. Note the interpositional bloc hydroxyapatite graft that was used to achieve successful chin lengthening.

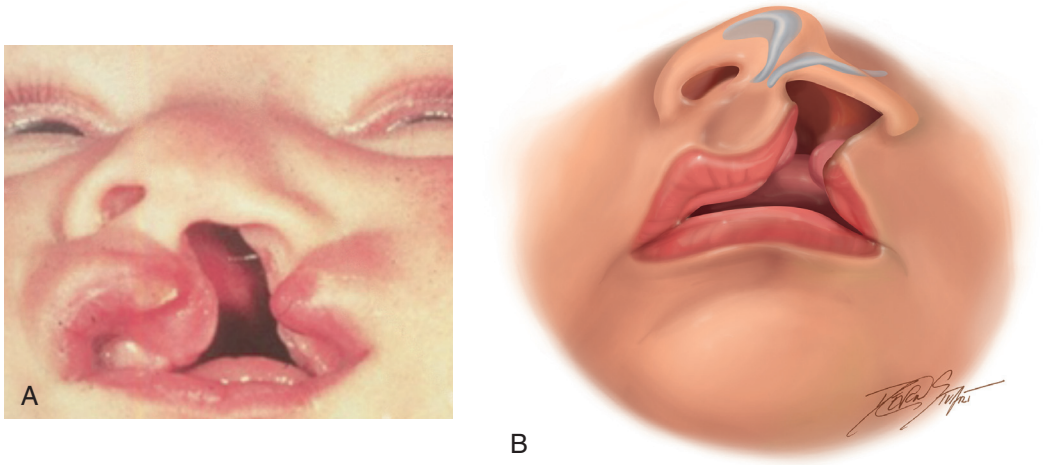
• **Figure 38-14** A teenage girl arrived with her parents and requested cosmetic rhinoplasty to narrow the mid dorsum, to remove the dorsal hump, and to improve tip projection. Her history also confirmed significant nasal obstruction. She underwent an open rhinoplasty that included nasal osteotomies (in-fracture); dorsal reduction (bone and cartilage); tip refinement (reshaping of the lower lateral cartilages and septal cartilage caudal strut grafting); septoplasty; and inferior turbinate reduction. Note that the thick quality of the nasal skin limits the aesthetic result. **A,** Frontal views before and after reconstruction. **B,** Oblique facial views before and after reconstruction. **C,** Profile views before and after reconstruction.

• **Figure 38-15** A woman in her mid 20s requested cosmetic rhinoplasty and the improvement of her nasal breathing. She also has a mild long face growth pattern and prominent ears. She requested that surgery be limited to the nose. The nasal objectives included the removal of the dorsal hump, the narrowing of the mid dorsum, and the improved positioning of the tip. She underwent an open rhinoplasty that included nasal osteotomies (in-fracture); dorsal reduction (bone and cartilage); tip refinement (reshaping of the lower lateral cartilages and septal cartilage caudal strut grafting); septoplasty; and inferior turbinate reduction. **A,** Frontal views before and after reconstruction. **B,** Oblique facial views before and after reconstruction. **C,** Profile views before and after reconstruction.

• **Figure 38-16** A woman in her early 50s requested cosmetic rhinoplasty and the improvement of her nasal breathing. She hoped for improved tip projection and the removal of the dorsal hump. She underwent an open rhinoplasty that included nasal osteotomies (in-fracture); dorsal reduction (bone and cartilage); tip refinement (reshaping of the lower lateral cartilages and septal cartilage caudal strut grafting); septoplasty; and inferior turbinate reduction. **A,** Oblique facial views before and after reconstruction. **B,** Profile views before and after reconstruction.

• **Figure 38-17** A woman in her late 40s requested cosmetic rhinoplasty to improve tip projection and to reduce the dorsal hump. She also requested relief of the hooding of the upper eyelids. She underwent an open rhinoplasty that included nasal osteotomies (in-fracture); dorsal reduction (bone and cartilage); and tip refinement (reshaping of the lower lateral cartilages and septal cartilage caudal strut grafting). She also underwent upper blepharoplasty (removal of excess skin). **A,** Oblique facial views before and after reconstruction. **B,** Frontal views before and after reconstruction.

• **Figure 38-18** A 28-year-old man of Arabic descent had previously sustained a fracture of the nasal bones. As a result, he had difficulty breathing and was displeased with the aesthetics (i.e., dorsal hump, crooked nose, drooped nasal tip). He underwent an open rhinoplasty that included nasal osteotomies (in-fracture); dorsal reduction (bone and cartilage); nasal tip maneuvers (reshaping of the lower lateral cartilages and septal cartilage caudal strut grafting); spreader grafts (septal cartilage); septoplasty; and inferior turbinate reduction. **A,** Frontal views before and after reconstruction. **B,** Oblique facial views before and after reconstruction. **C,** Profile views before and after reconstruction.



• **Figure 38-19** Demonstration of a typical unilateral cleft lip nasal malformation. **A,** Worm's-eye view of a newborn with unilateral cleft lip and palate (UCLP). **B,** Illustration showing the typical dysmorphology of the maxillary base, the caudal septum, and the nasal tip in a newborn with unrepaired unilateral cleft lip and palate.

malformation, asymmetric anatomic distortions (e.g., displacement of the septum to the non-cleft side) also require special consideration.[225,252] In the bilateral complete cleft lip nasal malformation, there is minimal asymmetry but shortening of the height of the columella is to be expected (see Figs. 38-31 and 38-32).

Nasal Deformities in the Newborn with Unilateral Cleft Lip and Palate (see Figure. 38-19)

- The perpendicular plate of the ethmoid deviates toward the cleft side.
- The posterior aspect of the quadrangular cartilage deviates toward the cleft side.
- The anterior aspect of the quadrangular cartilage deviates toward the non-cleft side.
- The anterior nasal spine deviates toward the non-cleft side.
- The bony pyramid of the nose (nasal bones) deviates toward the non-cleft side.
- The nasal bones are asymmetric and generally flattened on the cleft side.
- The ULCs are asymmetric and have a disturbed junction with the LLCs on the cleft side.
- There is a tendency toward the bifidity of the nasal tip.
- There is a buckling of the lateral crus of the LLC on the cleft side.
- There is a downward position of the medial crus of the LLC on the cleft side.
- There is a deviation of the nasal tip to the cleft side.
- There is a deviation at the nasal base to the non-cleft side.
- There is a lateral, downward, and posterior displacement of the alar base on the cleft side.

Basic Nasal Reconstructive Maneuvers during the Primary Unilateral Lip Repair (Figures. 38-20 and 38-21)

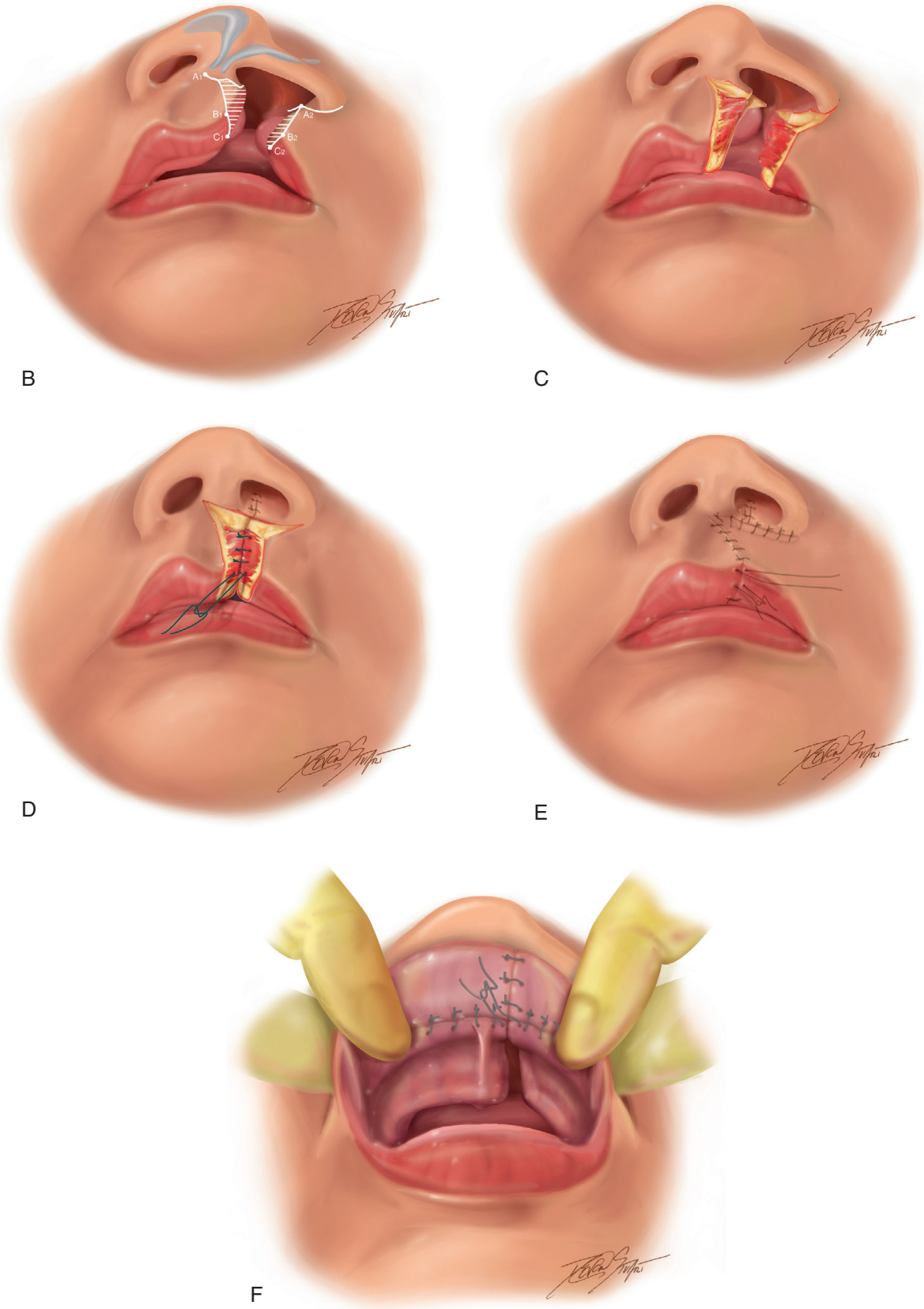
- Along the lateral aspect of the nose, the nasal soft tissues (including the inferior turbinate) are released from the piriform rim and the lateral nasal wall with the use of an elevator (Fig. 38-21, *A*).
- Next, the buckled and flattened lateral crus of the LLC is released from the overlying nasal soft tissues with the use of Stevens scissors (Fig. 38-21, *B*).
- The elevated lateral nasal flap (including the inferior turbinate) will be advanced medially, superiorly, and anteriorly to facilitate nasal sill and floor reconstruction (Fig. 38-20, *D*).
- Along the medial aspect of the nose, the nasal soft tissues (including the medial crus of the LLC) is released from the anterior nasal spine and septal cartilage with the use of an elevator and Stevens scissors (Fig. 38-21, *C*). The elevated medial nasal flap will be advanced laterally to facilitate nasal sill and floor reconstruction (Fig. 38-20, *D*).
- The release of the orbicularis oris muscle from its abnormal soft-tissue and bone attachments is accomplished with the use of Stevens scissors. Reconstruction of the orbicularis oris muscle with interrupted sutures achieves continuity across the lip (Fig. 38-20, *D*).
- Recurrent buckling of the lateral crus of the LLC early after surgery is limited by the placement of an intranasal stint (i.e., packing). Sutures through the nasal soft tissues and LLC may also be used to elevate and maintain the shape of the rim (Fig. 38-21, *D*).

Three typical patients with UCLP that have been treated with the techniques described are shown after primary repair (Figs. 38-22 through 38-24). Maneuvers that are

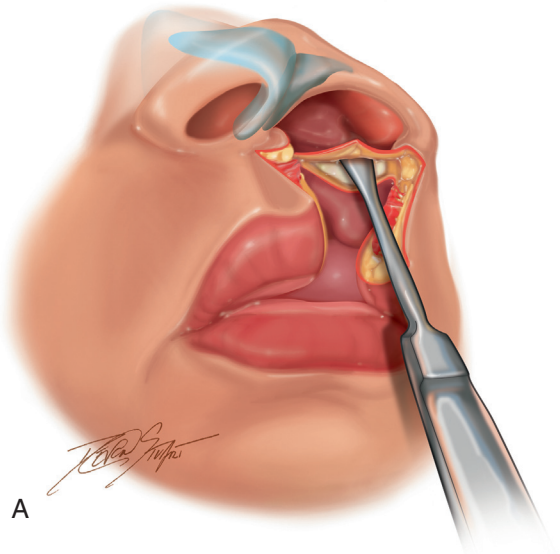
Text continued on p. 1650

A

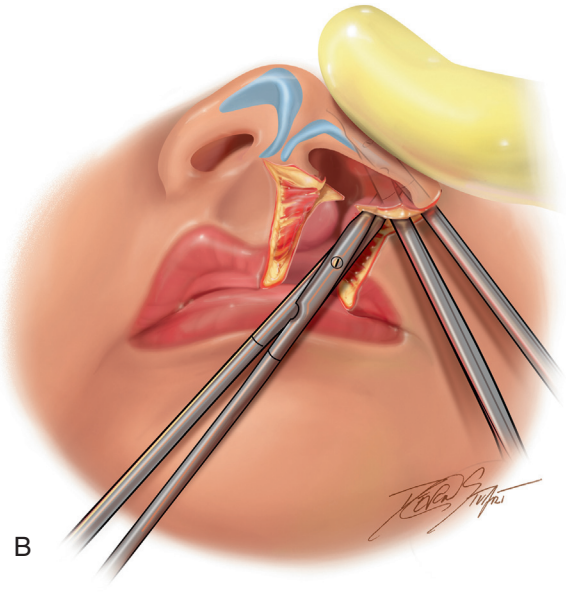
• **Figure 38-20 A,** Illustration of the typical facial malformations and distortions in a newborn with unilateral cleft lip and palate.



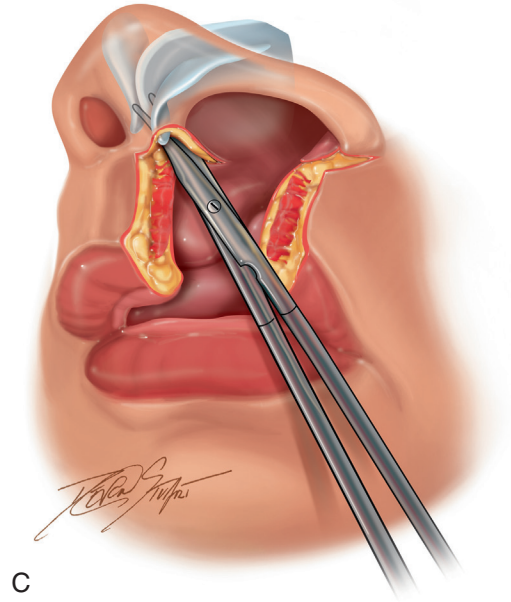
• **Figure 38-20, cont'd B,** Illustration of the modified "Millard" rotation and advanced cutaneous flaps used by this author for primary lip reconstruction and the basic nasal maneuvers that are simultaneously carried out. Incision locations for the lip rotation flap (greater segment) and the lip advancement flap (lesser segment) are outlined. Incision locations for the medial and lateral nasal flaps are also shown. **C,** The incisions are made and the flaps are elevated. Redundant tissue is excised, and the dissection of each lip flap from the underlying bone and cartilage is completed. The three tissue layers (i.e., the skin, the orbicularis oris muscle, and the underlying oral mucosa) are further separated from each other. **D,** The orbicularis oris muscle, having been released from its abnormal bony and soft-tissue attachments, is sutured across the midline to the base of the columella. Before this is done, the medial and lateral nasal flaps will have been elevated (see Fig. 38-21). After the orbicularis oris muscle has been repaired with sutures, the anterior aspect of the nasal floor is reconstructed with the use of the medial and lateral nasal flaps. **E,** The cutaneous lip rotation and advancement flaps are then approximated without tension over the repaired muscle for wound closure. The corner of the advancement lip flap fills the void at the base of the columella and is secured with suture. Precise approximation of anatomic landmarks with fine sutures is then undertaken to align the vermilion–mucosa junction and the vermilion–cutaneous junction. The philtral column and the nasal sill are also sutured. **F,** The underlying oral mucosa is reconstructed and sutured to establish a functional vestibule. Note: Alveolar clefting and oronasal fistula remains.



Lateral nasal flap elevation prior to lip repair



Dissection of LLC from overlying skin prior to lip repair



Nasal stent in place (UCLP)

• **Figure 38-21** Illustrations of unilateral cleft nasal maneuvers that are carried out as part of the primary lip and nasal reconstruction. **A,** Along the lateral aspect of the nose, the nasal soft tissues (including the inferior turbinate) are released from the pyriform rim and the lateral nasal wall with the use of an elevator that is inserted through the open lip. **B,** Next, the buckled and flattened lateral crus of the lower lateral cartilage is released from the overlying nasal soft tissues with the use of Stevens scissors inserted through the open lip. The elevated lateral nasal flap (including the inferior turbinate) will be advanced medially, superiorly, and anteriorly to facilitate nasal floor reconstruction (see Fig. 38-20, *D*). **C,** Along the medial aspect of the nose, the nasal soft tissues (including the medial crus of the lower lateral cartilage) are released from the anterior nasal spine and septal cartilage with the use of an elevator and Stevens scissors. The elevated medial nasal flap will be advanced laterally to facilitate nasal floor reconstruction (see Fig. 38-20, *D*). **D,** Recurrent buckling of the lateral crus early after surgery is limited with the use of an intranasal stint (packing). Sutures through the nasal soft tissues are also placed to pexy the lower lateral cartilage.

• **Figure 38-22** A newborn with a complete unilateral cleft lip and palate on the left side is shown before and 4 years after primary repair. He did not undergo any taping, molding, pinning, or adhesion procedures before the lip repair. When he was 3 months old, he underwent primary cleft lip repair in combination with primary nasal reconstruction as described (see Figures 38-20 and 38-21). When he was 12 months old, he underwent cleft palate repair with the use of the Bardach two-flap technique. He is shown **A,** as a newborn; **B,** at 18 months of age (6 months after palate repair); and **C,** at 3 years of age.

• **Figure 38-23** A child was born with incomplete clefting of the left primary palate; the secondary palate is also clefted. There is marked nasal deformity and clefting through the alveolus. She did not undergo any taping, molding, pinning, or adhesion procedures before the lip repair. At the time of primary cleft lip repair, she underwent nasal reconstructive maneuvers as described previously in the legends for Figs. 38-20 and 38-21. This was followed by palate repair when she was 12 months old with the Bardach two-flap technique. She is shown **A,** as a newborn; **B,** at 3 months of age (1 week after primary repair); **C,** at 1 year of age just before palate repair; **D,** at 4 years of age; and **E,** at 8 years of age after alveolar bone grafting.

• **Figure 38-24** A newborn with a complete UCLP on the right side is shown before and 7 years after primary repair. She did not undergo any taping, molding, pinning, or adhesion procedures before the lip repair. At 3 months of age, she underwent primary cleft lip repair in combination with primary cleft nasal reconstruction as described previously in the legends for Figs. 38-20 and 38-21. When she was 12 months old, she underwent cleft palate repair with the Bardach two-flap technique. She is shown **A,** as a newborn; **B,** at 3 months of age (2 weeks after primary lip and nose repair); and **C,** at 7 years of age just before alveolar bone grafting.

performed by some clinicians to alter the nasomaxillary anatomy before primary reconstruction (i.e., taping, nasoalveolar molding, Latham pinning, lip adhesion) were not carried out in any of these patients.[2,78,117,229,244] Interestingly, the American Cleft Palate–Craniofacial Association parameters of care document that addresses primary cleft lip and nasal repair includes only the following statements[172]:

- Surgical repair of the cleft lip is usually initiated within the first 12 months of life and may be performed as early as is considered safe for the infant.
- Presurgical maxillary orthopedics to improve the position of the maxillary alveolar segments before surgical closure of the lip may be indicated for some infants.
- The nasal deformity is an integral part of the cleft lip [malformation]. Depending on the severity, primary nasoplasty may be performed at the time of primary lip repair.
- A preliminary lip adhesion is a procedure that may be used in selected patients before definitive lip repair.
- The goal of lip repair is to restore the normal functional and anatomic features.

Interestingly, a recent survey of North American cleft surgeons indicated that only 50% of clinicians carry out any form of primary nasal reconstruction.[12,236] Ongoing evidence-based clinical research will be required to further clarify standards of care that address primary cleft lip and nasal repair.

Residual Nasal Deformities in the Adolescent or Adult with Repaired Unilateral Cleft Lip and Palate

Residual nasal deformities in the adolescent or adult with repaired UCLP are characterized first by uncorrected features of the primary malformation that may be compounded by previous surgical maneuvers and ongoing unfavorable nasomaxillary growth. Lesser degrees of the malformation will be seen in the adult, depending on the success of primary nasal reconstructive maneuvers.[31,33,60,127,147,160,177,178,232,253,254] The most favorable definitive rhinoplasty results in the patient with UCLP are achieved when issues with the underlying maxillary skeleton (including irregularities and defects of the floor of the nose, the piriform rims, and the anterior nasal spine) are first corrected (Figs. 38-25 through 38-30). Even if mixed-dentition bone grafting of the cleft skeletal defects is successful, nasal rim and floor recontouring and orthognathic procedures will be required in the majority of patients before definitive rhinoplasty is performed (see Chapter 32). The correction of the presenting UCLP nasal deformities in the adolescent or adult requires consideration of the following:

- The maxillary position in relation to the mandible below and the facial skeleton above
- The shape and symmetry of the piriform rims, the nasal floor, and the anterior nasal spine
- The morphology of the osseocartilaginous nasal vault
- The symmetry and projection of the nasal lobule and tip
- The quality of the nasal soft-tissue envelope
- The quality of the internal nasal lining
- The deformities of the septum and the turbinates

Alar Rims and Nasal Tip

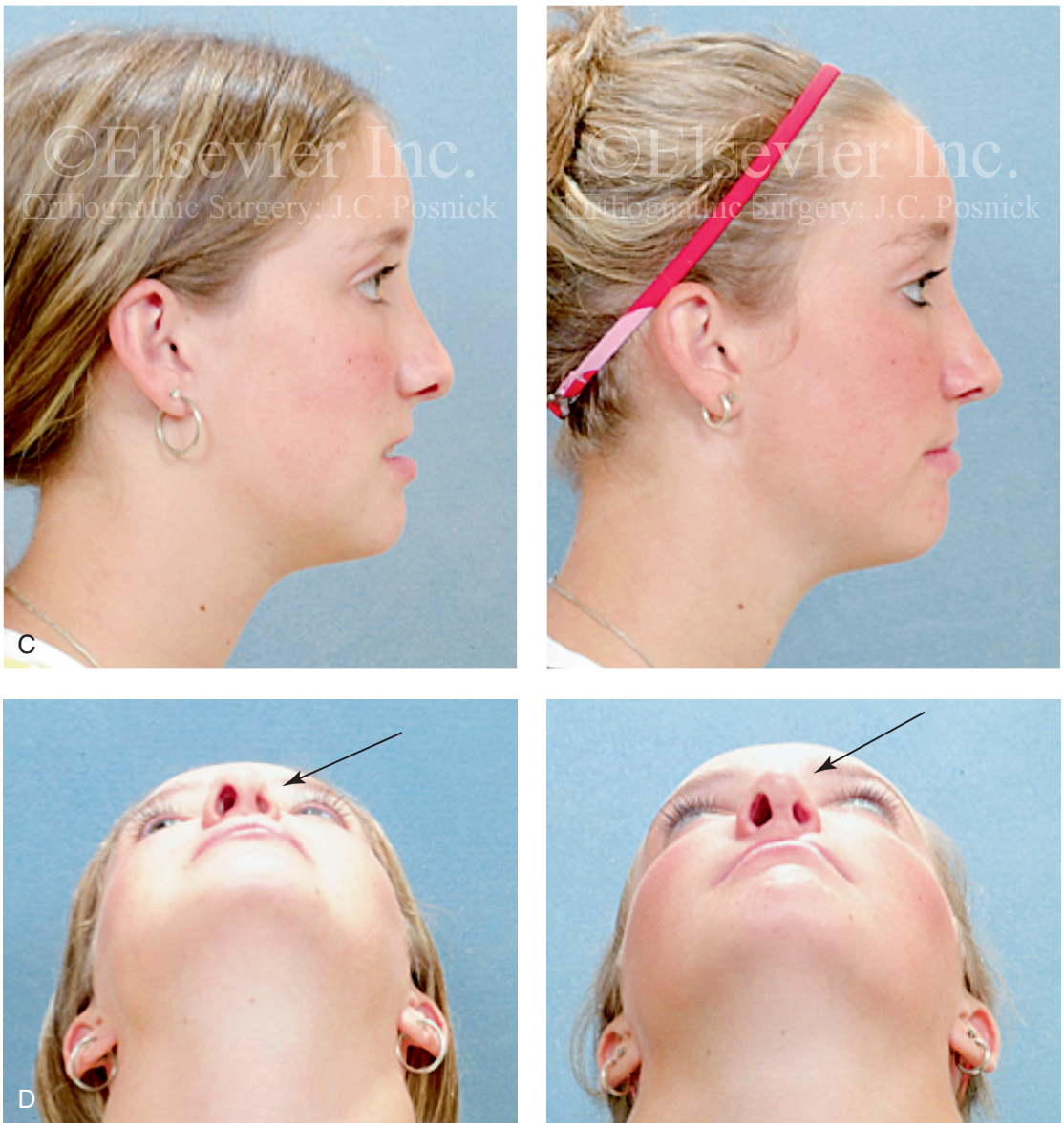
The ipsilateral alar rim may lie caudal, lateral, and posterior as compared with the contralateral side. A hypoplastic malposed LLC may be superimposed on a residual underlying maxillary deformity. An incompletely repaired orbicularis oris muscle may also be a factor. Nasal tip projection is often inadequate, with asymmetry and foreshortening toward the cleft side.

©Elsevier Inc.
Orthognathic Surgery; J.C. Posnick

• **Figure 38-25** A teenage girl was born with UCLP of the right side. Despite her having recently undergone a rhinoplasty by another surgeon, residual nasal deformities remained. She was referred by her orthodontist to this surgeon with concern about the residual cleft dental issues and jaw alignment. The patient wished to discuss the appearance of protruding ears and residual cleft nasal deformities. The bothersome nasal features included a dorsal hump, a drooped and asymmetric nasal tip, and difficulty breathing. She refused any further maxillary skeletal procedures. She agreed to an open rhinoplasty that included nasal osteotomies (in-fracture and straightening); dorsal reduction (bone and cartilage); nasal tip maneuvers (modification of the lower lateral cartilages and septal cartilage caudal strut grafting); septoplasty; and inferior turbinate reduction. She also underwent simultaneous external otoplasty that included conchal reduction (elliptical excisions) and deepening of the scaphal folds (Mustarde stitches). **A,** Frontal views before and after reconstruction. **B,** Oblique facial views before and after reconstruction. **C,** Profile views before and after reconstruction.

©Elsevier Inc. Orthognathic Surgery; J.C. Posnick

• **Figure 38-26** A teenage girl who was born with UCLP of the left side underwent primary lip and palate repair at another institution. She was referred to this surgeon and underwent successful bone grafting during the mixed dentition followed by the orthodontic closure of the cleft–dental gap. Growth of the maxilla was satisfactory, with good occlusion achieved with the use of orthodontic mechanics. She was displeased with the residual cleft nasal deformities, which included an asymmetric flat tip of the nose, a cicatrized left nasal opening, and a lifelong history of difficulty breathing through the nose. She underwent an open rhinoplasty that included nasal osteotomies (in-fracture and straightening); minimal dorsal reduction (bone and cartilage); nasal tip maneuvers (modification of the lower lateral cartilages and septal cartilage caudal strut grafting); septoplasty; and inferior turbinate reduction. **A,** Frontal views with smile before and after reconstruction. **B,** Oblique facial views before and after reconstruction.



• **Figure 38-26, cont'd C,** Profile views before and after reconstruction. **D,** Worm's-eye views before and after reconstruction. Note the irreversibly cicatrized left external nasal valve as a result of overresection at the time of primary lip repair.

Skeletal Framework of the Nose

The nasal bones are frequently wide and asymmetric. The dorsum itself may be high, low, or normal, with much individual variation. The mid cartilaginous vault may be curved, with collapse toward the side of the cleft.

Maxillary Base

The maxilla may be sagittally retrusive but more so on the cleft side, which results in the ipsilateral pyriform aperture being located caudal, lateral, and posterior. Previous bone grafting to the hard palate, the alveolus, and the floor of the nose may have been ineffective, with continued defects and asymmetry.

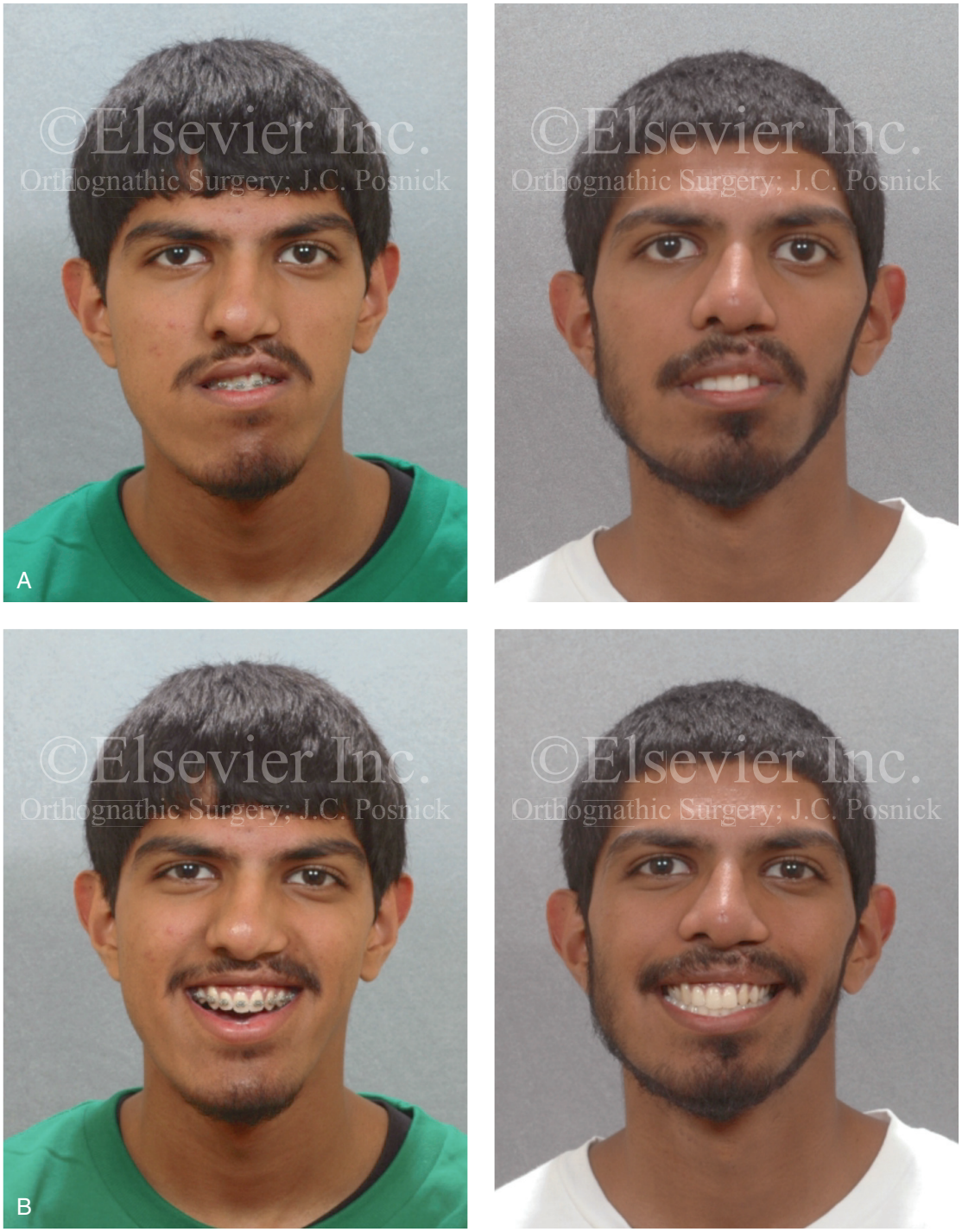
Nasal Airway

Asymmetric narrowing of the nasal cavity with obstruction to airflow is generally present. The septum may be displaced from the vomerine groove, and the cartilaginous portion may be thickened and buckled. The inferoanterior portion of the septum is typically displaced away from the cleft, whereas the superior cartilaginous septum curves into the cleft as it rises to the tip. The inferior turbinates, especially on the cleft side, are frequently hypertrophic; they consume much of the airway space with the collapse of the internal nasal valve. The bony septum may also be deviated. The floor of the nose just above the incisors will be asymmetric

Text continued on p. 1661

©Elsevier Inc. Orthognathic Surgery; J.C. Posnick

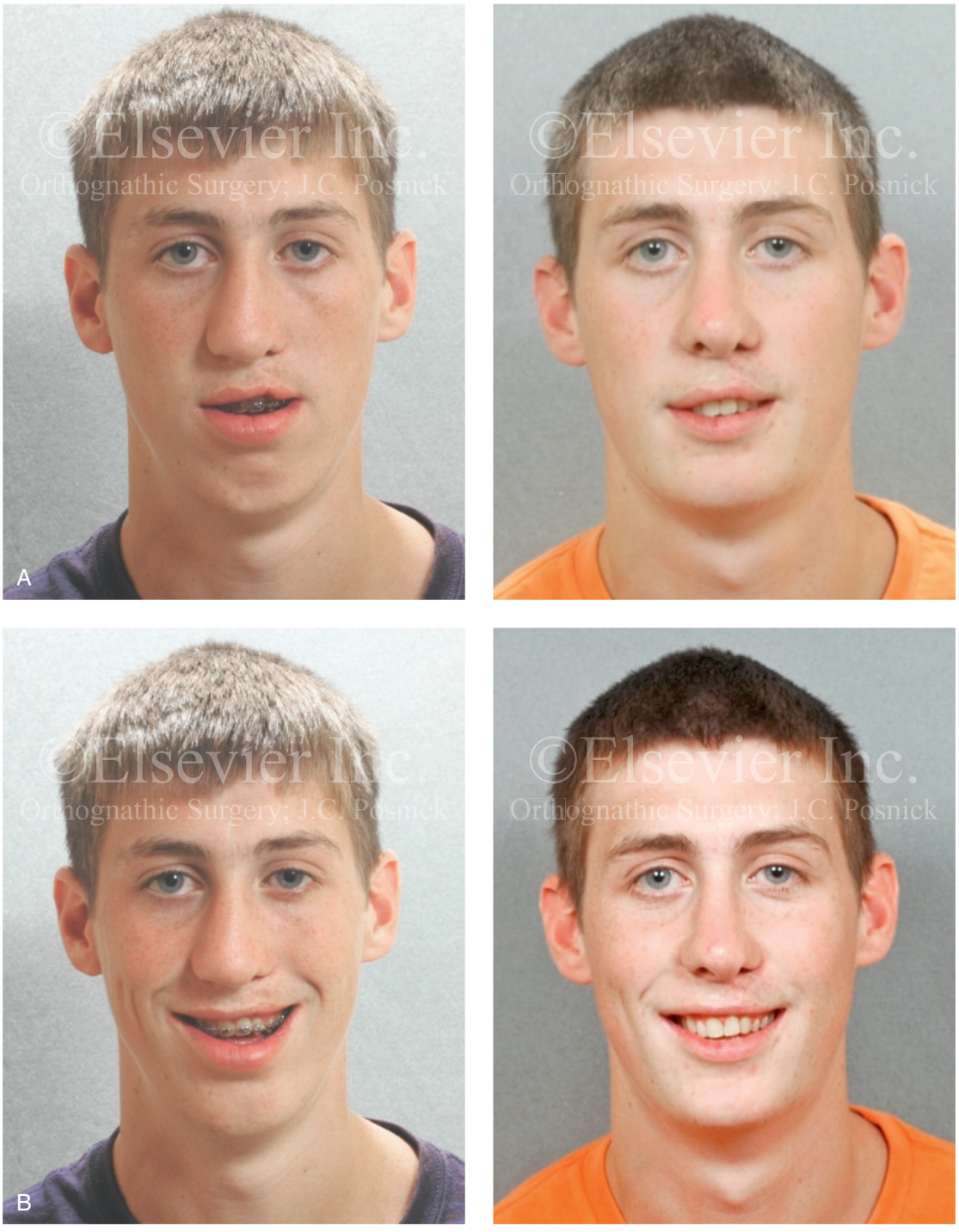
• **Figure 38-27** A 16-year-old girl who was born with van der Woude syndrome that included complete UCLP and lower lip pits was managed by this surgeon since the time of her birth. She underwent lip and palate repair during early childhood, and this was followed by successful bone grafting and fistula closure during the mixed dentition. She had normal maxillary growth and underwent standard orthodontic treatment with maintenance of the cleft–dental gap (see Fig. 32-7). She underwent an open rhinoplasty when she was 15 years old that included nasal osteotomies (in-fracture and straightening); minimal dorsal reduction (bone and cartilage); nasal tip maneuvers (reshaping of the lower lateral cartilages and septal cartilage caudal strut grafting); septoplasty; and inferior turbinate reduction. **A,** Oblique facial views before and after rhinoplasty. **B,** Profile views before and after rhinoplasty.



• **Figure 38-28** A high school student was born with a complete UCLP of the left side. He underwent lip and palate repair at another institution. He was then referred to this surgeon during the mixed dentition and underwent successful bone grafting and fistula closure. The cleft–dental gap was closed with orthodontic mechanics. He developed a jaw discrepancy that was characterized by maxillary deficiency with secondary deformity of the mandible and chin region (see Fig. 32-8). Six months after successful orthognathic surgery and orthodontic treatment, he underwent cleft rhinoplasty that included rib cartilage (caudal strut) grafting. He is shown before and then after orthognathic and nasal reconstruction. **A,** Frontal views in repose before and after reconstruction. **B,** Frontal views with smile before and after reconstruction.

Continued

• **Figure 38-28, cont'd C,** Oblique facial views before and after reconstruction. **D,** Profile views before and after reconstruction.



• **Figure 38-29** A young man was born with complete UCLP. He was treated by other surgeons with procedures that included primary cleft lip repair; cleft lip revision; primary cleft palate repair; cleft palate revision; the placement of superiorly based pharyngeal flap; iliac grafting of the cleft defect; cleft rhinoplasty; and Le Fort I osteotomy with distraction osteogenesis (RED device). When he was 17 years old, he was then referred to this surgeon with residual cleft deformities that included the following:

- Nasopharyngeal stenosis with obstructive sleep apnea (apnea–hypopnea index: 11.4 events/hour, with desaturations to 89%)
- Persistent oronasal fistula
- Residual cleft jaw deformity with Class III open-bite malocclusion
- Chronic nasal obstruction with septal deviation and enlarged turbinates
- Residual cleft nasal deformity
- Cleft lip scar deformity with a lack of continuity of the orbicularis oris muscle

The patient agreed to a comprehensive orthodontic and surgical approach. His procedures included the following: redo Le Fort I osteotomy in two segments (+7 mm horizontal advancement , transverse widening, and oronasal fistula closure) with iliac grafting; bilateral sagittal split osteotomies of the mandible (horizontal advancement and clockwise rotation); oblique osteotomy of the chin (horizontal advancement and vertical shortening); redo septoplasty; redo inferior turbinate reduction; and recontouring of the nasal rims. One year later, the patient underwent cleft rhinoplasty, including rib cartilage (caudal) strut grafting; cleft lip scar revision, including repair of the orbicularis oris muscle; and the revision of the pharyngeal flap to relieve stenosis. A postoperative attended sleep study confirmed complete resolution of the obstructive sleep apnea. **A,** Frontal views in repose before and after reconstruction. **B,** Facial view with smile before and after reconstruction.

Continued

• **Figure 38-29, cont'd C,** Oblique facial views before and after reconstruction. **D,** Profile views before and after reconstruction. Note that the pharyngeal flap revision (release of stenosis) was important to resolve mucous trapping and to improve breathing.

• **Figure 38-30** A teenager was born with complete UCLP of the left side. He was treated by this surgeon with procedures that included cleft lip repair (3 months of age); cleft palate repair (12 months of age); superiorly based pharyngeal flap (5 years of age); and cleft bone grafting with fistula closure during the mixed dentition (8 years of age). He is congenitally missing the left maxillary lateral incisor and the first bicuspid. He underwent standard orthodontic treatment and had satisfactory growth of the jaws. Dental gaps remain for eventual implants and dental restorations. When he was 15 years old, he underwent nasal reconstruction that included septoplasty; inferior turbinate reduction; and rhinoplasty including rib cartilage (caudal) strut grafting. **A,** Frontal views in repose before and after reconstruction. **B,** Frontal views with smile before and after reconstruction.

Continued

• **Figure 38-30, cont'd C,** Oblique facial views before and after reconstruction. **D,** Profile views before and after reconstruction.

and irregular, thereby further preventing the normal flow of air. The external valve may be scarred and cicatrized from previous surgery, which also further constricts nasal airflow.

Analysis of Residual Nasal Deformities in the Adolescent or Adult with Repaired Unilateral Cleft Lip and Palate

Key Points

- Is the nasal lining deficient or just distorted?
- Is the overlying nasal soft-tissue envelope deficient or just distorted?
- Are the nasal bones deformed and require reconstruction?
  - How is the dorsal height? How is the mid-dorsal width?
  - Is the bony vault crooked?
- Is the mid-cartilaginous vault crooked?
- Is the nasal tip projection adequate?
- Does the external nasal valve have scar cicatrization?
- What is the extent of the LLC deficiency? Will augmentation be required?
- Is the skeletal component of the nasal aperture deficient and asymmetric? If so, bone grafting, fistula closure, and/or recontouring will be required.
- Is the maxilla hypoplastic? If so, orthognathic surgery will be indicated.
- What are the current areas of nasal obstruction
  - External nasal valves?
  - Internal nasal valves?
  - Turbinated cavities?
  - Posterior (cavity) apertures?
  - Tight pharyngeal flap?
  - Tonsils and adenoids?

In most patients with repaired UCLP, an adequate overlying soft-tissue envelope and underlying internal nasal lining are present. Although the nasal bones are deformed, they are usually adequate to allow for reconstruction through a combination of osteotomies and dorsal reduction. The mid-cartilaginous vault may be crooked to the extent that spreader grafts are required. A cartilage caudal strut graft will be required for tip straightening and projection in virtually all cases. If the septal cartilage is available, it is usually adequate. If greater structural support is required, the use of rib cartilage is preferred. If the quality of the LLC is adequate, reconstruction simply by recontouring and suture-pexy is carried out. If not, then augmentation of the LLC with additional cartilage grafting is required (i.e., alar contour graft or lateral crural strut graft).

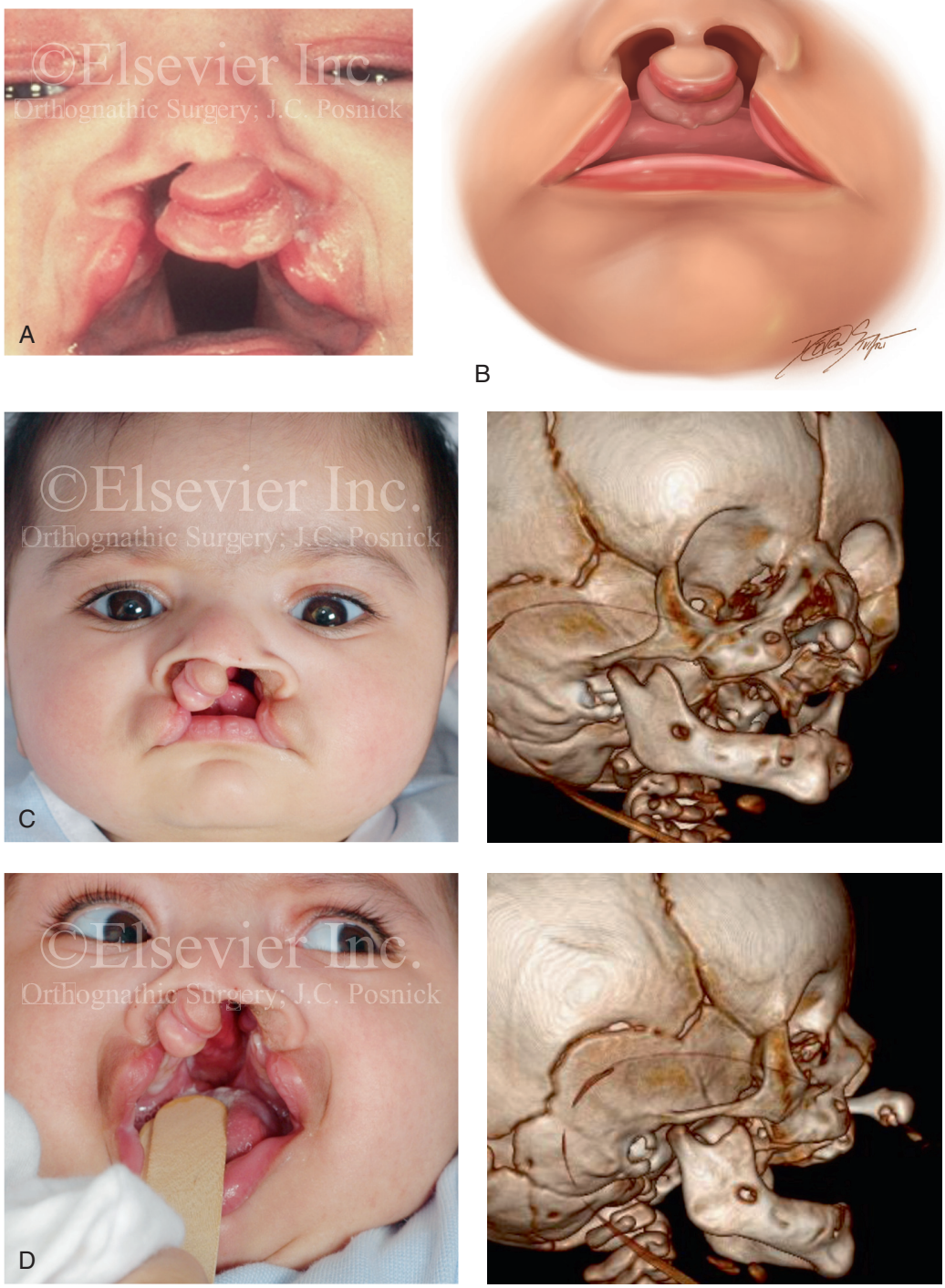
Nasal Deformities in the Newborn with Bilateral Cleft Lip and Palate (Fig. 38-31)

- The columella is short, but there is a wide variation in this feature among individuals.
- The nasal tip is flat and broad, and it appears bifid.
- The nasal alae are flat and wide. From a worm's-eye view, the alar rims appear to be "S"-shaped.
- The base of the alae on each side are displaced laterally and sometimes inferiorly and posteriorly.
- Both nostrils are horizontally oriented.
- The LLCs are typically severely buckled and deformed. The medial crura are short and widely separated at the nasal tip. The lateral crura are flat and elongated.
- The dome of the nasal tip is angled obtusely.
- The nasal floor is clefted (open).
- The columella, the caudal end of the septum, and the anterior nasal spine are displaced anteriorly as a reflection of the forwardly projected premaxilla.
- The nasal tip and the nostrils may be asymmetric as a reflection of the displaced premaxilla.

Basic Nasal Reconstructive Maneuvers for the Primary Bilateral Lip Repair (Figs. 38-32 and 38-33)

- The philtral flap is raised as part of the primary bilateral lip repair. Circulation to the philtral flap is based on attachment to the columella soft tissues. Elevation of the medial and lateral nasal soft-tissue flaps for nasal sill and floor reconstruction is carried out after the philtral skin flap is accomplished (Fig. 38-32, *B,C*).
- Along the lateral aspect of the nose, the nasal soft tissues (including the inferior turbinate) are released from the pyriform rim and the lateral nasal wall with the use of an elevator (Fig. 38-33, *A*).
- Next, the buckled and flattened lateral crus of the LLC is released from the overlying nasal soft tissues with the use of Stevens scissors (Fig. 38-33, *B*).
- The elevated lateral nasal flap (including the inferior turbinate) will be advanced medially, superiorly, and anteriorly to facilitate nasal sill and floor reconstruction (Fig. 38-32, *D*).
- Along the medial aspect of the nose, the nasal soft tissues (including the medial crus of the LLC) are released from the anterior nasal spine and septal cartilage with the use of an elevator and Stevens scissors. The elevated medial nasal flap will be advanced laterally to facilitate nasal sill and floor reconstruction) (Fig. 38-33, *C*).
- The release of the orbicularis oris muscle from its abnormal bone and soft-tissue attachments is accomplished with the use of Stevens scissors. Reconstruction of the orbicularis oris muscle with interrupted sutures achieves continuity across the lip. Just before muscle approximation, gentle mechanical (orthopedic) "thumb" pressure can be applied to the premaxilla if needed to improve its position. Reasonable alignment of the premaxillary segment just before repair is preferred to achieve a tension-free soft-tissue lip closure (Fig. 38-32, *D*).
- Recurrent buckling of the lateral crus of the LLC early after surgery is limited by the placement of a stent (i.e.,

Text continued on p. 1666

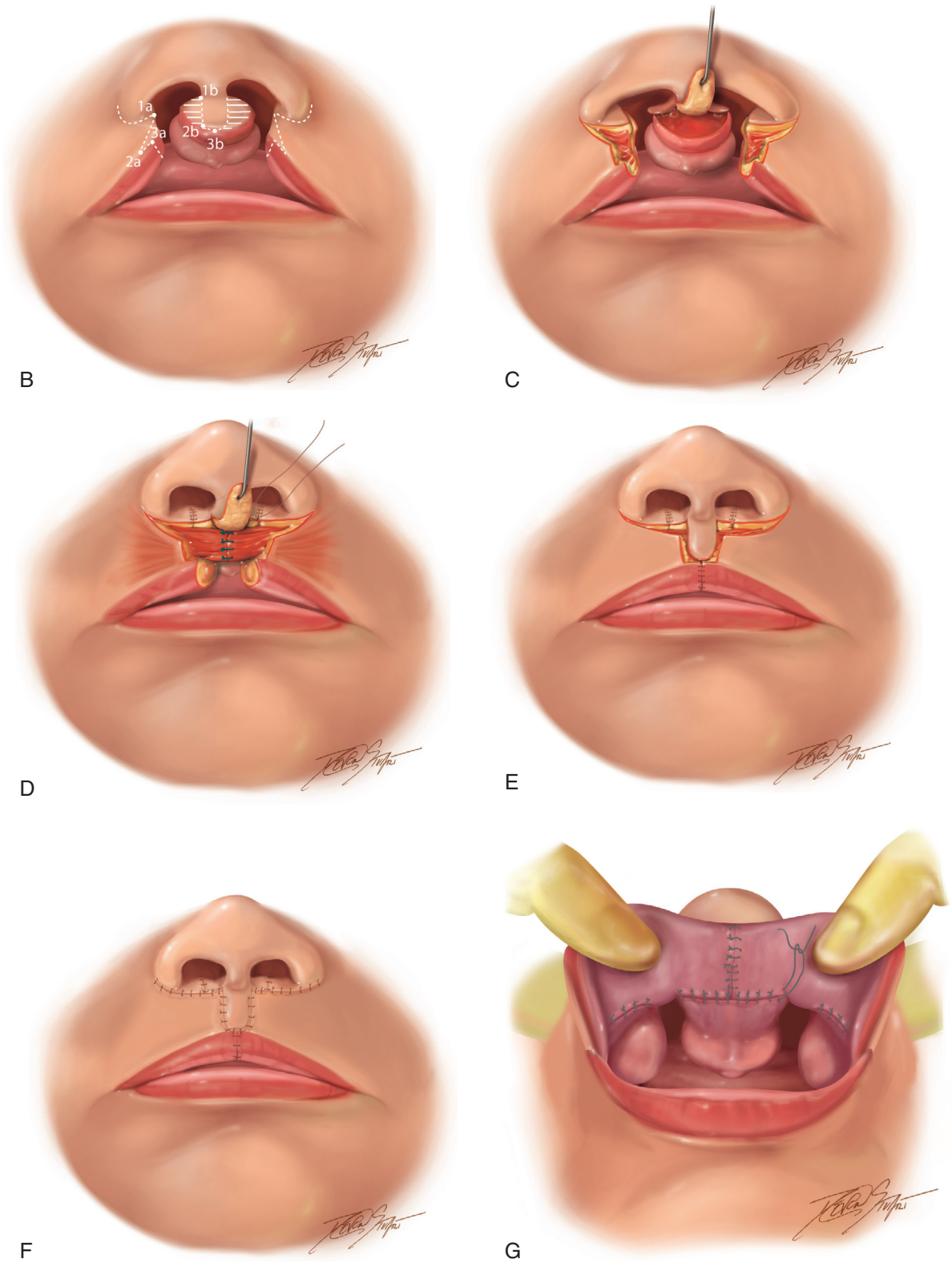


• **Figure 38-31** Demonstration of typical bilateral cleft lip nasal malformation. **A,** Worm's-eye view of a newborn with complete bilateral cleft lip and palate. **B,** Illustration that indicates typical dysmorphology of the maxillary base, the caudal septum, and the nasal tip in a newborn with unrepaired bilateral cleft lip and palate (BCLP). **C** and **D,** Facial and computed tomography scan views of a newborn with BCLP. An asymmetric forwardly projecting premaxilla is shown.

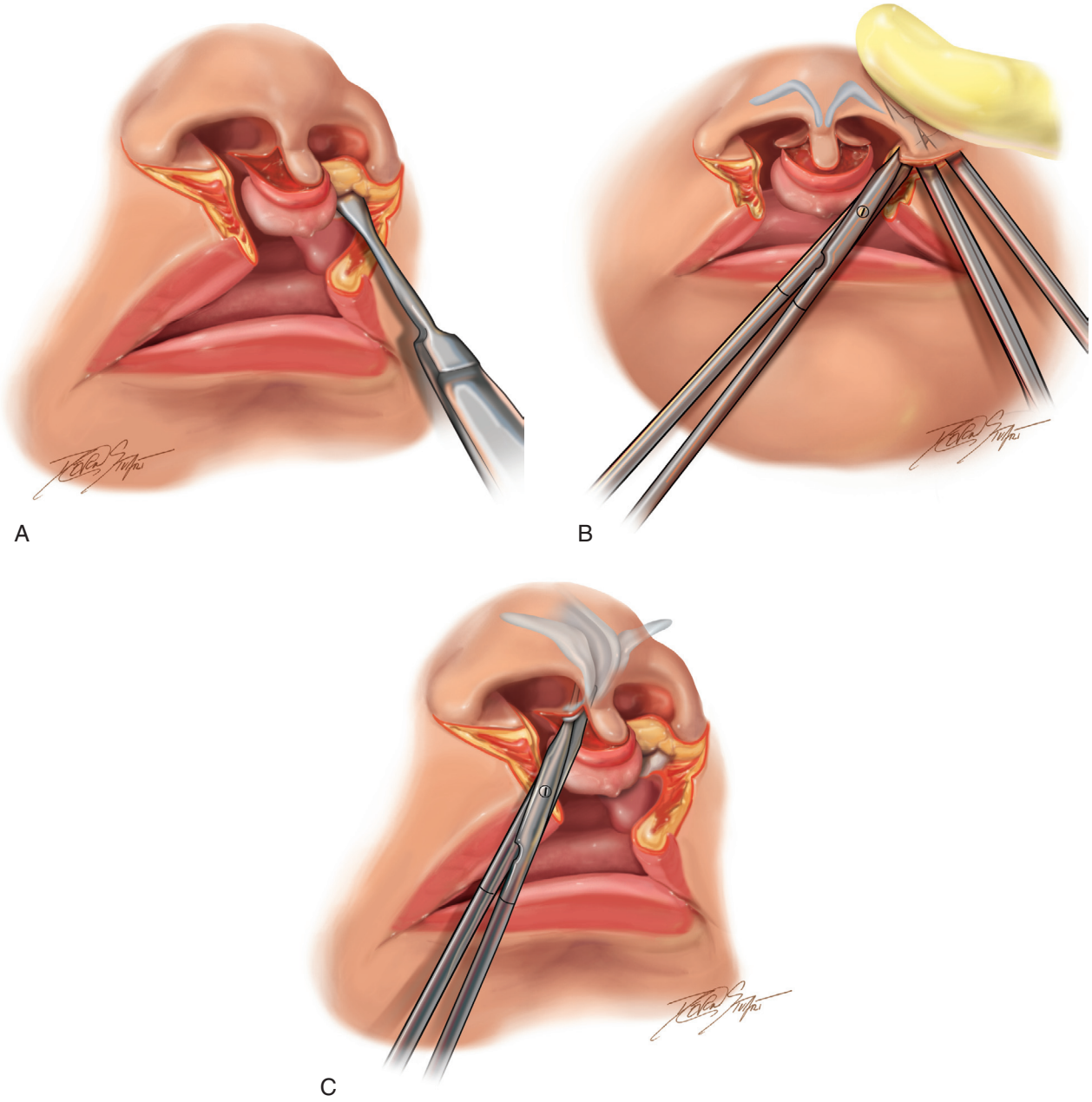
A

• **Figure 38-32 A,** Illustration of typical facial malformations and distortions in a newborn with complete BCLP.

Continued



• **Figure 38-32, cont'd** **B** and **C,** Illustrations of the basic technique used by this surgeon for primary bilateral cleft lip reconstruction. In **B,** the incisions are indicated *(dotted lines)*, with redundant tissue to be excised shown *(diagonal lines)*. In **C,** the philtral skin flap has been elevated with circulation maintained through the soft tissues of the columella. The lateral lip flaps include vermilion–mucosa soft-tissue extensions to be used for the reconstruction of the central red lip. The orbicularis oris muscle has been dissected free of its abnormal bony and soft-tissue attachments and also released from the overlying lip skin. The medial and lateral nasal floor flaps have already been elevated within each nostril (see Fig. 38-33). **D,** The orbicularis oris muscle is then approximated across the midline and sutured together at the columella base. Just before muscle approximation, gentle mechanical orthopedic "thumb" pressure can be applied to the premaxilla if needed to improve its position. Improved alignment of the premaxillary segment just before repair will allow for a tension-free soft-tissue lip closure. The anterior portion of the nasal floor is then reconstructed on each side by suturing the advanced medial and lateral nasal floor flaps. **E,** The vermilion–mucosa extensions of each lateral lip flap are approximated and sutured in the midline to create the vermilion–mucosa junction. **F,** The philtral flap is aligned with the advanced vermillion extensions of the lateral lip flaps (i.e., the vermilion–cutaneous junction) and sutured in place. The cutaneous layer of each lip flap is advanced and sutured first at the columella base and then at the vermillion on each side. The philtral columns and the nasal sills are sutured. **G,** The intraoral mucosa is aligned to establish a functional vestibule. Excision of redundant red lip was initially accomplished just after elevating the philtral flap. The lip side of the vestibule is reconstructed with mucosa from the advanced lateral lip flaps. The alveolar side of the vestibule is reconstructed with the retained mucosa on the premaxillary segment. Note: Alveolar clefts and fistulas remain.



• **Figure 38-33** Illustrations of the bilateral cleft nasal maneuvers that are carried out as part of the primary repair. **A,** Along the lateral aspect of the nose, the nasal soft tissues (including the inferior turbinate) are released from the pyriform rim, and the lateral nasal wall is released with an elevator inserted through the open lip. **B,** Next, the buckled and flattened lateral crus of the lower lateral cartilage is released from the overlying nasal soft tissues with a Stevens scissors inserted through the open lip. The elevated lateral nasal flap (including the inferior turbinate) will be advanced medially, superiorly, and anteriorly to facilitate nasal floor reconstruction. **C,** Along the medial aspect of the nose, the nasal soft tissues (including the medial crus of the lower lateral cartilage) are released from the anterior nasal spine, and septal cartilage with the use of a Stevens scissors inserted through the open lip. The elevated medial nasal flap will be advanced laterally to facilitate nasal floor reconstruction. Recurrent buckling of the lateral crus is limited early after surgery with the use of an intranasal stint (packing) in each nostril. Sutures through the nasal soft tissues are also placed to pexy each lower lateral cartilage.

packing) in each nostril. Sutures through the nasal soft tissues and LLCs on each side may also be used to elevate and maintain the shape of the nasal rims.

Two typical patients with bilateral cleft lip and palate (BCLP) who have been treated with the use of the techniques described are shown after primary lip and nasal repair (Figs. 38-34 and 38-35). Pre-BCLP lip-repair maneuvers have been described in the literature for the purpose of repositioning a protrusive premaxilla and to improve nasal anatomy. They include: taping; the use of a nasoalveolar molding device; the use of a Latham pinning device; lip adhesion; and the application of gentle direct mechanical (orthopedic) backward pressure on the premaxilla.[2,26,78,117,229,244,153] Each of these maneuvers has its strong advocates and its vocal detractors. Despite decades of dispute and discussion, standards of care that involve this important cleft reconstruction topic remain limited.[172]

Residual Nasal Deformities in the Adolescent and Adult with Repaired Bilateral Cleft Lip and Palate

Residual nasal deformities in the adolescent or adult with repaired BCLP include any uncorrected features of the primary malformation that may be compounded by previous surgical maneuvers and ongoing unfavorable nasomaxillary growth. Lesser degrees of the malformation will be seen in the adolescent and adult depending on the success of primary nasal reconstructive maneuvers. The most favorable rhinoplasty results are achieved only after deformities of the underlying maxillary skeleton (i.e., the floor of the nose, the piriform rims, and the anterior nasal spine [e.g., maxillary hypoplasia]) are first reconstructed. It is now clear that, for a majority of individuals with repaired BCLP, orthognathic procedures will be required to correct baseline facial disharmony before definitive rhinoplasty (see Chapter 33).

©Elsevier Inc.
Orthognathic Surgery; J.C. Posnick

• **Figure 38-34** A newborn with complete BCLP, tracheomalacia, and other congenital anomalies is shown. At the time of primary cleft lip repair, he underwent nasal reconstructive maneuvers as described in the text (see Figs. 38-32 and 38-33). He is shown **A,** soon after birth and **B,** at 3 months of age, just 1 week after primary cleft lip and nasal reconstruction. **C,** He is next shown at 2.5 years of age having also undergone cleft palate repair via the Bardach two-flap technique.

©Elsevier Inc.
Orthognathic Surgery; J.C. Posnick

• **Figure 38-35 A,** A newborn girl with complete BCLP is shown. At the time of primary cleft lip repair, she underwent nasal reconstructive maneuvers as described in the text (see Figs. 38-32 and 38-33). **B,** She is next shown just 1 week after primary lip and nasal reconstruction. **C,** She is then shown at 1 year of age just before cleft palate repair. Note the decreased height of the columella that is part of the malformation; she will require definitive nasal reconstruction with the use of an open technique and a rib cartilage caudal strut during the early teenage years (Fig. 38-36).

Residual BCLP nasal deformities in the adolescent or adult that frequently require correction include the following:

- A short columella and a lack of caudal projection of the inferior aspect of the septum
- The flattening of the osseocartilaginous vault
- Nasal tip bifidity
- The buckling of the downward rotation of the LLCs
- The lateral displacement of the alar bases that results in the horizontal rotation of the nostrils

In addition to the usual open rhinoplasty maneuvers carried out in the adolescent or adult with a repaired BCLP, the short columella defines the deformity.[178,221,253] Correction of the short columella in a patient with BCLP is preferably accomplished with the use of a crafted autogenous rib cartilage caudal strut graft. This option is an effective method to provide structural support for the stretching of the columella skin and the nasal soft-tissue envelope over the graft to achieve needed projection (Figs. 38-36 through 38-39). The rib cartilage graft is stabilized to the base of the maxilla with a no. 35 threaded Kirschner wire (K-wire). After modification, the LLCs are sutured together and over to top of the rib cartilage caudal strut to form the superficial aspect of the new tip (Fig. 38-40).

The American Cleft Palate–Craniofacial Association parameters of care document that addresses secondary cleft lip nasal reconstruction includes only the following statements[172]:

- Although rhinoplasty and nasal septal surgery are usually advocated only after the completion of nasal growth, earlier intervention for reasons of airway problems or nasal tip deformity may be indicated.
- The repair of the cleft lip nasal deformity can be accomplished with limited external incisions on the nose.
- The timing of nasal surgery should be discussed with the patient and the family so that the goals are understood and the expectations are realistic.
- The patency of the nasal airway should be considered when planning either nasal reconstructive procedures or secondary velopharyngeal operations (e.g., a pharyngeal flap, another type of pharyngoplasty).

Until clear parameters regarding the timing and technique of secondary cleft nasal surgery become available, individual surgeon experience and personal preferences will remain standard practice. Unfortunately, this can easily lead to frustration and miscommunication among the patient, the family, and the treating clinicians.

Timing of Definitive Cleft Nasal Reconstruction

To maximize long-term nasal breathing, adequate sinus drainage, and the enhancement of facial aesthetics, a definitive cleft rhinoplasty should only be undertaken after successful alveolar bone grafting, including the stabilization of the premaxilla in the case of BCLP and fistula closure. If orthognathic corrections are required, then definitive nasal reconstruction is best delayed until at least 6 months after successful skeletal reconstruction. Recent studies conducted at both Boston Children's Hospital and the Hospital for Sick Children in Toronto, Ontario, Canada, confirm that approximately 50% of adolescents and adults with UCLP and 65% to 77% of adolescents and adults with BCLP will require orthognathic surgery. The correction of the cleft jaw deformity is preferably carried out when the patient is between 15 and 18 years old and before graduation from high school (see Chapters 32, 33, and 34).

NOTE: This author does not favor repetitive limited rhinoplasty maneuvers performed throughout childhood. Although they are well intended, nasal procedures carried out after the primary repair but before definitive reconstruction may result in additional scarring and distortions that will limit long-term aesthetic and functional airway results.

Congenital and Posttraumatic Saddle-Nose Deformities

The hallmark of most *congenital and posttraumatic saddle-nose deformities* is the deficiency of both the *osseous and cartilaginous vaults.* The surgical correction of a saddle-nose deformity was first described by Robert Weir in 1889, when he wrote a report entitled "Restoring Sunken Noses." He implanted the breastbone of a freshly killed duck below the skin of a severely shrunken nose in a young syphilitic patient as a means of reconstruction.[250] In current times, a frequent form of saddle nose results from either congenital deformity (e.g., bifid cleft nose, Binder syndrome, cranio–fronto–nasal dysplasia; Figs. 38-41 through Fig. 38-44) or from an acquired postnasal fracture cause (e.g., complex naso–orbito–ethmoid fractures, direct low-velocity trauma to the nasal bones in the growing child; Fig. 38-45).[184] The congenital and posttraumatic saddle nose as defined includes the deformity of the osseous vault (i.e., the nasal bones) and the cartilaginous vault (i.e., the quadrangular cartilage). These saddle-nose deformities generally present with an acceptable soft-tissue envelope and nasal lining. They also generally have an intact septum (i.e., no septal perforation). The reconstruction required for these saddle-nose deformities will vary with their cause (e.g., cocaine related, autoimmune conditions, iatrogenic postseptoplasty-induced "depressed" nose).* The postseptoplasty saddle-nose deformities present with an intact osseous vault but with significant septal perforations that result in a depressed cartilaginous vault; this is discussed later in this chapter.

Text continued on p. 1687

*References 42, 63, 64, 106, 107, 119, 154, 175, 180, 181, 183, 221, 230, 242, 250, 251, 253

• **Figure 38-36** A child was born with complete BCLP. She underwent two attempts at cleft lip repair at another institution that resulted in wound dehiscence and necrosis of the philtral soft tissues. This was likely the result of a combination of factors, including the following: 1) a protrusive premaxilla before repair; 2) inadequate release of and then repair of the orbicularis muscle; 3) and the incisions used to elevate the philtral flap. She was referred to this surgeon and then underwent successful lip reconstruction that included gentle mechanical and orthopedic "thumb" pressure on the premaxilla just before approximation of the orbicularis oris muscle. This was followed by palate repair with the use of the Bardach two-flap technique when she was 14 months old and then successful bone grafting during the mixed dentition. During the patient's early teenage years, there was harmonious growth and adequate sagittal projection of both jaws. The teeth were orthodontically aligned, with closure of the cleft–dental gaps. The cleft nasal deformity was characterized by a short columella and a wide depressed tip. The soft-tissue deficiency of the upper lip (philtrum) remained as a result of necrosis during infancy. A decision was made to not construct an Abbe flap, which would scar the lower lip and result in a "patch" (color mismatch) with the upper lip. An open rhinoplasty was carried out that included modification of the lower lateral cartilages and rib cartilage caudal strut grafting with stretching of the nasal soft tissues to create improved tip position and shape. Composite resin buildups and contouring of the hypoplastic anterior maxillary teeth were carried out by a restorative dentist after definitive nasal reconstruction. **A,** The patient is shown at 4 months of age, when she was referred to this surgeon, and at 14 years of age after successful lip and palate repair during infancy, bone grafting during the mixed dentition, and just before the removal of the orthodontic appliances and definitive nasal reconstruction. **B,** Oblique facial views before and at 15 years of age after nasal reconstruction. **C,** Profile views before and at 15 years of age after nasal reconstruction.

• **Figure 38-37** A teenage girl of Arabic descent was born with complete BCLP. She has been managed by this surgeon since birth. She underwent primary lip and nasal repair and palate reconstruction during childhood and then successful bone grafting during the mixed dentition. She required the correction of a cleft jaw deformity when she was 15 years old. The procedures included maxillary Le Fort I osteotomy; sagittal split ramus osteotomies; osseous genioplasty; septoplasty, inferior turbinate reduction, and nasal floor recontouring. Six months later, the patient underwent definitive nasal reconstruction to correct a wide nasal bridge and a depressed tip. Her open rhinoplasty included nasal osteotomies (in-fracture and straightening); dorsal reduction (bone and cartilage); and nasal tip maneuvers (modification of the lower lateral cartilages and rib cartilage caudal strut grafting). **A,** Frontal views before and after cleft orthognathic and nasal reconstruction. **B,** Oblique views before and after cleft orthognathic and nasal reconstruction. **C,** Profile views before and after cleft orthognathic and nasal reconstruction.

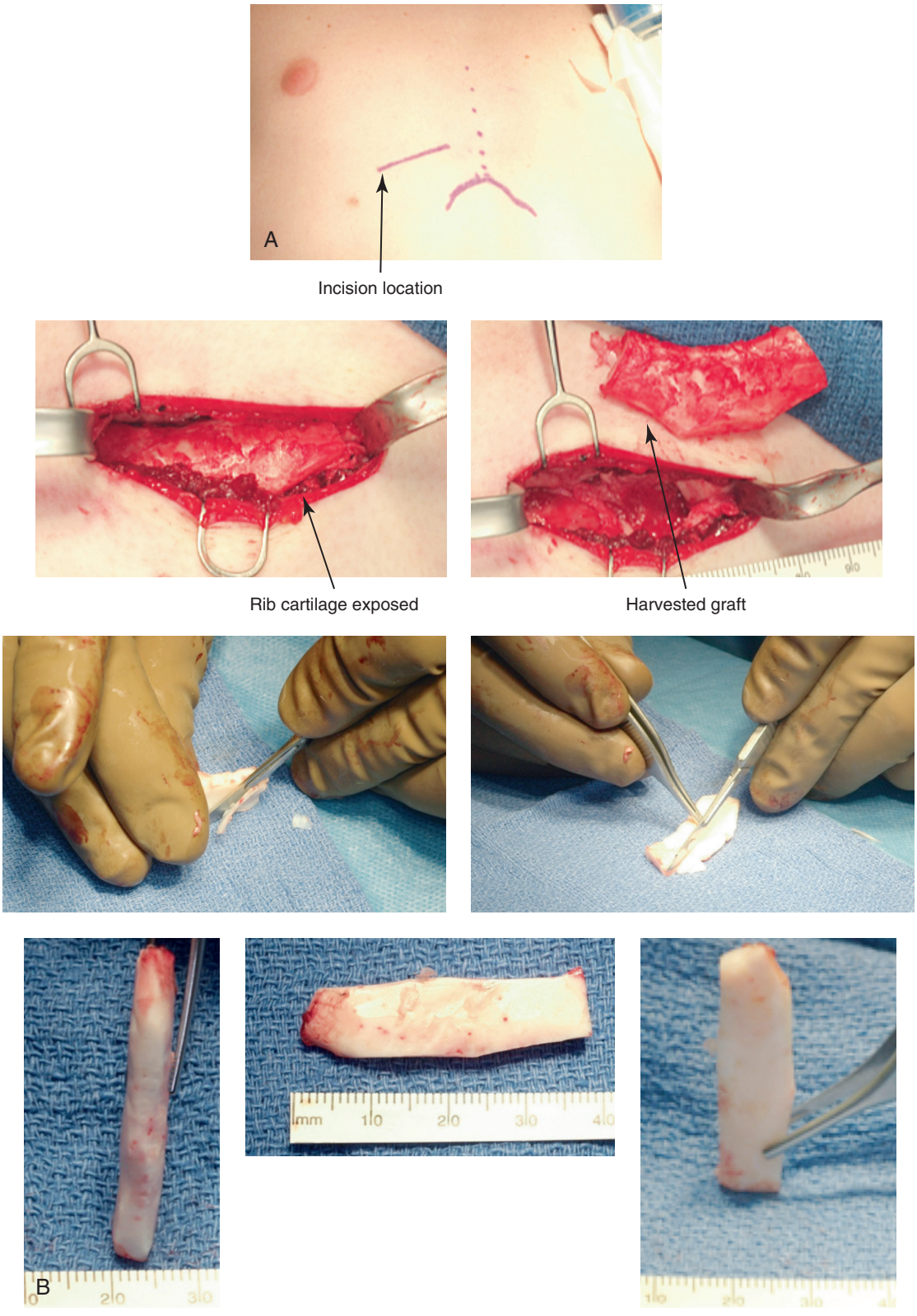
• **Figure 38-38** A teenage Hispanic male was born with complete BCLP. He underwent lip and palate repair and ineffective bone grafting during the mixed dentition by another surgeon. When referred to this surgeon, he presented with maxillary hypoplasia, mobile premaxilla, cleft dental gaps, residual fistula, and alveolar clefts. He underwent maxillary Le Fort I osteotomy in three segments and osseous genioplasty. Six months later, he underwent definitive rhinoplasty to manage residual and secondary cleft deformities, including a wide bridge, a depressed nasal tip, and difficulty breathing (see Fig. 33-10). His open rhinoplasty included nasal osteotomies (in-fracture and straightening); dorsal reduction (bone and cartilage); nasal tip maneuvers (modification of the lower lateral cartilages and rib cartilage caudal strut grafting); septoplasty; and inferior turbinate reduction. **A,** Frontal views before and after reconstruction. **B,** Profile views before and after reconstruction.

©Elsevier Inc.
Orthognathic Surgery; J.C. Posnick

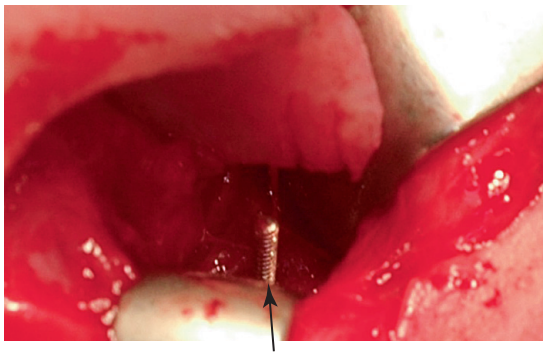
• **Figure 38-39** A boy who was born with complete BCLP. He underwent lip and palatal repair and unsuccessful bone grafting at another institution. He presented to this surgeon when he was 17 years old with severe jaw and nasal deformities, malocclusion, and dental needs. After successful orthognathic surgery and dental rehabilitation, he underwent an open rhinoplasty that included nasal osteotomy (in-fracture); dorsal reduction (bone and cartilage); and nasal tip maneuvers including rib cartilage (caudal strut) placement (see Fig. 33-16). **A,** Frontal views in repose before and after reconstruction and rehabilitation. **B,** Frontal views with smile before and after reconstruction and rehabilitation.

Continued

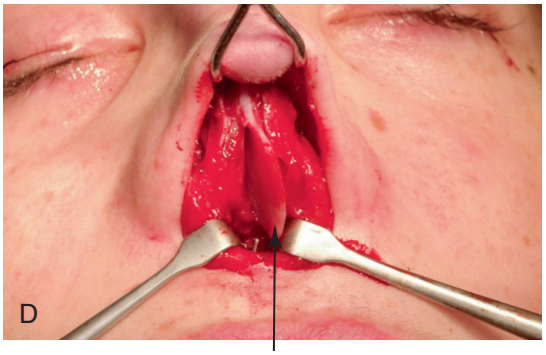
• **Figure 38-39, cont'd** **C,** Oblique views before and after reconstruction and rehabilitation. **D,** Profile views before and after reconstruction and rehabilitation.



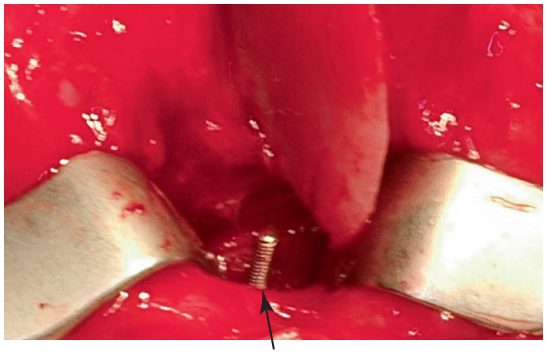
• **Figure 38-40** Intraoperative views of rib cartilage grafting technique. **A,** The incision, which is 4 cm in length, is directly over the rib cartilage to be harvested. The rib cartilage is then exposed, sectioned, and removed from the chest wall. **B,** The harvested rib cartilage graft is crafted as a caudal strut that is approximately 4 cm. *Continued*



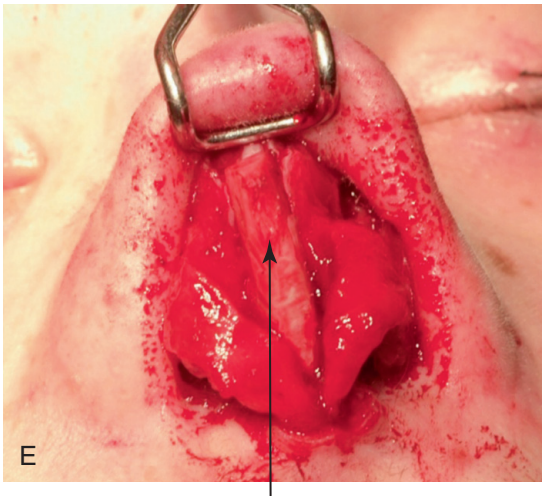
K-wire secured at nasal base
(through anterior maxilla)
projecting up to pierce and secure strut graft



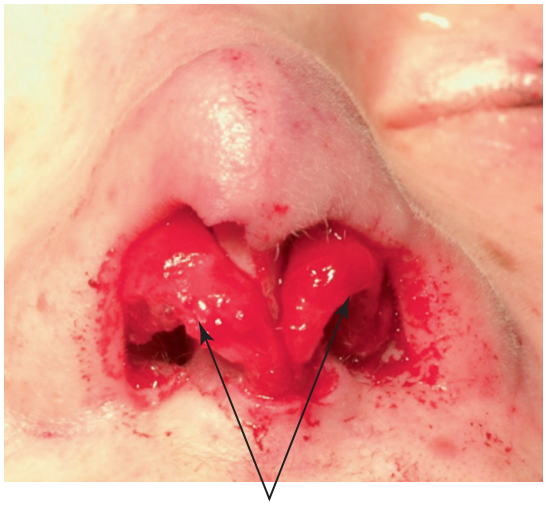
Caudal portion of septal cartilage



K-wire secured at nasal base
(through anterior maxilla)
projecting up to pierce and secure strut graft



Crafted rib cartilage graft secured
to nasal base through K-wire

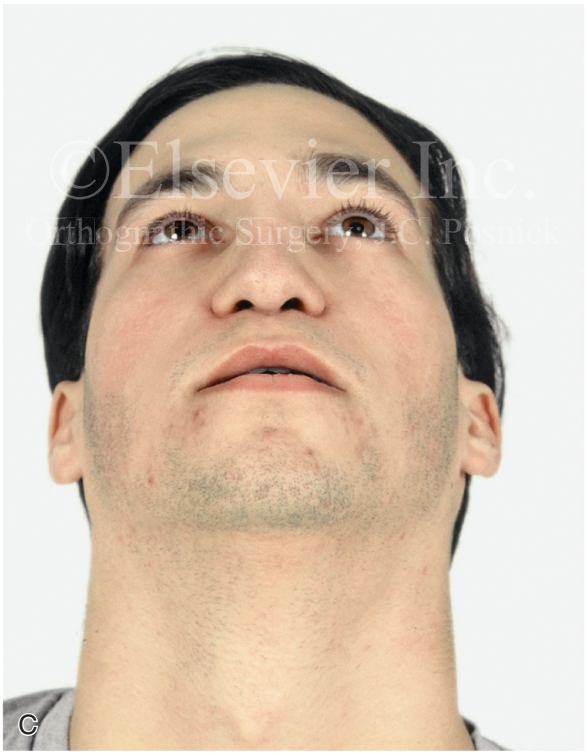


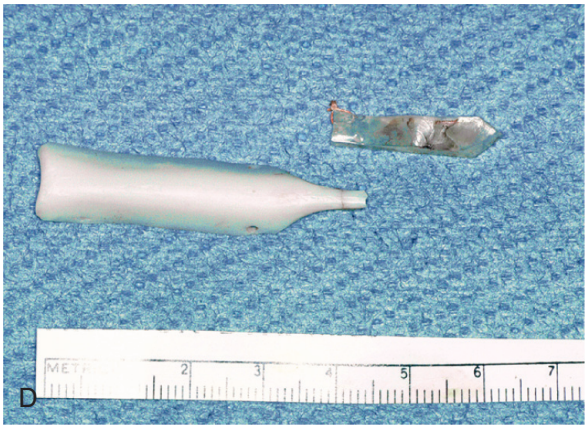
Lower lateral cartilages to be
"suture-pexed" over top of rib graft

• **Figure 38-40, cont'd C,** Through the open rhinoplasty technique, the base of the anterior maxilla is exposed. A Kirschner wire (#35 threaded) is inserted through the nasal base and the anterior maxilla. It is left to project 4 mm out of the bone. **D,** The caudal portion of the intact septal cartilage is shown in relationship to the projecting Kirschner wire. The caudal aspect of the quadrangular cartilage may be cut back to prevent buckling against the graft. **E,** The crafted rib strut graft is secured to the nasal base through the projecting Kirschner wire to ensure stability. The lower lateral cartilages will then be "suture-pexied" to each other and over the top of the caudal graft. The soft-tissue envelope of the nose is shown draped over the strut graft before the lower lateral cartilage are sutured together and over the tip.

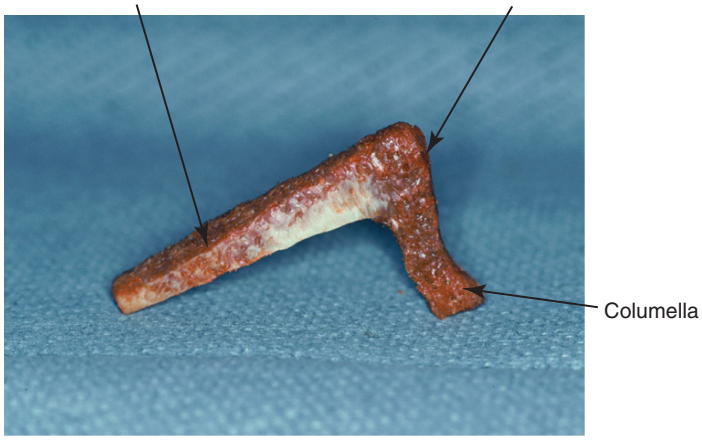
• **Figure 38-41** A 17-year-old boy who was born with Binder syndrome was referred to this surgeon with nasomaxillary deficiency. Examination confirmed cartilaginous vault and premaxillary deficiency. He underwent orthodontics that included maxillary bicuspid extractions, orthognathic surgery (Le Fort I osteotomy and genioplasty), and nasal reconstruction (corticocancellous iliac graft; see Fig. 29-1). **A,** Frontal views before and after reconstruction. **B,** Profile views before and after reconstruction.

Continued





Removed silastic implants



Crafted iliac bone graft

• **Figure 38-41, cont'd C,** Worm's-eye views before and after reconstruction. **D,** Intraoperative views of removed Silastic implants (placed by another surgeon) and crafted iliac corticocancellous bone graft placed at the time of reconstruction.

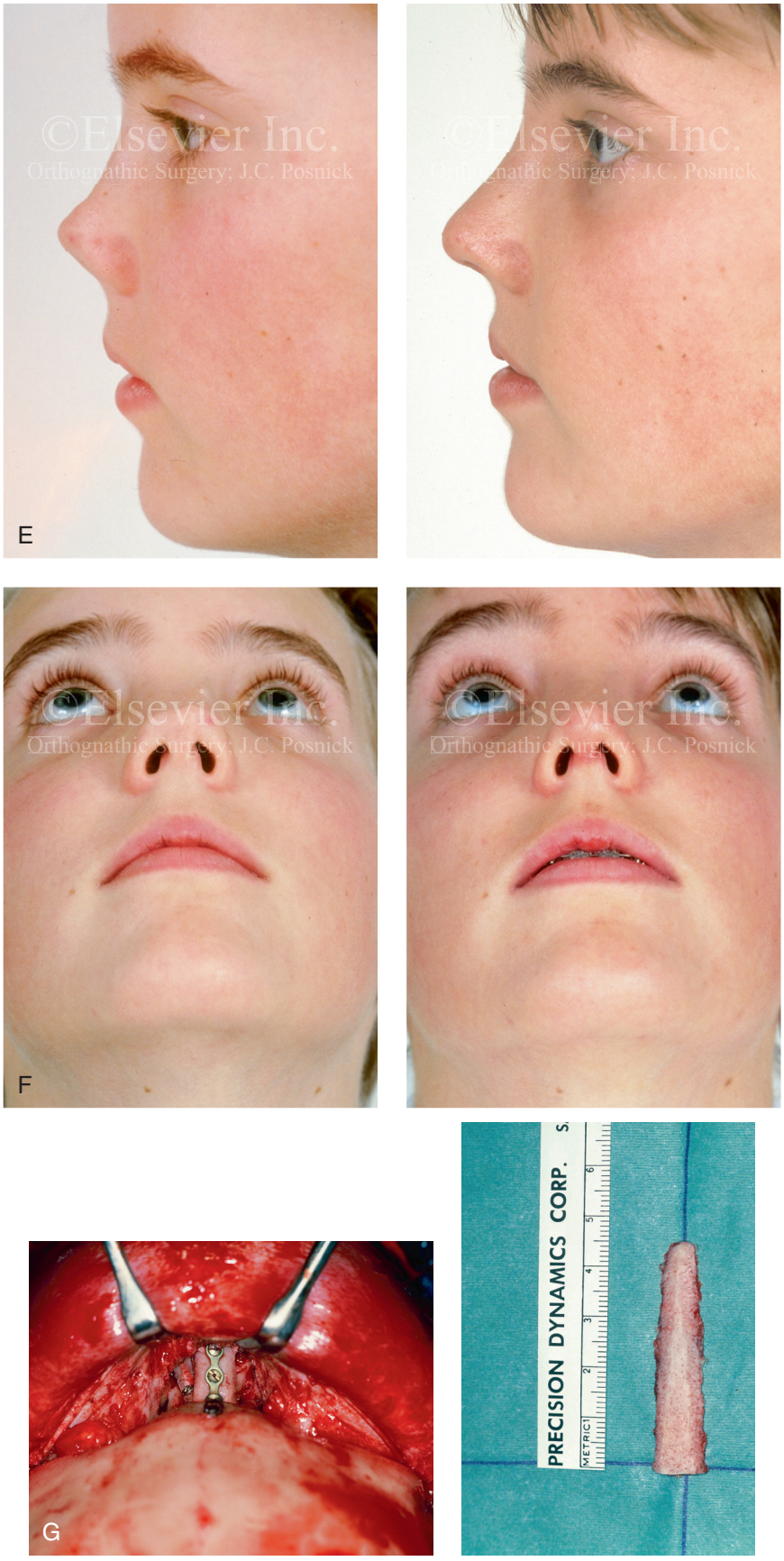
©Elsevier Inc.
Orthognathic Surgery; J.C. Posnick

• **Figure 38-42** A 15-year-old girl was born with a mild Tessier 0 and 14 craniofacial cleft. There is congenital separation of the nasal bones and the septum. The degree of orbital hypertelorism is minimal. She underwent reconstruction that included nasal osteotomies (in-fracture) and augmentation of the dorsum of the nose (from the radix to the tip) with a crafted fixed full-thickness cranial bone graft. The lower lateral cartilages were sutured over the top of the cranial graft to create the new nasal tip. Access was gained through coronal scalp and open rhinoplasty (columella splitting) incisions. **A,** Frontal views before and after reconstruction. **B,** Close-up frontal view of the nose before and after reconstruction.

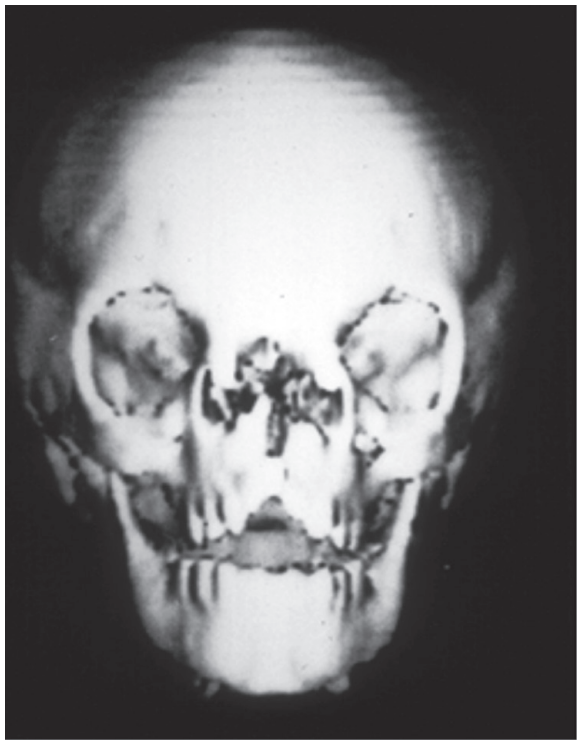
Continued

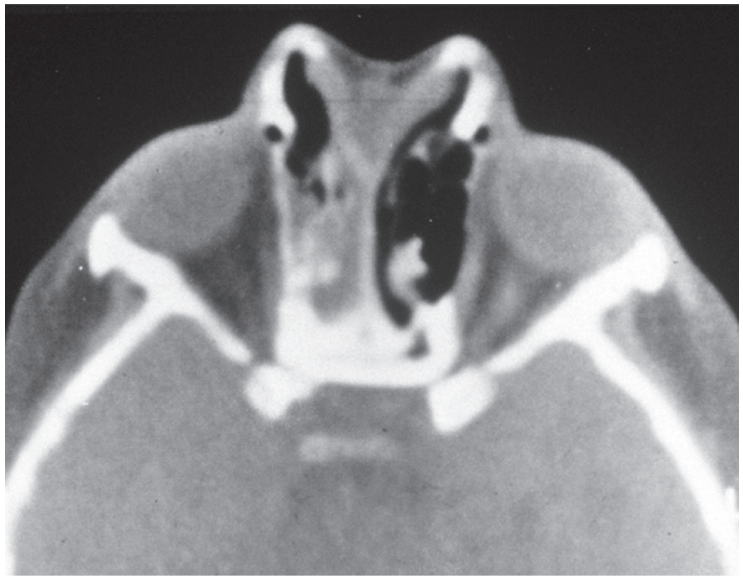
©Elsevier Inc.
Orthognathic Surgery; J.C. Posnick

• **Figure 38-42, cont'd** **C,** Oblique views before and after reconstruction. **D,** Profile views of nose before and after reconstruction.



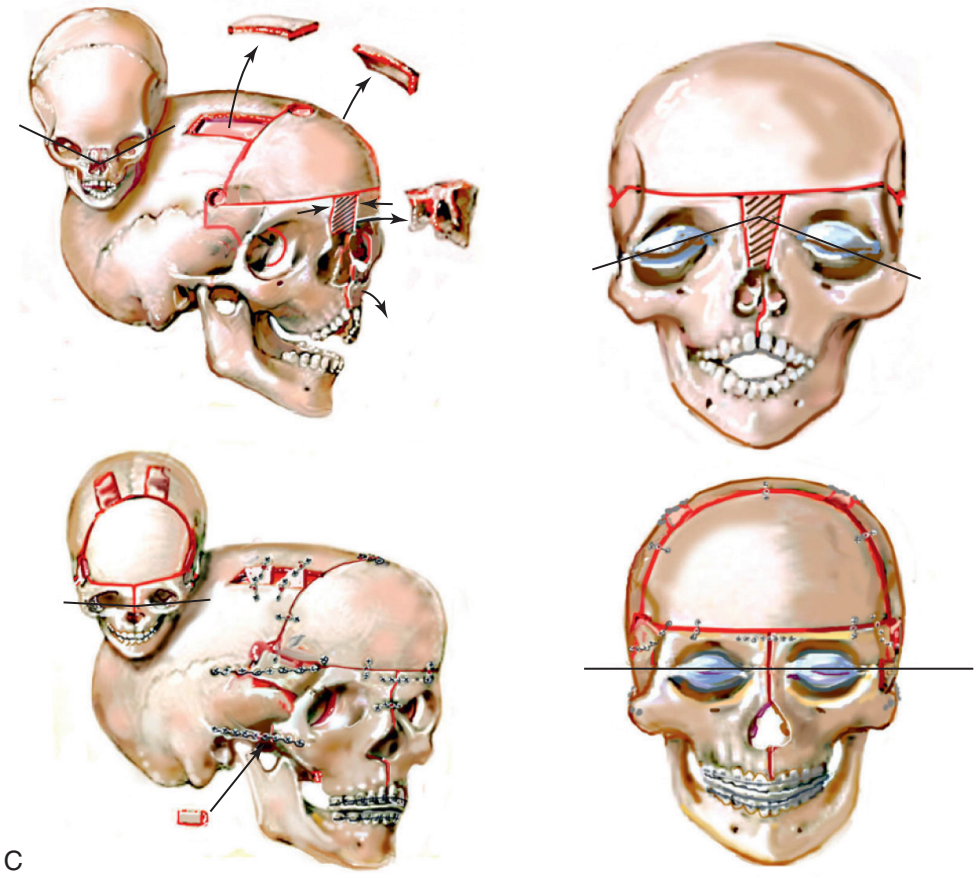
• **Figure 38-42, cont'd E,** Close-up profile view of nose before and after reconstruction. **F,** Worm's-eye view of nose before and after reconstruction. **G,** Intraoperative view of crafted full-thickness cranial graft (45 mm in length). View of the frontonasal region with plate and screw fixation to create the correct nasofrontal angle.



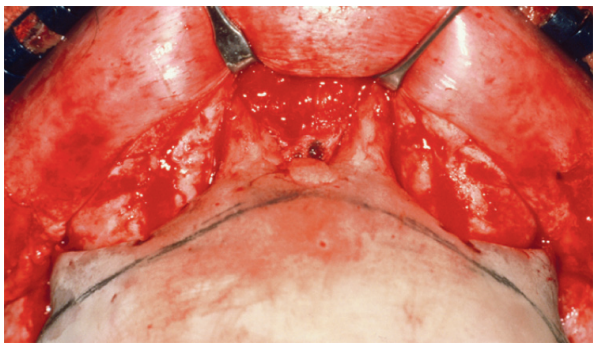


©Elsevier Inc.
Orthognathic Surgery; J.C. Posnick

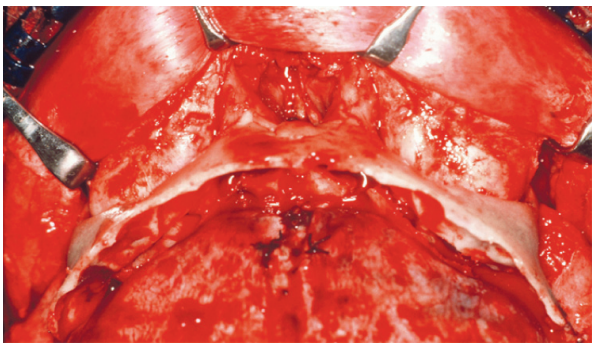
• **Figure 38-43** A child was born with a severe Tessier 0 and 14 craniofacial cleft. She underwent repair of the cleft lip when she was 1 year old; this was followed by cleft palate closure when she was 18 months old at another institution. She was referred to this surgeon when she was 4 years old for craniofacial reconstruction. She underwent a bifrontal craniotomy and facial bipartition (FB) osteotomies. The two halves of the face were then three-dimensionally repositioned for the correction of upper-face hypertelorism and to close the cleft of the nose and the upper jaw. **A,** Frontal and computed tomography scan views early after birth. **B,** Facial and computed tomography scan views after lip repair but before palate closure.



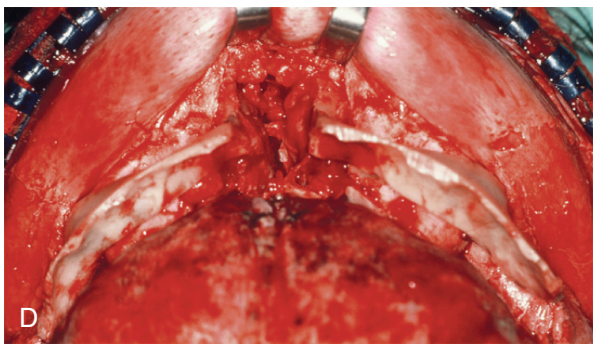
C



Prior to craniotomy

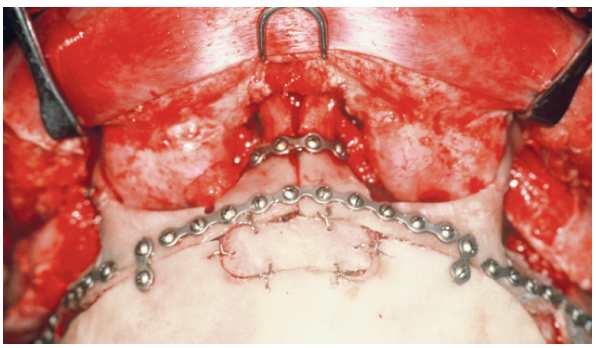


After craniotomy



D

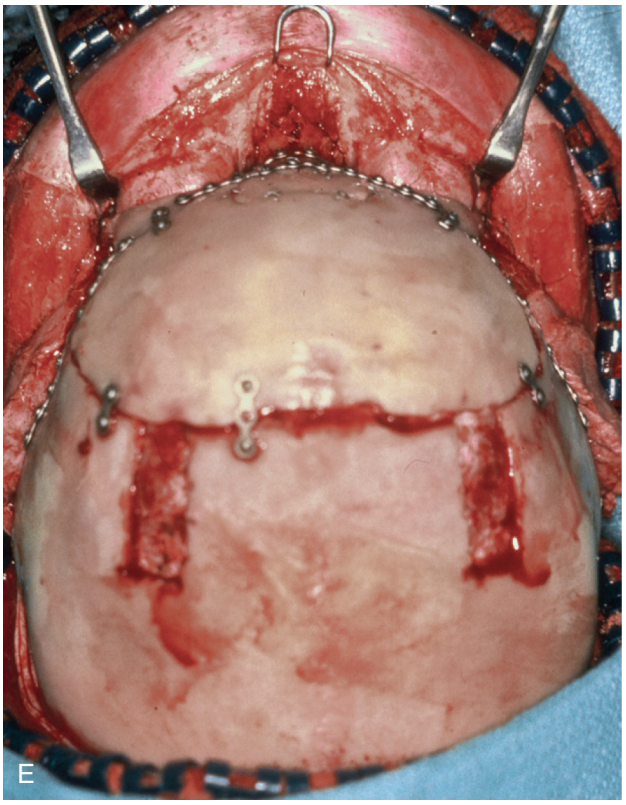
After facial bipartition and resection of central segment

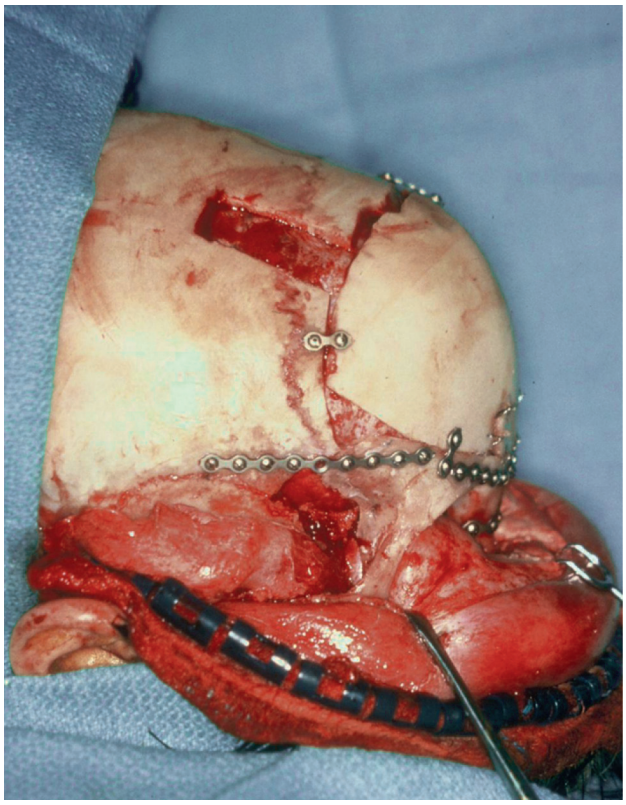


After reconstruction

• **Figure 38-43, cont'd C,** Illustrations of the craniofacial skeleton with proposed and completed facial bipartition and anterior cranial vault osteotomies and reconstruction. **D,** Intraoperative views before craniotomy; after craniotomy; after FB osteotomy and resection of the central segment of the nasofrontal region; and after FB and cranial vault reconstruction.

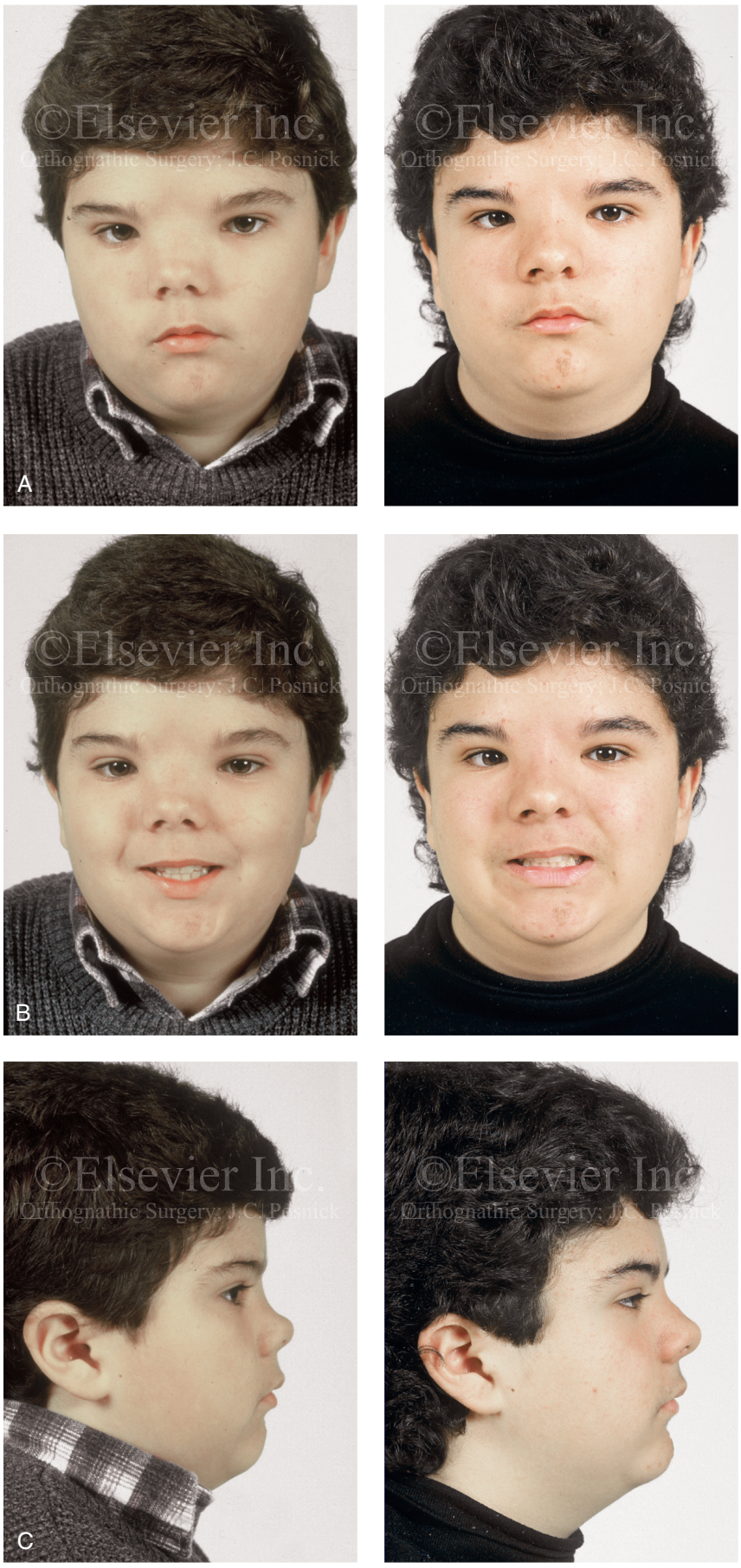
Continued



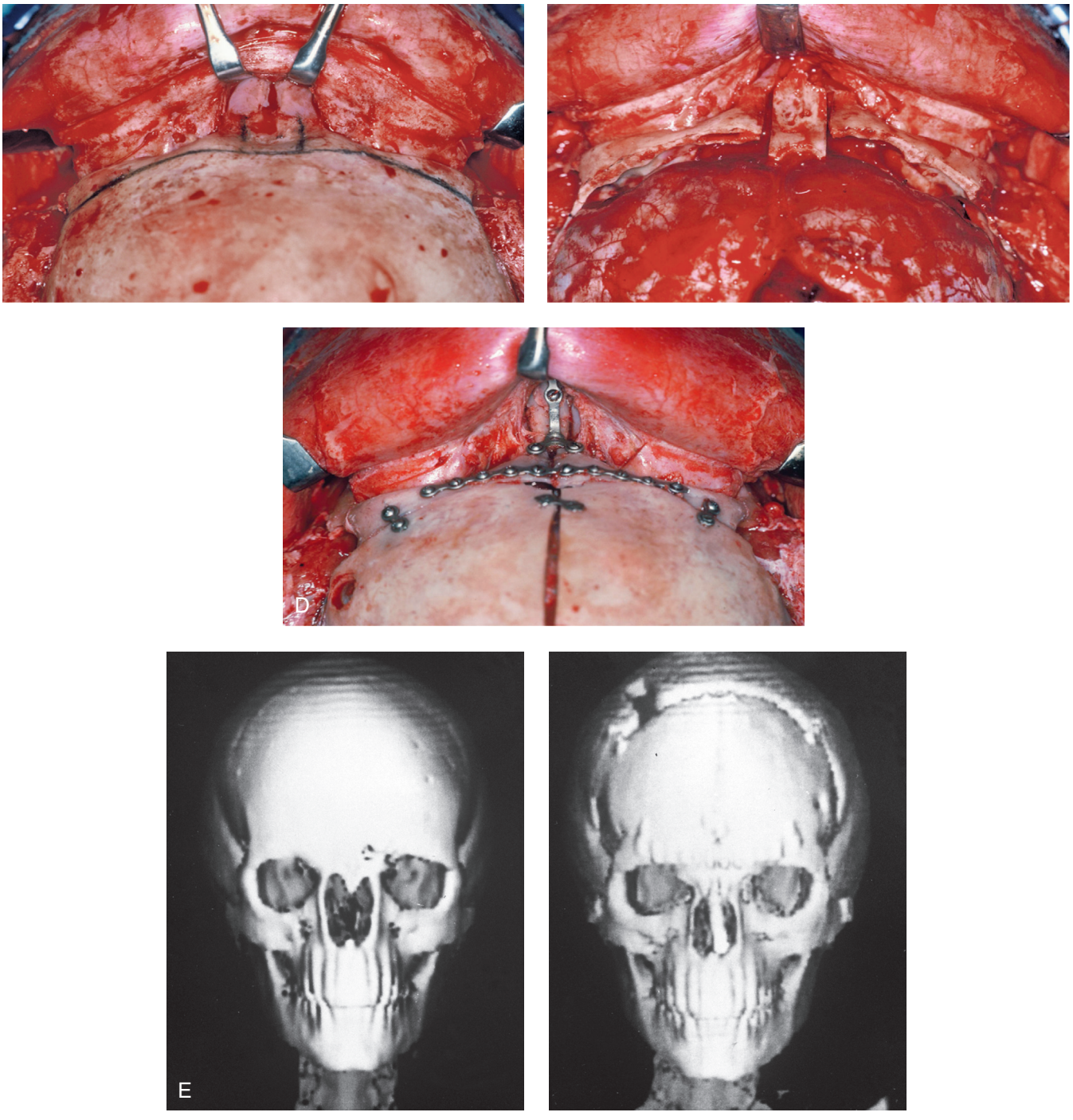


©Elsevier Inc.
Orthognathic Surgery; J.C. Posnick

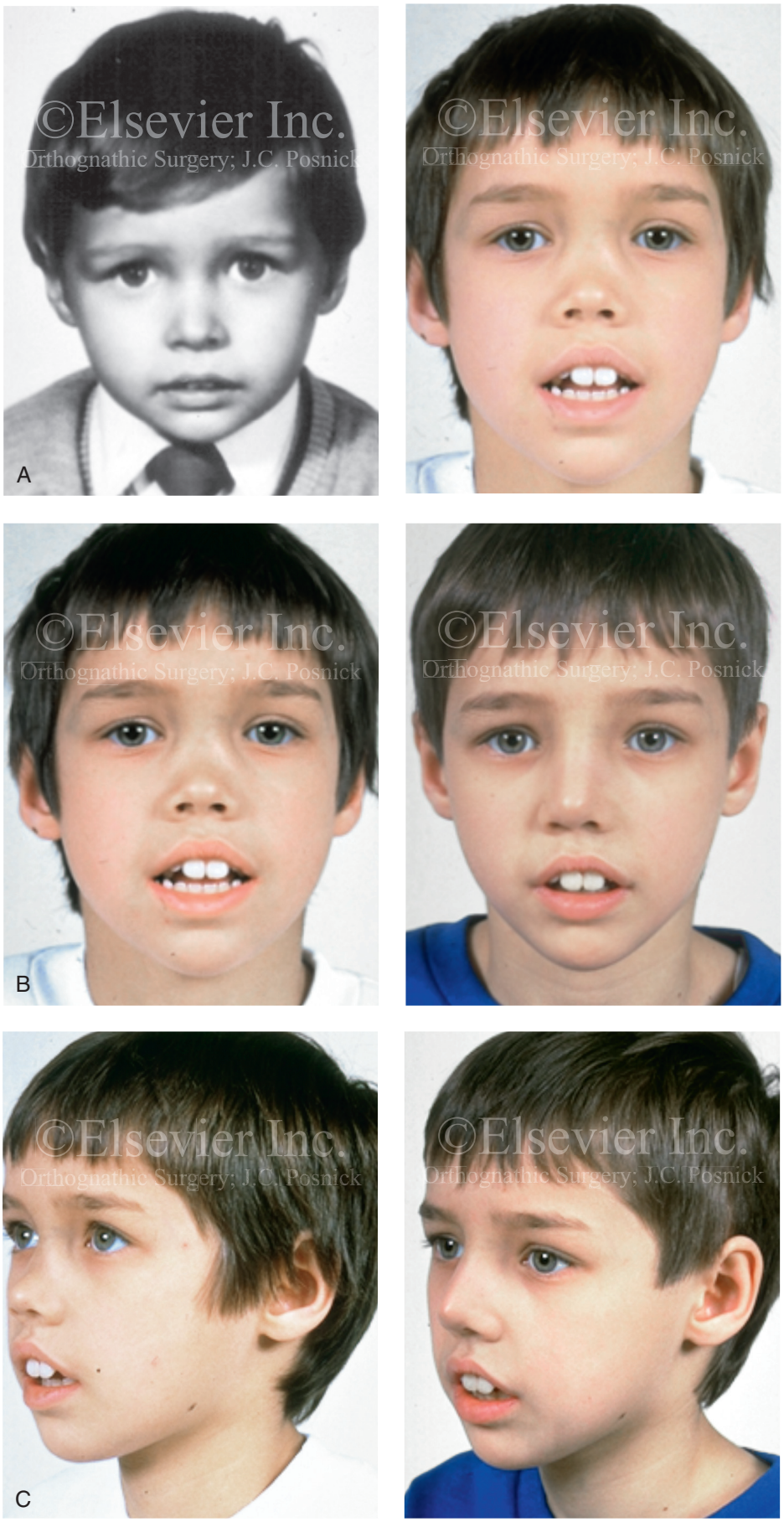
• **Figure 38-43, cont'd E,** Intraoperative views through coronal scalp incision after cranial vault and FB reconstruction. **F,** Facial views before and after reconstruction. **A, C, D, E,** *and* **F** *from Posnick JC: The role of plate and screw fixation in the treatment of craniofacial malformations. In Gruss JS, Manson PM, Yaremchuk MJ, eds:* Rigid fixation of the craniomaxillofacial skeleton, *Stoneham, Mass, 1992, Butterworth, pp 512-526.*



• **Figure 38-44** A child was born with frontonasal dysplasia that was characterized by upper-face hypertelorism and a bifid nose. This proved to be a non-progressive malformation without significant functional consequences, but it did result in facial dysmorphology. When he was 9 years old, the patient underwent anterior cranial vault and facial bipartition osteotomies through an intracranial approach to improve the upper-face hypertelorism, including nasal reconstruction. **A,** Facial views in repose before and after surgery. **B,** Facial views with smile before and after surgery. **C,** Profile views before and after surgery. *Continued*

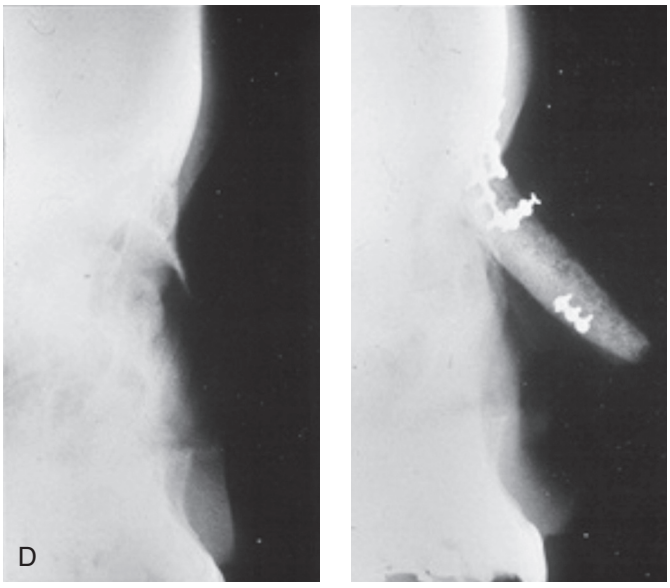


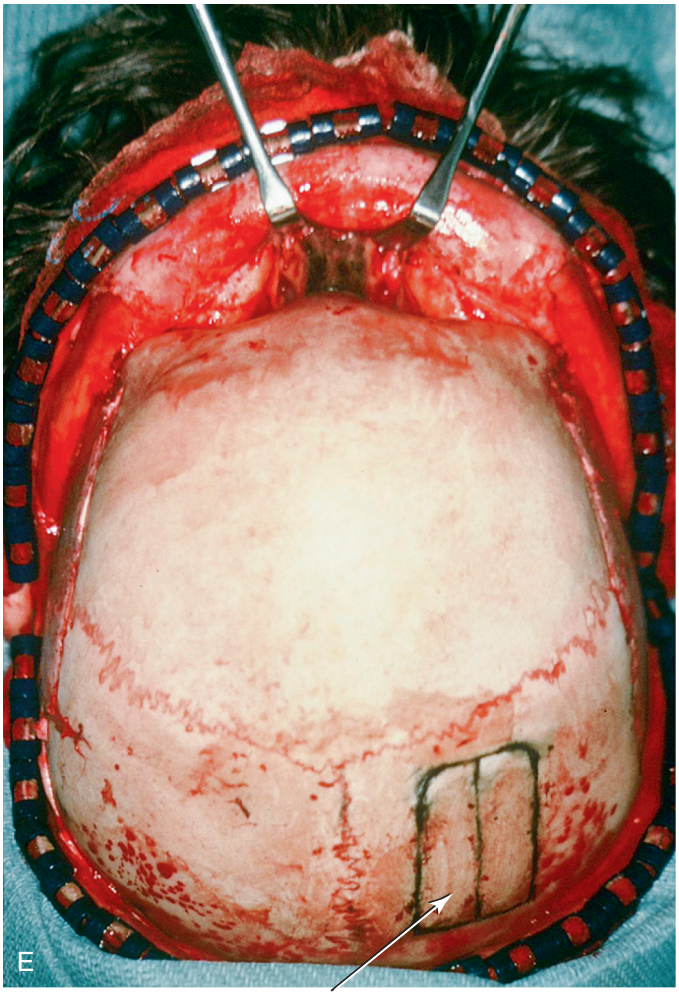
• **Figure 38-44, cont'd** **D,** Intraoperative views of the upper orbits and the anterior cranial vault before and after reconstruction. The views indicate the planned craniotomy, the osteotomies, and the naso–fronto–ethmoid ostectomy; after craniotomy, the facial bipartition and the mid-nasal ostectomy; and after the correction of the upper and midface hypertelorism, including a full-thickness cranial bone graft that is fixed in place from the radix to the tip of the nose. After stabilization of the nasal bone graft, the lower lateral cartilages were pexied over the tip of the graft through the exposure provided by the columella skin incision. **E,** Three-dimensional craniofacial computed tomography scan views before and after reconstruction.

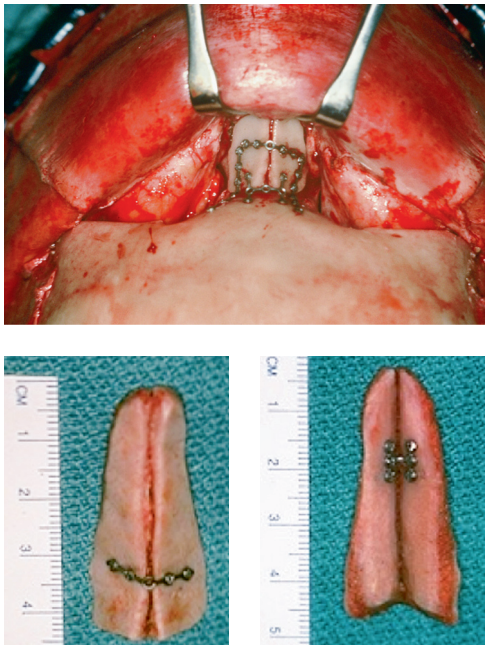


• **Figure 38-45** A 4-year-old boy fell and hit the bridge of the nose on a hard table. Over the next several years, a saddle deformity developed. He arrived to this surgeon for evaluation when he was 8 years old and then underwent reconstruction; this was carried out through coronal scalp and open rhinoplasty (columella splitting) incisions. The reconstruction included nasal osteotomies (in-fracture); the placement of fixed full-thickness cranial grafts; and the suture pexy of the lower lateral cartilages over the top of the graft (see Fig. 35-2). **A,** Frontal view at 3 years of age just before injury and then at 8 years of age just before surgery. **B,** Frontal views before and 6 months after reconstruction. **C,** Oblique views before and 6 months after reconstruction.

Continued







• **Figure 38-45, cont'd** **D,** Lateral skull radiographs of the nose before and 6 months after reconstruction. **E,** Intraoperative views through the coronal scalp incision demonstrating the frontonasal region and the proposed right cranial vault donor site. Crafted full-thickness cranial bone grafts are shown before placement for nasal reconstruction. The close-up view of the frontonasal region shows the full-thickness cranial bone grafts in place and stabilized with microplates and screws.

• **Figure 38-45, cont'd F,** Close-up lateral views of the nose just before surgery and at 6 months and then 16 years after reconstruction.

Surgical Approach to the Combined Collapse of the Osseous and Cartilaginous Vaults

Surgical attempts to correct the postseptoplasty pattern of saddle-nose deformity have been reported for at least the past century. McDowell and colleagues discussed the multiple heterogeneous implant methods used during his time, which included inorganic materials (e.g., gold, silver, tin, thallium, Vitallium, plastic, silicone) and organic implants (e.g., ivory, cartilage, bone, fibrin, fascia).[138] Fomon and colleagues described the use of autogenous materials for the restoration of nasal contour (i.e., fat, dermis, cartilage, and bone).[64] Historically, local transposition flaps were often recommended to augment the internal (mucosa) lining or the external (skin) cover. By the 1950s, most clinicians agreed that individuals who presented with marked depressions of the cartilaginous and bony dorsum required firm, deep, layer grafts or implants. Surgical reconstruction is now thought to require grafting from the radix to the tip and often from the tip down to the base of the maxilla. Today, autogenous tissue is generally used, but donor-site preference, fixation techniques, and how the graft holds up at the recipient site remain important questions.

Posttraumatic saddle-nose deformities of the bony vault often result from displaced fractures with secondary collapse after incomplete reduction (see Fig. 38-45). In addition, a crush injury to the nose during early childhood will result in impaired growth and progressive deformities. Another subgroup of patients with saddle-nose deformity presents during adolescence with congenital nasal and dentofacial anomalies (see Fig. 38-41). Whatever the cause, the end result is a flattened, splayed, and sometimes asymmetric osseous vault in combination with a depressed cartilaginous vault. The trauma event or the congenital anomaly also leads to ULC and LLC displacement with poor tip projection, flaring of the alae, widening of the domes, and rounding of the anterior nares. The extensive traumatic or congenital anomalies that caused the saddling of the cartilaginous and osseous vault will also result in mechanical nasal airway obstruction. When this occurs during childhood, the forced oral respiratory pattern with a constant open-mouth posture often results in a long face growth pattern over the ensuing years (see Chapters 4 and 21).

The correction of a severe saddle-nose deformity of the bony and cartilaginous vaults requires the insertion of a deep dorsal graft from the radix to the tip that is ideally constructed from either autogenous cartilage or autogenous bone (see Fig. 35-45). Rib cartilage has advantages over bone in that it restores flexibility to the cartilaginous vault and tip, it is often considered easier to harvest, and it is less prone to resorption. It has the disadvantages of having a tendency to warp, not adhering to the underlying osseous vault, and not replacing like for like over the osseous vault. Autogenous bone (e.g., calvarial, iliac, rib) has the advantages of ease of crafting, adherence to the underlying basal bone when properly immobilized (fixed), and replacing like for like tissue over the osseous vault. It has the disadvantages of requiring more complex donor site harvesting, having a tendency for partial resorption, and resulting in a firm nasal tip that is prone to fracture. Whether autogenous cartilage or bone is used to correct the osseous and cartilaginous vaults, reliable reconstruction requires satisfactory skin

cover, internal nasal lining, and LLCs that can be pexied over the graft to form the superficial aspect of the new nasal tip.

Autogenous graft options to consider include the following:

1. *Rib costochondral graft (radix to tip).* This is combined with a separate rib cartilage caudal strut graft that extends from the base of the maxilla (stabilized with a K-wire). It is then secured with sutures to the dorsal costochondral graft at the tip. The LLCs are sutured over the grafts to form the height of the new nasal tip.
2. *Rib cartilage graft (radix to tip).* A no. 35 K-wire may be inserted through the dorsal strut graft to prevent warping. The dorsal graft is combined with a separate rib cartilage caudal strut graft that extends from the base of the maxilla (stabilized with a K-wire). The caudal graft is then secured with sutures to the dorsal strut rib cartilage graft at the tip. The LLCs are sutured over the grafts to form the height of the new nasal tip.
3. *Calvarial graft (radix to tip).* The crafted calvarial bone graft is firmly secured with K-wires or titanium plates and screws to the underlying nasal bones. The LLCs are sutured over the graft to form the height of the new nasal tip.
4. *Iliac graft (radix to tip).* The crafted iliac graft is firmly secured with a K-wire or titanium plates and screws to the underlying nasal bones. The LLCs are sutured over the graft to form the height of the new nasal tip. Extension of the bone graft into an "L" shape as a caudal strut may be done to limit the fracture and resorption of the dorsal strut.

Cocaine-Related and Autoimmune and Iatrogenic Saddle-Nose Deformities

The hallmark of most *cocaine-related, autoimmune, and iatrogenic saddle-nose deformities* is significant *septal perforations* that result in *deficiency (depression) of the cartilaginous vault,* generally with an intact osseous vault (Fig. 38-46).

Cocaine is an alkaloid that was originally used by South American Indians as a stimulant. It was introduced to medicine by Koller in the 1880s as a local anesthetic.[119] The recreational use of cocaine is a common form of drug abuse throughout the world. At least 30 million Americans are estimated to have used cocaine at least once (i.e., 20% of the adult population), and 6 million use it with some regularity (i.e., once a month or more).[56] It is estimated that approximately 20% of users become addicted. The most commonly used route of administration for the drug is intranasal inhalation or "snorting."[58] Cocaine's intense local vasoconstrictive effects and mucosal irritation from substances with which it is "cut" (e.g., talc, mannitol, lactose, amphetamines, borax, plaster of Paris) may lead to inflammation and ulceration of the nasal mucosa.[66,227] Permanently exposed septal cartilage in combination with bacterial overgrowth will lead to osteochondritis and further necrosis and perforations, thereby resulting in the loss of structural support and the collapse of the nasal dorsum.[19,246] Less common but reported complications in the sinonasal region after cocaine abuse include oronasal fistulas, palatal destruction, and even complete midface necrosis.[216,224,228] All reported cases of palatal perforations from cocaine insufflation initially presented with a limited perforation of the nasal septum followed by progressive destruction of the associated structures.[7,14,65,71,87,134,212,242]

The adverse effects of cocaine on the nasal structures were first systematically reported by Owens in 1912.[167,170] He described a range of cocaine-induced deformities, from a pinhole perforation to progressive degrees of mucosal ulceration, destruction of the septal cartilage, and, in extreme cases, the destruction of the nasal and maxillary bones. When a nasal septal perforation is small and anterior, it can cause a disconcerting whistle during breathing. When the hole is larger and more posterior, as it is in most individuals who present for the surgical evaluation of a saddle nose, it will have destroyed most of the septal cartilage.[145] Businco and colleagues reported on a series of 104 patients who habitually inhaled cocaine more than 10 times per month.[22] They were observed for at least 2 years. Among them, 11 patient (11%) had nasal septal perforation. Of these 11 patients, 8 had nasal septal perforation of just the quadrangular cartilage; in the other 3, the perforation also involved the bony tract (i.e., the vomer-perpendicular ethmoidal lamina).

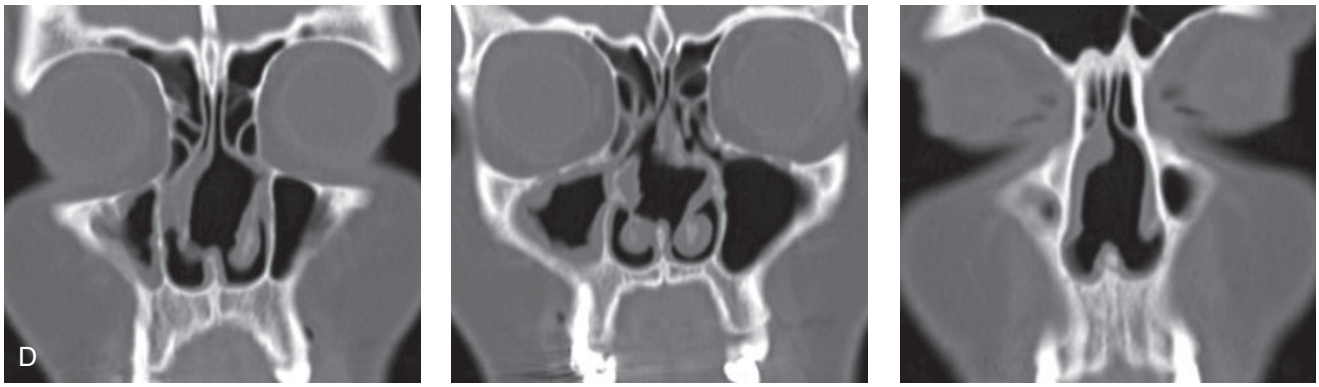
Over the years, the management of cocaine-induced septal perforations has included the placement of "buttons" to block the defect; the use of local nasal mucosal flaps; the use of regional nasolabial flaps; and the use of distant microvascular repairs.[28,56,120,122,132,140,154,155,158,171,204] The flap options, even when they effectively close the perforation, typically obstruct normal function of the airway as a result of their bulk. This author in agreement with most clinicians that, until a safer and more reliable vascularized and thin flap is found and proven to be effective, it is generally best to maintain good nasal hygiene and not attempt perforation closure.[143]

Similar septal perforations and secondary saddle deformities of the cartilaginous vault are sometimes seen after overly aggressive surgical submucosal resections that have been carried out to relieve nasal obstruction or after postoperative or posttraumatic hematoma or infection (see Fig. 38-46).

The same pattern of saddle-nose deformity is frequently observed in patients with *Wegener granulomatosis*.[8,61,159] This is a rare multisystem autoimmune disease of unknown etiology. Its hallmark features include necrotizing granulomatosis, inflammation, and pauci-immune vasculitis in small and medium-sized blood vessels. In 1897, Peter McBride gave the first written description of a patient

• **Figure 38-46** A middle-aged woman of Arabic descent with a postsurgical (iatrogenic) saddle-nose deformity. Two years earlier, she underwent an aggressive septoplasty procedure at another institution. This resulted in a large septal perforation with the collapse of the cartilaginous vault. She arrived for evaluation and then underwent nasal reconstruction that included rib cartilage grafting with caudal and dorsal struts as described in text. **A,** Frontal views before and after reconstruction. **B,** Oblique views before and after reconstruction. **C,** Profile views before and after reconstruction.

Continued



• **Figure 38-46, cont'd D,** Computed tomography sinus scan views (coronal cut) that confirm the extent of the septal deficits before reconstruction.

with this condition.[145] Thirty-four years later, Klinger described a 70-year-old physician with joint symptoms, proptosis, widespread upper respiratory tract inflammation that led to saddle-nose deformity, glomerulonephritis, and pulmonary lesions.[145] In 1936, Wegener described three patients with similar clinical features and published his findings.[145] The prevalence of Wegener granulomatosis in the United States is estimated to be 3 cases per 100,000 people. Chronic sinusitis is the most common initial complaint, with approximately 60% to 70% of cases involving a failure to respond to conventional treatment. The Wegener granulomatosis saddle-nose deformity is caused by the perforation of the posterior aspects of the quadrangular cartilage with a loss of dorsal support. When the autoimmune disease is in remission and the need for medications that affect wound healing is no longer present, then reconstruction of the collapsed nasal cartilaginous vault can go forward as described in the following section.

Surgical Approach to the Collapsed Cartilaginous Vault

Before the reconstruction of a collapsed cartilaginous vault, the surgeon must assess the presenting nasal anatomy, including all defects and their effects on function.* More than half a century ago, Millard pointed out that, in these cases—despite the apparent overwhelming "destruction of the nose"—the covering skin is generally intact and unscarred, and it can be salvaged to serve as the final nasal cover.[145] It is also recognized that, in most cases, the nasal bones, the pyriform rims, and the floor of the nose are unaffected. The same is true for the LLCs and the ULCs. A full-thickness nasal septal defect that involves much of the quadrangular cartilage and overlying mucosa and that extends inferiorly toward the nasal floor and posteriorly toward the vomer is typical (see Fig. 38-46, *D*).

*References 7, 28, 56, 61, 63, 64, 71, 95, 96, 119, 120, 122, 132, 154, 175, 180, 221, 230, 247, 251

As part of the reconstruction, the bony dorsum may benefit from surgical reduction if there is a true "hump"; however, augmentation is rarely indicated. In general, nasal osteotomies are avoided unless they are absolutely required (i.e., a crooked bony vault as a result of previous nasal fracture or surgery or excessive widening of the bridge caused by hereditary tendencies). The ULCs and the LLCs, although flattened, rarely require replacement. During the operation, these cartilages are dissected from the overlying skin cover via an open approach and then repositioned as needed. The cephalic portions of the LLC may be conservatively excised. The middle crura are separated down to the base of the maxilla and the anterior nasal spine region but without continuing the dissection more posterior, which would expose the septal perforation.

Typically, a rib cartilage graft is harvested and crafted into two struts to be used for the reconstruction (see Chapter 18) (Video 10). The first is a *caudal strut* of approximately 25 to 30 mm in length that extends from the base of maxilla to the new nasal tip (see Fig. 38-40). The second is a *dorsal strut* of approximately 35 to 40 mm in length that is notched and inset deep to (only ≈2 mm) and then flush with the nasal bones before extending to the tip. The grafts are secured to each other at their points of insertion and then to each other where they join at the tip. The LLCs are sutured over the top of the grafts to form the most superficial aspect of the nasal tip. The soft-tissue envelope is then redraped over the new cartilaginous vault and tip.

Reconstruction of a Collapsed Nasal Cartilaginous Vault: Step-by-Step Approach

- General anesthesia is given through orotracheal intubation. The endotracheal tube is secured to the chin region with tape.
- Local anesthesia (Xylocaine 1% with 1:100,000 epinephrine) is injected for hemostasis below the soft-tissue envelope of the nose.
- An open technique is used. Stair-step columella (skin) and bilateral marginal (intranasal) incisions are completed.

- The overlying cutaneous soft-tissue envelope is elevated up the columella, superficial to the LLCs and ULCs, subperiosteal over the nasal bones to the radix, and laterally toward the maxillary process on each side. The fully elevated soft-tissue envelope will later be advanced caudally over the reconstructed cartilaginous vault and nasal tip at wound closure.
- The elevated mucosa lining is protected throughout the dissection without perforation into the nasal cavity.
- Dissection also extends under the nasal bones (≈5 mm) for later insertion (i.e., tongue and groove) of the dorsal strut graft.
- If a dorsal bony "hump" requires reduction, this is conservatively completed with rasps.
- The medial crura are separated from each other via scissors dissection down to the base of the maxilla and the anterior nasal spine. This is accomplished without perforation through the mucosal lining into the nasal cavity.
- The deficient dorsal and caudal portions of the quadrangular cartilage are reconstructed with rib cartilage that has been crafted into two separate struts. After the struts are secured at their points of origin, they are sutured together to form the new tip and to serve as a replacement for the deficient cartilaginous vault.
- The caudal strut graft extends from the base of the maxilla (i.e., the anterior nasal spine region) and then up to the new nasal tip; this graft is approximately 25 to 30 mm in length. It is secured to the base of the maxilla with a no. 35 threaded K-wire that has been drilled into the anterior nasal spine region for a depth of approximately 5 mm. If there is perforation through the palatal mucosa, the K-wire is backed out. The K-wire is cut, with an extension of several millimeters left above the exposed base of the maxilla. The caudal strut graft is then pierced into the K-wire to prevent the movement of the graft during the healing process.
- The dorsal strut graft is notched via a tongue and groove to extend several millimeters deep to the nasal bones and then flush with the osseous surface that extends caudal to the new nasal tip; the graft is approximately 35 to 40 mm in length. It is positioned either in between or on top of the ULCs and then sutured in place to limit movement during healing.
- The dorsal strut graft meets the caudal strut graft at the tip. The two grafts are further crafted using a knife (no. 15 blade) to form the new nasal tip. They are secured together with several interrupted non-resorbable sutures (4-0 Prolene).
- The LLCs, having been trimmed of any cephalic excess, are sutured together and then over the strut grafts to form the most superficial aspect of the nasal tip (4-0 Prolene).
- One of the challenges is the management of any scar contracture of the mucosa lining, which may limit the caudal and anterior advancement of the LLC for suturing over the grafts. This is managed by the following:
  - The dissection and release of the nasal lining, but without perforation
  - If necessary, the reduction of the height of the strut grafts (i.e., the new nasal tip) to accommodate any intrinsic soft-tissue limitations
- The dorsal and caudal strut grafts can be reinforced with an internal no. 35 threaded K-wire to limit the risk of warping. The decision to do so must be made before inset and before the grafts are secured in place.
- The stair-step columella (skin) incision is closed with one deep layer stitch (5-0 Vicryl). The closure of the skin layer is accomplished (6-0 nylon), and the rim incisions are closed with resorbable sutures (5-0 chromic).
- Antibiotic ointment (Bactroban) is injected into the nasal cavity. Gentle packing in each nostril (Telfa) is carried out to decrease intranasal swelling and bleeding.
- Tape is placed over the dorsal skin (Steri-Strips).
- A custom splint (Aquaplast) is conformed over the dorsum.
- The packs are removed after 48 hours.
- The dorsal splint is removed after 5 to 7 days.

The reconstructive outcomes for cocaine-related, autoimmune, and iatrogenic cartilaginous vault collapse may not be perfect, but they should provide a relatively normal nasal form, often with an improved airway. The presenting septal perforations will remain. The dorsal and caudal support for the external nose that was deficient should be restored without the need for replacement of the soft-tissue envelope. The goal is for the individual to become socially presentable without excessive attention being drawn to the previous nasal deformity. This should be possible with a single-stage reconstruction without extensive donor-site scarring or disability in most patients.

Conclusions

Successful rhinoplasty is accomplished after the obtaining of an accurate patient history; the performance of a thorough head and neck examination that includes the intranasal cavity; and the completion of a facial aesthetic analysis. This is followed by engaging in a frank discussion with the patient and the family about the treatment objectives; the quality of the individual's skeletal and soft tissues; what is technically possible; and an appreciation of the individual's overall facial proportions. When combining sound clinical judgment, technical expertise, and safe conditions, the surgeon may perform precise functional and aesthetic rhinoplasty with a predictable level of patient satisfaction. Relieving nasal obstruction to improve breathing and achieving an aesthetically pleasing nose that preserves the individual's uniqueness through a combination of reduction and augmentation techniques remain the clear objectives.

Patient Education Materials

Rhinoplasty (Reshaping the Nose) Surgery

A rhinoplasty surgical procedure may be designed to improve the appearance of the nose, to correct nasal breathing, or both. The operation usually requires about 1.5 hours, and it is performed on an outpatient basis with the patient under anesthesia. An additional several hours are spent in recovery before the patient is discharged home. A customized splint is applied to the outside of the nose, and packing material is often placed inside. There will be some discomfort for which pain medication is prescribed. Antibiotics will be given just before surgery and continued at least until the packing is removed.

Discoloration and swelling occur in the cheeks and the eyelid region. The swelling gradually disappears, although inadvertent trauma or sunburn may cause it to persist a great deal longer. The tip of the nose is usually numb for several months after surgery, but this too improves with time.

The final results after rhinoplasty are not fully apparent for 6 to 12 months, at which point the nasal swelling has subsided. For some patients, after rhinoplasty, there are dramatic changes; for others, the improvements may be more subtle. Nasal surgery is an art and a science, but it is not magic. Your surgeon will try to preserve the positive characteristics that make your nose unique while modifying the aspects that distract from your appearance. The goal will be to modify specific undesirable characteristics to achieve a more harmonious balance of nasal and facial features given your specific anatomic limitations. In general, aesthetic judgments will be conservative. When it comes to rhinoplasty, it is better to remove less than more.

INSTRUCTIONS FOR RHINOPLASTY SURGERY

The purpose of these instructions is to help you to prepare for and then recover from your operation with as little discomfort as possible and to minimize the risk of complications.

Preoperative Instructions

1. For at least 2 weeks before surgery, do not take any aspirin, aspiring-containing products, or aspirin-like products (e.g., ibuprofen). Do not take any medication that may increase bleeding during surgery or bruising afterward.
2. No smoking or nicotine in your bloodstream for at least 2 weeks before and 2 weeks after surgery.
3. Do not eat or drink anything after midnight the night before surgery, not even a sip of water.
4. Starting 2 days before surgery, we request that you shampoo your hair and shower at least once per day. The morning of surgery, you may also shower and shampoo again.
5. Do not apply makeup the morning of surgery, and leave your jewelry at home.
6. Make arrangements for someone to accompany you home from the hospital the day of surgery, because you will not be able to drive yourself.

Postoperative Instructions

1. After discharge from the hospital, eat lightly (mostly liquids). During the first week, avoid eating anything that is difficult for you to chew.
2. Unfortunately, nausea and sometimes vomiting during the first 12 to 24 hours after surgery is not uncommon. If you develop nausea or vomiting, take only clear fluids for the next 12 hours. Resume a normal diet gradually, as tolerated.
3. Apply cold compress intermittently over your eyelids during the first 24 to 36 hours after surgery. By 36 hours after surgery, the maximum facial swelling will have occurred. Cold compresses are of limited value after this time, but they may be applied for comfort, if desired.
4. For the first week after surgery, sleep primarily on your back, with your head elevated on two pillows.
5. As before surgery, do not take aspirin or aspirin-like products (e.g., ibuprofen) for 2 weeks after surgery. You may take Tylenol (acetaminophen) as you wish.
6. For comfort, you may wish to use Vaseline petroleum jelly or any over-the-counter antibiotic ointment around the nostrils to keep them from drying out. Do not insert anything deep inside the nostril. Use and change the dressing beneath your nose as needed.
7. You may shower from the neck down the day after surgery. You should also wash your face and hair, but do not get the nasal splint wet. After the splint has been removed, you may shower and shampoo your hair and face in a more regular way.

8. Take your usual prescription medication as directed and approved by your surgeon beginning the day after surgery.
9. Get lots of rest and don't do anything that requires more than limited physical exertion during the first week after surgery. You may then gradually increase your activity level, but no sports or gym activities may be performed during the next 4 to 6 weeks as per discussion with your surgeon.
10. You may take the prescribed pain medication as directed on the label. Take the antibiotic medications as directed until the supply is exhausted.
11. When you sneeze, try to do so through your mouth. Do not blow your nose for 2 weeks, and only use nasal sprays that have been approved by your surgeon.
12. Do not consume beverages that contain alcohol or drive while taking prescription pain medications.
13. If you wear contact lenses, you may be comfortable enough to resume lens wearing within 48 to 72 hours. In addition, eyeglasses may be adjusted to rest on or just above the splint. After the removal of the splint (i.e., 5 to 7 days after surgery), you should avoid letting your glasses press heavily on your nose for at least 6 weeks.
14. Make an appointment to see your surgeon on the specified day by calling the office.

Remember the following:

1. Your skin will be noticeably discolored around the eyes and swollen for about 2 weeks. The discoloration will move down your face and neck. The extent of the swelling and discoloration will vary on the two sides of the face. When the nasal packs are removed (approximately 2 days after surgery), further intranasal swelling will occur and create ongoing difficulty with breathing through the nose. This will gradually resolve over several weeks.
2. The shape of your nose will go through its biggest changes during the initial 3 to 6 weeks after surgery, but it will take about 6 months to 1 year before the final swelling subsides. This is especially true for the nasal tip region.
3. Because of the surgery and the need to cover the nasal soft tissues with a splint during the first week, expect peeling of the skin and blocked pores. You may also go through a short period of acne breakouts. Just wash the affected area gently. The condition of your skin will improve over the course of 5 to 10 days and should gradually return to normal.

Jeffrey C. Posnick, DMD, MD
Director, Posnick Center for Facial Plastic Surgery

Consent for Rhinoplasty Surgery

_____ Initial here

INSTRUCTIONS

This is a document that has been prepared by your surgeon to help educate and inform you about your rhinoplasty surgery and its risks as well as alternative treatment options. It is important that you read this document carefully and completely. Please initial each section on each page to indicate that you have read and understand it. When all of your questions are answered, please also sign the consent for surgery on the last page as proposed by your surgeon.

_____ Initial here

INTRODUCTION

Rhinoplasty (surgery of the nose) is an operation that is frequently performed to produce changes in the appearance, structure, and function of the nose. Rhinoplasty maybe carried out to alter the dorsum (height or width) of the nose; to change the shape of the nose's tip; or to change the angle between the nose and the upper lip. This operation can also help to correct birth defects or nasal injuries and to help relieve breathing problems.

There is not a universal type of rhinoplasty surgery that will meet the needs of every patient. The surgery is customized in accordance with your needs and any specific anatomic limitations. Incisions are generally made within the nose and also into the skin of the nose. Internal nasal surgery to improve breathing can often be performed at the same time.

Continued

Nasal surgery is an art and a science, but it is not magic. Your surgeon must work with your specific tissues (e.g., skin, bone, cartilage, mucosa lining) in an attempt to improve nasal appearance and function. The results of the operation depend on the characteristics and qualities of your specific tissues and on complicating factors such as scarring, tissue deficiencies, and irregularities from previous surgery, injuries, or nature.

No nasal surgery is totally perfect. If you believe that small imperfections in the surgical result will prevent you from appreciating the positives, you should not proceed with rhinoplasty. Every small imperfection cannot be corrected or reoperated on; this approach would lead likely to an "operated" appearance that is unfavorable. Your surgeon will do his or her best to communicate what the planned procedure can and cannot do for you. You are encouraged to ask questions and to openly communicate your concerns.

To reshape a nose, cartilage or other types of grafts may be useful to provide structure and support. Usually these grafts are not visible, but sometimes they can be seen as small bumps or edges through the skin. You may be able to feel them, even if they cannot be easily seen. In some patients, it is possible that cartilage or bone grafts can be partially absorbed by the body or change shape to the extent that they may need to be replaced.

The best candidates for rhinoplasty surgery are individuals who are looking for improvement but not perfection with regard to the appearance of their nose. In addition to realistic expectations, good health and psychological stability are important qualities for a person who is considering rhinoplasty surgery. You must also understand the possible risks and complications and consider the other options available. Rhinoplasty can be performed in conjunction with other procedures (e.g., genioplasty) to enhance your facial appearance.

_____ **ALTERNATIVE TREATMENT**

Initial here Alternative forms of management include not undergoing any surgery at all or considering other more or less extensive nasal procedures. Risks and potential complications are also associated with alternative forms of treatment or surgery and should be considered before you make a decision about the procedure that is right for you.

_____ **RISKS OF RHINOPLASTY SURGERY**

Initial here With any type of surgery, there is inherent risk. An individual's decision to undergo a specific rhinoplasty procedure is based on the comparison of the risk to the potential benefit. Although the majority of patients do not experience the complications detailed below, you should discuss each of them with your surgeon. Be sure that you understand the risks, potential complications, and consequences of the specific rhinoplasty procedure that you intend to undergo.

_____ **BLEEDING**

Initial here It is possible, although unusual, that you will have problems with bleeding during or after surgery. Should postoperative bleeding occur, it may require urgent treatment. Do not take any aspirin or similar anti-inflammatory medications for 2 weeks before and after surgery, because such medications contribute to a greater risk of bleeding. Hypertension (high blood pressure) that is not under medical control may also cause bleeding during or after surgery.

_____ **INFECTION**

Initial here Infection is unusual after rhinoplasty surgery. If an infection does occur, further treatment (e.g., change in antibiotics, drainage, additional procedures) may be necessary.

_____ **SCARRING**

Initial here Although good wound healing after a rhinoplasty procedure is expected, abnormal healing may occur within both the skin and the deeper tissues. Scars may be unattractive and of a different color than the surrounding skin. Additional treatments may be possible to treat scarring.

_____ **DAMAGE TO DEEPER STRUCTURES**

Initial here Deeper structures (e.g., nerves, tear ducts, blood vessels, muscles) may be altered during the course of surgery. The potential for this to occur varies with the type of rhinoplasty procedure performed and your individual anatomy. Injury to the deeper structures may be temporary or permanent.

_____ **UNSATISFACTORY RESULTS**

Initial here The rhinoplasty surgery may result in visible or tactile deformities, loss of function, or structural malposition. You may be disappointed that the aesthetic results of surgery do not meet your expectations. Additional treatment or surgery may be possible should the result be unfavorable for you.

_____ **NUMBNESS**

Initial here There is a potential for permanent numbness within the nasal skin after rhinoplasty. The occurrence of this is not entirely predictable. Diminished sensation in the nasal soft tissues may not totally resolve after rhinoplasty.

_____ **FACIAL ASYMMETRY**

Initial here The human face is normally asymmetric. There can be variations from one side of the face to the other with regard to the results obtained with a rhinoplasty procedure.

_____ **CHRONIC PAIN**

Initial here Chronic pain may occur infrequently after rhinoplasty. The chronic pain or discomfort may be felt on either the inside or outside of the nose.

_____ **SKIN DISORDER/SKIN CANCER**

Initial here Rhinoplasty is a surgical procedure that is performed to potentially reshape both the internal and external structures of the nose. Skin disorders and skin cancer may occur independently of an elective rhinoplasty procedure.

_____ **ALLERGIC REACTIONS**

Initial here Skin allergies to tape, suture material, and topical preparations can occur. Systemic allergic reactions, which are more serious, may occur in response to drugs used during surgery and prescription medicines taken afterward. Allergic reactions may require additional treatment.

_____ **DELAYED HEALING**

Initial here Wound disruption or delayed wound healing is possible. Some areas of the face may not heal normally or may take a long time to heal. If so, further treatment or surgery may be possible to improve the affected tissue.

_____ **LONG-TERM EFFECTS**

Initial here Subsequent alterations in nasal appearance may occur as a result of aging, sun exposure, or other conditions that are not related to the rhinoplasty surgery. In these circumstances, future surgery or other treatments may be possible.

_____ **NASAL SEPTAL PERFORATION**

Initial here There is the possibility that nasal septal surgery will cause an opening between the two sides of the nose. If a perforation (opening) occurs, additional treatment may be possible.

Continued

_____ **NASAL AIRWAY ALTERATIONS**

Initial here After rhinoplasty, septoplasty, or turbinate reduction procedures, changes may interfere with the ideal passage of air through the nose. If this occurs, additional surgery or treatment may be possible to improve breathing.

_____ **SURGICAL ANESTHESIA**

Initial here Both local anesthesia with an intravenous anesthetic and general anesthesia involve risk. Complications and injuries are possible with all forms of anesthesia and sedation.

_____ **HEALTH INSURANCE**

Initial here Most health insurance companies exclude coverage for surgical operations such as rhinoplasty and any related complications that may occur. Please carefully review your medical health insurance subscriber information to better understand your specific policy.

_____ **FINANCIAL RESPONSIBILITIES**

Initial here The cost of surgery involves several charges for the services provided, including the fees charged by your surgeon (i.e., surgeon's fees); the cost of surgical supplies and the use of the operating room (i.e., facility/hospital charges); and the costs associated with anesthesia (i.e., anesthesiologist's fees). Depending on whether the cost of surgery is covered by your medical insurance plan, you will be responsible for necessary copayments, deductibles, and all other charges that are not covered. Additional costs may occur should complications develop from the surgery. Any secondary surgeries and all charges involved with additional procedures (i.e., surgeon's fees, facility/hospital charges, anesthesiologist's fees) would also be your responsibility.

_____ **DISCLAIMER**

Initial here This consent document is written to communicate information about the proposed nasal surgery as well as to disclose information about risks and alternative forms of treatment. The informed-consent process attempts to define principles of risk disclosure that should generally meet the needs of most patients in most circumstances.

However, this consent document should not be considered all-inclusive with regard to defining other methods of treatment to consider and potential risks encountered. Your surgeon will provide you with additional verbal and written information that is based on all of the facts of your particular case and the current state of medical knowledge.

Consent documents are not intended to define or serve as the standard of medical care. Standards of medical care are determined on the basis of all of the facts involved in an individual case and are subject to change as scientific knowledge and technology advance and practice patterns evolve.

There are many variable factors in addition to the specific risks and potential complications that may influence the long-term results of rhinoplasty. The risks cited are those that are particularly associated with rhinoplasty. The practice of medicine and surgery is not an exact science. Although good results are hoped for, there is no guarantee or warranty expressed or implied with regard to the results that may be obtained.

CONSENT FOR RHINOPLASTY SURGERY

1. I hereby authorize my surgeon, _____, and such assistants as may be selected by my surgeon to perform the following procedure: rhinoplasty.
2. I have received, read, and understand the following information sheets:
   - Rhinoplasty (Reshaping of the Nose) Surgery
   - Book chapter about Rhinoplasty Surgery
   - Consent for Rhinoplasty Surgery
3. I recognize that, during the course of the operation, unforeseen conditions may necessitate different procedures than those listed above. I therefore authorize my surgeon, _____, and my surgeon's assistants or designees to perform such other procedures that are in the exercise of professional judgment necessary and desirable. The authority granted under this paragraph shall include all conditions that require treatment and that are not known to my surgeon at the time that the procedure is begun.
4. I consent to the administration of such anesthetics as are considered necessary or advisable. I understand that all forms of anesthesia involve risk and the possibility of complications, injury, and even death.
5. I acknowledge that no guarantee has been given by anyone with regard to the results that may be obtained from this procedure.
6. I consent to the photographing of me (head and neck region) before, during, and after surgery. These photographs may be used by my surgeon for medical, scientific, or educational purposes now and in the future.
7. For the purposes of advancing medical education, I consent to the admittance of observers into the operating room.
8. I consent to the disposal of any body tissues or medical devices that may be removed.
9. **THE FOLLOWING INFORMATION HAS BEEN EXPLAINED TO ME IN A WAY THAT I UNDERSTAND:**
   - **THE PREVIOUSLY DESCRIBED PROCEDURES TO BE UNDERTAKEN**
   - **ALTERNATIVE PROCEDURES AND METHODS OF TREATMENT THAT I HAVE DECIDED AGAINST**
   - **THE SPECIFIC RISKS AND POTENTIAL COMPLICATIONS OF THE PROCEDURES THAT I PLAN TO UNDERGO**

I CONSENT TO THE PROCEDURES AND THE ABOVE LISTED ITEMS (1 THROUGH 9). I AM SATISFIED WITH THE EXPLANATION GIVEN TO ME.

_____	_____
Patient (or Person Authorized to Sign for Patient)	Date
_____	_____
Witness	Date

References

1. Adamson P, Smith O, Cole P: The effect of cosmetic rhinoplasty on nasal patency. *Laryngoscope* 100:357–359, 1990.
2. Alajmi H, Tahiri Y, Jamal B, Gilardino MS: Montreal Children's Hospital formula for nasoalveolar molding cleft therapy. *Plast Reconstr Surg* 131:349, 2013
3. Anderson JR: New approach to rhinoplasty: A five-year reappraisal. *Arch Otolaryngol* 93:284–291, 1971.
4. Anderson JR, Johnson CM, Adamson P: Open rhinoplasty: An assessment. *Otolaryngol Head Neck Surg* 90:272, 1982.
5. Anderson JR: A reasoned approach to nasal base surgery. *Arch Otolaryngol* 110:349–358, 1984.
6. Andre RF, D'Souza AR, Kunst HP, Vuyk HD: Sub-alar batten grafts as treatment for nasal valve incompetence: Description of technique and functional evaluation. *Rhinology* 44:118–122, 2006.
7. Antia NH: Reconstruction of the face in leprosy. *Ann R Coll Surg Engl* 32:71, 1963.
8. Armstrong M Jr, Shikani AH: Nasal septal necrosis mimicking Wegener granulomatosis in a cocaine abuser. *Ear Nose Throat J* 75(9):623–627, 1996.
9. Arunchalam PS, Kitcher E, Gray J, Wilson JA: Nasal septal surgery: Evaluation of symptomatic and general health outcomes. *Clin Otolaryngol Allied Sci* 26:36–70, 2001.
10. Ashchi M, Widemann HP, James KB: Cardiac complication from use of cocaine and phenylephrine in nasal septoplasty. *Arch Otolaryngol Head Neck Surg* 121:681–684, 1995.
11. Ballert JA, Park SS: Functional considerations in revision rhinoplasty. *Facial Plast Surg* 24:348–357, 2008.
12. Bartlett SP: Discussion: Current surgical management of bilateral cleft lip in north America. *Plast Reconstr Surg* 129:1356–1357, 2012.
13. Bateman ND, Woolford TJ: Informed consent for septal surgery: The evidence-base. *J Laryngol Otol* 117:186–189, 2003.
14. Becker DG, Hill S: Midline granuloma due to illicit cocaine use. *Arch Otolaryngol Head Neck Surg* 114:90–91, 1988.
15. Becker DG: Septoplasty and turbinate surgery. *Aesthet Surg J* 23:393–403, 2003.

16. Becker DG, Bloom JD, Gudis D: A patient seeking aesthetic revision rhinoplasty and correction of nasal obstruction. *Otolaryngol Clin North Am* 42:557–565, 2009.
17. Becker DG, Ranson E, Guy C, Bloom J: Surgical treatment of nasal obstruction in rhinoplasty. *Aesthet Surg J* 30:347–378, 2010.
18. Becker SS, Dobratz EJ, Stowell N, et al: Revision septoplasty: Review of sources of persistent nasal obstruction. *Am J Rhinol* 22:440–444, 2008.
19. Blaise G, Vanhooteghem O, De La Brassinne M: Perforation of the nasal septum in cocaine abusers. *Rev Med Liege* 60:845–848, 2005.
20. Bloom JD, Kaplan E, Bleier BS, Goldstein SA: Septoplasty complications: Avoidance and management. *Otolaryngol Clin North Am* 42:463–481, 2009.
21. Brown JB: Preserved and fresh homotransplants of cartilage. *Surg Gynecol Obstet* 70:1079, 1940.
22. Businco LD, Lauriello M, Marsico C, et al: Psychological aspects and treatment of patients with nasal septal perforation due to cocaine inhalation. *Acta Otorhinolaryngol Ital* 28:247–251, 2008.
23. Byrd HS, Andochick S, Copit S, et al: Septal extension grafts: A method of controlling tip projection shape. *Plast Reconstr Surg* 100:999, 1997.
24. Byrd HS, Salomon J, Flood J: Correction of the crooked nose. *Plast Reconstr Surg* 102:2148–2157, 1998.
25. Byrd HS, El-Musa KA, Yazdani A: Definitive repair of the unilateral cleft lip nasal deformity. *Plast Reconstr Surg* 120:1348, 2007.
26. Byrd HA, Ha RY, Khosia RK, Gosman AA: Bilateral cleft lip and nasal repair. *Plast Reconstr Surg* 122:1181, 2008.
27. Cakmak O, Coskum M, Celik H, et al: Value of acoustic rhinometry for measuring nasal valve area. *Laryngoscope* 113:295–302, 2003.
28. Caravaca A, Casas F, Mochón A, et al: Centrofacial necrosis secondary to cocaine use. *Acta Otorrinolaringol Esp* 50:414–416, 1999.
29. Cavaliere M, Mottola G, Iemma M: Monopolar and bipolar radiofrequency thermal ablation of inferior turbinates: 20-month follow-up. *Otolaryngol Head Neck Surg* 137:256–263, 2007.
30. Chandra RK, Patadia MO, Raviv J: Diagnosis of nasal airway obstruction. *Otolaryngol Clin North Am* 42:207–225, 2009.
31. Chang CS, Por YC, Liou EJ, et al: Long-term comparison of four techniques for obtaining nasal symmetry in unilateral complete cleft lip patients: A single surgeon's experience. *Plast Reconstr Surg* 126: 1276–1284, 2010.
32. Chauhan N, Alexander AJ, Sepehr A, Adamson PA: Patient complaints with primary versus revision rhinoplasty: Analysis and practice implications. *Aesthet Surg J* 31(7):775–780, 2011.
33. Chen KT, Noordhoff MS: Open tip rhinoplasty. *Ann Plast Surg* 28:119, 1992.
34. Chen YL, Tan CT, Huang HM: Long-term efficacy of microdebrider-assisted inferior turbinoplasty with lateralization for hypertrophic inferior turbinates in patients with perennial allergic rhinitis. *Laryngoscope* 118:1270–1274, 2008.
35. Clark JM, Cook TA: Immediate reconstruction of extruded alloplastic nasal implants with irradiated homograft costal cartilage. *Laryngoscope* 112(6):968–974, 2002.
36. Clark JM, Cook TA: The "butterfly" graft in functional secondary rhinoplasty. *Laryngoscope* 112:1917–1925, 2002.
37. Cohen BJ, Johnson JD, Raff MJ: Septoplasty complicated by staphylococcal spinal osteomyelitis. *Arch Intern Med* 145:556–557, 1985.
38. Constantian MB: Distant effects of dorsal and tip grafting in rhinoplasty. *Plast Reconstr Surg* 90:405, 1992.
39. Constantian MB: The incompetent external nasal valve: Pathophysiology and treatment in primary and secondary rhinoplasty. *Plast Reconstr Surg* 93:919–931, 1994.
40. Constantian MB, Clardy RB: The relative importance of septal and nasal valvular surgery in correcting airway obstruction in primary and secondary rhinoplasty. *Plast Reconstr Surg* 98:38–54, 1996.
41. Constantian MB: Four common anatomic variants that predispose to unfavorable rhinoplasty results. *Plast Reconstr Surg* 105:316–331, 2000.
42. Converse J, et al: Deformities of the nose. In Converse J, editor: *Reconstructive Plastic Surgery,* Vol 2, WB Saunders, 1964, Philadelphia PA, p 743.
43. Cook JA, Murrant NJ, Evans KL, et al: Intranasal splints and their effects on intranasal adhesions and septal stability. *Clin Otolaryngol Allied Sci* 17:24–27, 1992.
44. Cook PR, Begegni A, Bryant C, Davis WE: Effect of partial middle turbinectomy on nasal airflow and resistance. *Otolaryngol Head Neck Surg* 113:413–419, 1995.
45. Corey JP: A comparison of the nasal cross-sectional areas and volumes obtained with acoustic rhinometry and magnetic resonance imaging. *Otolaryngol Head Neck Surg* 117:349, 1997.
46. Daniel RK: Rhinoplasty and rib grafts: Evolving a flexible operative technique. *Plast Reconstr Surg* 24:405, 1971.
47. Daniel RK, Lessard ML: Rhinoplasty: A graded aesthetic-anatomical approach. *Ann Plast Surg* 13:436–451, 1984.
48. Daniel RK: Rhinoplasty: Creating an aesthetic tip. *Plast Reconstr Surg* 80:775–783, 1987.
49. Daniel RK, Le Tourneau A: Rhinoplasty: Nasal anatomy. *Ann Plast Surg* 20:5, 1988.
50. Daniel RK: Rhinoplasty: A simplified three-stitch, open tip suture technique. Part I: Primary rhinoplasty. *Plast Reconstr Surg* 103:1491–1502, 1991.
51. Daniel RK: The nasal tip: Anatomy and aesthetics. *Plast Reconstr Surg* 89:216–224, 1992.
52. Daniel RK: Secondary rhinoplasty following open rhinoplasty. *Plast Reconstr Surg* 96:1539–1546, 1995.
53. Daniel RK: Tip refinement grafts: The designer tip. *Aesthet Surg J* 29:528–537, 2009.
54. David PKB, Jones SM: The complications of Silastic implants: Experience with 137 consecutive cases. *Br J Plast Surg* 24:405, 1971.
55. Deautch E, Kaufman M, Eilon A: Transnasal endoscopic management of choanal atresia. *Int J Pediatr Otorhinolaryngol* 40:19–26, 1997.
56. Deutsch HL, Millard DR: A new cocaine abuse complex. *Arch Otolaryngol Head Neck Surg* 114:90–91, 1988.
57. Dinis PB, Haider H: Septoplasty: Long-term evaluation of results. *Am J Otolaryngol* 23:85–90, 2002.
58. Dowd ET, Rugle L: *Comparative treatments of substances abuse*, New York, 2001, McGraw Hill.
59. Dutt DN, Kameswaram M: The etiology and management of atrophic rhinitis. *J Laryngol Otol* 119:843–852, 2004.
60. Farkas LG, Hajnis K, Posnick JC: Anthropometric and anthroposcopic findings of the nasal and facial region in cleft patients before and after primary lip and palate repair. *Cleft Palate Craniofac J* 30(1):1–12, 1993.
61. Fauci AS, Haynes BF, Katz P: Wegener granulomatosis: Prospective clinical and therapeutic experience with 85 patients for 25 years. *Ann Intern Med* 98:76–85, 1983.
62. Flake CG, Ferguson CF: Congenital choanal atresia in infants and children. *Ann Otol Rhinol Laryngol* 73:458–473, 1964.
63. Fomon S, et al: Cancellous bone transplants for the correction of saddle nose. *Ann Otol* 54:518, 1945.
64. Fomon S, et al: Observations on the correction of saddle nose deformity. *Ann Otol* 64:1109, 1955.
65. Futran ND, Haller JR: Considerations for free-flap reconstruction of the hard palate. *Arch Otolaryngol Head Neck Surg* 125:665, 1999.
66. Gendeh BS, Ferguson BJ, Johnson JT, Kapadia S: Progressive septal and palatal perforation secondary to intranasal cocaine abuse. *Med J Malaysia* 53(4):435–438, 1998.
67. Gibson T, Davis WB: The distortion of autogenous cartilage grafts: Its cause and prevention. *Br J Plast Surg* 30:257, 1958.
68. Gibson T: Transplantation of cartilage. In Converse JM, editor: *Reconstructive plastic surgery*, Philadelphia, 1977, WB Saunders.

69. Gillies HD, Millard DR: *Principles and art of plastic surgery.* Vol 1, Boston, 1957, Little, Brown, pp 103–110.
70. Godin MS, Waldman R, Johnson CM: The use of expanded polytetrafluoroethylene (Gore-Tex) in rhinoplasty. *Arch Otolaryngol Head Neck Surg* 121:1131, 1995.
71. Goodger NM, Wang J, Pogrel MA: Palatal and nasal necrosis resulting from cocaine misuse. *Br Dent J* 198:333–334, 2005.
72. Goodman WS: External approach to rhinoplasty. *Can J Otolaryngol* 2:207, 1973.
73. Goodman WS, Charbonneau PA: External approach to rhinoplasty. *Otolaryngology* 84:2195, 1974.
74. Goodman WS, Charles DA: Techniques of external rhinoplasty. *J Otolaryngol* 7:13, 1978.
75. Goodman WS: Septo-rhinoplasty: Surgery of the nasal tip by external rhinoplasty. *J Laryngol Otol* 94:485, 1980.
76. Goodman WS: Recent advances in external rhinoplasty. *J Otolaryngol* 10:433, 1981.
77. Gray LP: Deviated nasal septum: Incidence and etiology. *Ann Otol Rhinol Laryngol* 87(3 Pt 3 Suppl 50):3–20, 1978.
78. Grayson BH: Discussion: Limited evidence for the effect of presurgical nasoalveolar molding in unilateral cleft on nasal symmetry: A call for unified research. *Plast Reconstr Surg* 131:76e, 2012.
79. Gruber R, Nahai F, Bogdan MA, Friedman GD: Changing the convexity and concavity of nasal cartilages and cartilage grafts with horizontal mattress sutures: Part II. Clinical results. *Plast Reconstr Surg* 115:595–606, 2005.
80. Grymer LF, Hilberg O, Elbrnd O, Pederson OF: Acoustic rhinometry: Evaluation of the nasal cavity with septal deviations, before and after septoplasty. *Laryngoscope* 99:1180–1187, 1989.
81. Grymer LF: Reduction rhinoplasty and nasal patency: Change in the cross-sectional area of the nose evaluated by acoustic rhinometry. *Laryngoscope* 105:429–431, 1995.
82. Gubisch W: Extracorporeal septoplasty for the markedly deviated septum. *Arch Facial Plast Surg* 7:218–226, 2005.
83. Gulsen S, Yilmaz C, Aydin E, et al: Meningoencephalocele formations after nasal septoplasty and management of this complication. *Turk Neurosurg* 40:185–188, 2008.
84. Gunter JP, Rohrich RJ: External approach for secondary rhinoplasty. *Plast Reconstr Surg* 80:161–174, 1987.
85. Gunter JP, Rohrich RJ: Management of the deviated nose—the importance of the septal reconstruction. *Clin Plast Surg* 15:1, 1988.
86. Gunter JP, Rohrich RJ: Management of the deviated nose. *Clin Plast Surg* 15:43–55, 1988.
87. Gunter JP, Rohrich RJ: Augmentation rhinoplasty: Dorsal onlay grafting using shaped autogenous septal cartilage. *Plast Reconstr Surg* 86:39, 1990.
88. Gunter JP, Rohrich RJ: Correction of the pinched nasal tip with alar spreader grafts. *Plast Reconstr Surg* 90:821–829, 1992.
89. Gunter JP, Rhorich RJ, Friedman RM: The classification and correction of alar-columellar discrepancies. *Plast Reconstr Surg* 97:643–648, 1996.
90. Gunter JP, Friedman RM: Lateral crural strut graft: technique and clinical applications in rhinoplasty. *Plast Reconstr Surg* 99:943–952, 1997.
91. Gunter JP: The merits of the open approach in rhinoplasty. *Plast Reconstr Surg* 99: 863–867, 1997.
92. Gunter JP, Clark CP, Friedmen RM: Internal stabilization of autogenous rib cartilage grafts in rhinoplasty: A barrier to cartilage warping. *Plast Reconstr Surg* 100:161, 1997.
93. Guyuron B: Precision rhinoplasty: Part I. The role of life-size photographs and soft tissue cephalometric analysis. *Plast Reconstr Surg* 81:489, 1988.
94. Guyuron B, Vaughan C: Evaluation of stents following septoplasty. *Aesthetic Plast Surg* 19:75–77, 1995.
95. Guyuron B, Michelow BJ, Englebardt C: Upper lateral splay graft. *Plast Reconstr Surg* 102:2169–2177, 1998.
96. Guyuron B: Alar rim deformities. *Plast Reconstr Surg* 107:856–863, 2001.
97. Guyuron B, Varghai A: Lengthening of the nose with a tongue-and-groove technique. *Plast Reconstr Surg* 111:1533, 2003.
98. Guyuron B, Behmand R: Nasal tip sutures: Part II. The interplays. *Plast Reconstr Surg* 112:1130–1145, 2003.
99. Guyuron B, Jackowe D: Modified tip grafts and tip punch devices. *Plast Reconstr Surg* 120:2004, 2007.
100. Hamra ST: Crushed cartilage grafts over alar dome reduction in open rhinoplasty. *Plast Reconstr Surg* 92:352, 1993.
101. Harrill WC, Pillsbury HC, McGuirt F, Stewart MG: Radiofrequency turbinate reduction: A NOSE evaluation. *Laryngoscope* 117:1912–1919, 2007.
102. Harris S, Pan Y, Peterson R, et al: Cartilage warping: An experimental model. *Plast Reconstr Surg* 92:912, 1993.
103. Hasegawa M, Kern EB: The human nasal cycle. *Mayo Clin Proc* 52:28–34, 1977.
104. Hellings PW, Nolst Trenité GJ: Long-term patient satisfaction after revision rhinoplasty. *Laryngoscope* 117:985–989, 2007.
105. Hilberg O, Jackson AC, Swift DL, et al: Acoustic rhinometry: Evaluation of nasal cavity geometry by acoustic reflection. *J Appl Phys* 66:295–303, 1989.
106. Holmes E: A new concept in nasal bone grafts. *Arch Otol* 62:253, 1955.
107. Holmes E: The correction of nasal skeletal defects. *Trans Am Acad Ophthalmol Otolaryngol* 63:501–531, 1959.
108. Horton CE: (Discussion of) Achieving more nasal tip projection by use of small autogenous vomer or septal cartilage graft. *Plast Reconstr Surg* 56:322, 1975.
109. Huang IT, Podkomorksa D, Murphy MN, et al: Toxic shock syndrome following septoplasty and partial turbinectomy. *J Otolaryngol* 15:310–312, 1986.
110. Jackson LE, Koch R, James MD: Controversies in the management of inferior turbinate hypertrophy: A comprehensive review. *Plast Reconstr Surg* 103:300–312, 1999.
111. Joe SA: The assessment and treatment of nasal obstruction after rhinoplasty. *Facial Plast Surg Clin North Am* 12:451–458, 2004.
112. Johnson CMJ, Toriumi DN: *Open structure rhinoplasty*, Philadelphia, 1990, WB Saunders.
113. Kemker B, Liu X, Gungor A, et al: Effect of nasal surgery on the nasal cavity as determined by acoustic rhinometry. *Otolaryngol Head Neck Surg* 121:567–571, 1999.
114. Kennedy DW, Sinreich SJ: Functional endoscopic approach to inflammatory sinus disease: Current perspectives and technique modifications. *Am J Rhinol* 2:89–96, 1988.
115. Kim CS, Moon BK, Jung DH, Min YG: Correlations between nasal obstruction symptoms and objections parameters of acoustic rhinometry and rhinomanometry. *Auris Nasus Larynx* 25:45–48, 1998.
116. Kim DW, Shah AR, Toriumi DM: Concentric and eccentric carved costal cartilage: A comparison of warping. *Arch Facial Plast Surg* 8(1):42–46, 2006.
117. King TW, Bentz ML: Discussion: Montreal Children's Hospital formula for nasoalveolar molding cleft therapy. *Plast Reconstr Surg* 131:354–355, 2013.
118. Konstantinidis I, Triaridis S, Triaridis A, et al: Long-term results following nasal septal surgery: Focus on patient satisfaction. *Auris Nasus Larynx* 32:369–374, 2005.
119. Kostecki J: Correction of saddle nose deformity with a turnover hump segment procedure. *Plast Reconstr Surg* 38:372, 1966.
120. Kuriloff DB, Kimmelman CP: Osteocartilaginous necrosis of the sinonasal tract following cocaine abuse. *Laryngoscope* 99:918–924, 1989.
121. Lal D, Corey JP: Acoustic rhinometry and its uses in rhinology and diagnosis of nasal obstruction. *Facial Plast Surg Clin North Am* 12:397–405, 2004.
122. Lancaster J, Belloso A, Wilson CA, McCormick M: Rare case of naso-oral fistula with extensive osteocartilaginous necrosis secondary to cocaine abuse: Review of otorhinolaryngological presentations in cocaine addicts. *J Laryngol Otol* 114: 630–633, 2000.
123. Lane AP: Nasal anatomy and physiology. *Facial Plast Surg Clin North Am* 12:387–395, 2004.
124. Lanfranchi PV, Steiger J, Sparano A, et al: Diagnostic and surgical endoscopy in functional rhinoplasty. *Facial Plast Surg* 20:207–215, 2004.
125. Lee K, White WM, Constantinides M: Surgical and non-surgical treatments of the

nasal valves. *Otolaryngol Clin North Am* 42:495–511, 2009.
126. Lindemann J, Keck T, Leiacker R, et al: Early influence of bilateral turbinoplasty combined with septoplasty on intranasal air conditioning. *Am J Rhinol* 22:542–545, 2008.
127. Lo LJ: Primary correction of the unilateral cleft lip nasal deformity: Achieving the excellence. *Chang Gung Med J* 29:262–267, 2006.
128. Lund VJ: Objective assessment of nasal obstruction. *Otolaryngol Clin North Am* 22:279–290, 1989.
129. Makitie A, Aaltonen LM, Hyonen M, et al: Postoperative infection following nasal septoplasty. *Acta Otolaryngol Suppl* 543:165–166, 2000.
130. Maliniac J: Correction of nasal depressions by transposition of the lateral cartilages. *Arch Otol* 15:280, 1932.
131. Malki D, Quine SM, Pfleiderer AG: Nasal splints, revisited. *J Laryngol Otol* 113: 725–727, 1999.
132. Mari A, Arranz C, Gimeno X, et al: Nasal cocaine abuse and centrofacial destructive process: Report of three cases including treatment. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 93:435–439, 2002.
133. Martin TJ, Loehrl TA: Endoscopic CSF leak repair. *Curr Opin Otolaryngol Head Neck Surg* 15:35–39, 2007.
134. Mattson-Gates G, Jabs AD, Hugo NE: Perforation of the hard palate associated with cocaine abuse. *Ann Plast Surg* 26(5): 466–468, 1991.
135. McCollough EG, Mangat D: Systematic approach to correction of nasal tip in rhinoplasty. *Arch Otolaryngol* 107:12, 1981.
136. McComb H: Treatment of the unilateral cleft lip nose. *Plast Reconstr Surg* 55:596, 1975.
137. McComb H: Primary correction of unilateral cleft lip nasal deformity: A 10-year review. *Plast Reconstr Surg* 75:791–797, 1985.
138. McDowell F, Valone J, Brown J: Bibliography and historical note on plastic surgery of the nose. *Plast Reconstr Surg* 10:149, 1952.
139. Meredith GM: Surgical reduction of hypertrophied inferior turbinates: A comparison of electrofulguration and partial resection. *Plast Reconstr Surg* 81: 891–897, 1988.
140. Messinger E: Narcotic septal perforations due to drug addiction. *JAMA* 179:964–965, 1962.
141. Metzembaum M: Replacement of the lower end of the dislocated septal cartilage versus submucous resection of the dislocated end of the septal cartilage. *Arch Otolaryngol* 9:282–296, 1929.
142. Millard DR: Aesthetic reconstructive rhinoplasty. *Clin Plast Surg* 8:169, 1981.
143. Millard DR: *Principlization of plastic surgery*, Boston, 1986, Little, Brown, pp 143–146.
144. Millard DR: *A rhinoplasty tetralogy*, Boston, 1996, Little, Brown, pp 471–489.
145. Millard DR, Mejia FA: Reconstruction of the nose damaged by cocaine. *Plast Reconstr Surg* 107:419, 2001.
146. Miman MC, Deliktas H, Ozturan O, et al: Internal nasal valve: Revisited with objective facts. *Otolaryngol Head Neck Surg* 134:41–47, 2006.
147. Mommaerts MY, Nagy K: Analysis of the cleft lip-nose in the submental-vertical view. Part II. Panel study: Which is the most important deformity? *J Craniomaxillofac Surg* 36:315–320, 2008.
148. Monteiro ML: Unilateral blindness as a complication of nasal septoplasty: Case report. *Arq Bras Oftalmol* 69:249–250, 2006.
149. Moore EJ, Kern EB: Atrophic rhinitis: A review of 242 cases. *Am J Rhinol* 15: 355–361, 2001.
150. Moore TD, Van Camp C, Clement PA: Results of the endonasal surgical closure of nasoseptal perforations. *Acta Otolaryngol Belg* 49:263–267, 1995.
151. Most S: Anterior septal reconstruction: Outcomes after a modified extracorporeal septoplasty technique. *Arch Facial Plast Surg* 8:202–207, 2006.
152. Most SP: Analysis of outcomes after functional rhinoplasty using a disease-specific quality-of-life instrument. *Arch Facial Plast Surg* 8:306–309, 2006.
153. Mulliken JB: Primary repair of bilateral cleft lip and nasal deformity. *Plast Reconstr Surg* 108:181–194, 2001.
154. Mowlem R: Bone (iliac) and cartilage transplants to ear and nose: Their use and behaviour. *Br J Plast Surg* 29:182, 1941.
155. Murrell GL, Karakla DW, Messa A: Free flap repair of septal perforation. *Plast Reconstr Surg* 102:818, 1998.
156. Murrell GL: Auricular cartilage grafts and nasal surgery. *Laryngoscope* 114(12): 2092–2102, 2004.
157. Nease CJ, Krempl GA: Radiofrequency treatment of turbinate hypertrophy: A randomized, blinded, placebo-controlled clinical trial. *Otolaryngol Head Neck Surg* 130:291–299, 2004.
158. Newton JR, White PS, Lee MS: Nasal septal perforation repair using open septoplasty and unilateral bipedicled flaps. *J Laryngol Otol* 117:52–55, 2003.
159. Nolle B, Specks U, Ludemann J: Anticytoplasmic autoantibodies: Their immunodiagnostic value in Wegener granulomatosis. *Ann Intern Med* 111:28–40, 1989.
160. Noordhoff SM, Chen Y, Chen K, et al: The surgical technique for the complete unilateral cleft lip nasal deformity. *Oper Techn Plast Reconstr Surg* 2:167–174, 1995.
161. Nuara MJ, Mobley SR: Nasal valve suspension revisited. *Laryngoscope* 117: 2100–2106, 2007.
162. Nurse LA, Duncavage JA: Surgery of the inferior and middle turbinates. *Otolaryngol Clin North Am* 42:295–309, 2009.
163. O'Ryan F, Schendel S: Nasal anatomy and maxillary surgery: I. Aesthetic and anatomic principles. *Int J Adult Orthodon Orthognath Surg* 4:27, 1989.
164. O'Ryan F, Schendel S: Nasal anatomy and maxillary surgery: II. Unfavorable nasolabial esthetics following the LeFort I osteotomy. *Int J Adult Orthodon Orthognath Surg* 4:75, 1989.
165. O'Ryan F, Schendel S: Nasal anatomy and maxillary surgery: III. Unfavorable nasolabial esthetics following the LeFort I osteotomy. *Int J Adult Orthodon Orthognath Surg* 4:157, 1989.
166. Okur E, Yildirim I, Aral M, et al: Bacteremia during open septorhinoplasty. *Am J Rhinol* 20:36–39, 2006.
167. Oneal R, Beil R Jr, Schlesinger J: Surgical anatomy of the nose. *Clin Plast Surg* 23:195–222, 1996.
168. Onerci TM, Ayhan K, Ogretmenoglu O: Two consecutive cases of cerebrospinal fluid rhinorrhea after septoplasty operation. *Am J Otolaryngol* 25:354–356, 2004.
169. Ortiz-Monasterio JF, Olmeda A, Oscoy LO: The use of cartilage grafts in primary aesthetic rhinoplasty. *Plast Reconstr Surg* 67:597, 1981.
170. Owens WD: Signs and symptoms presented by those addicted to cocaine. *JAMA* 58:329, 1912.
171. Paloma V, Samper A, Cervera-Paz FJ: Surgical technique for reconstruction of the nasal septum: The pericranial flap. *Head Neck* 22:90–94, 2000.
172. Parameters for evaluation and treatment of patients with cleft lip and palate or other craniofacial anomalies. American Cleft Palate–Craniofacial Association. March, 1993. *Cleft Palate Craniofac J* 30(Suppl):S1–S16, 1993.
173. Passali D, Passali MF, Damiani V, et al: Treatment of inferior turbinate hypertrophy: A randomized clinical trial. *Ann Otol Rhinol Laryngol* 112:683–688, 2003.
174. Peck GC: The onlay graft for nasal tip projection. *Plast Reconstr Surg* 71:27, 1983.
175. Peer LA: Cartilage grafting. *Br J Plast Surg* 7:250, 1955.
176. Picavet VA, Prokopakis EP, Gabriëls L, et al: High prevalence of body dysmorphic disorder symptoms in patients seeking rhinoplasty. *Plast Reconstr Surg* 128:509–517, 2011.
177. Pitak-Arnnop P, Hemprich A, Dhanuthai K, et al: Panel and patient perceptions of nasal aesthetics after secondary cleft rhinoplasty with versus without columellar grafting. *J Craniomaxillofac Surg* 39:319–325, 2011.
178. Pirila T, Tikanto J: Unilateral and bilateral effects of nasal septum surgery demonstrated with acoustic rhinometry, rhinomanometry, and subjective assessment. *Am J Rhinol* 15:127–133, 2001.
179. Pollock RA, Rohrich RJ: Inferior turbinate surgery: An adjunct to the successful

treatment of nasal obstruction in 408 patients. *Plast Reconstr Surg* 74:227, 1984.
180. Posnick JC, Seagle MB, Armstrong D: Nasal reconstruction with full-thickness cranial bone grafts and rigid internal skeletal fixation through a coronal incision. *Plast Reconstr Surg* 86:894, 1990.
181. Posnick JC, Tompson B: Binder syndrome: Staging of reconstruction and skeletal stability and relapse patterns after Le Fort I osteotomy using miniplate fixation. *Plast Reconstr Surg* 99(4):961–973, 1997.
182. Posnick JC: Aesthetic alteration of the nose: Evaluation and surgery. In Posnick JC, editor: *Craniofacial and maxillofacial surgery in children and young adults,* Vol 44, Philadelphia, 2000, W.B. Saunders, pp 1125–1142.
183. Posnick JC: Binder syndrome: Evaluation and treatment. In Posnick JC, editor: *Craniofacial and maxillofacial surgery in children and young adults,* Vol 21, Philadelphia, 2000, W.B. Saunders, pp 446–468.
184. Posnick JC: Cranio-fronto-nasal dysplasia/fronto-nasal dysplasia: Evaluation and treatment. In Posnick JC, editor: *Craniofacial and maxillofacial surgery in children and young adults,* Vol 22, Philadelphia, 2000, W.B. Saunders, pp 469–486.
185. Posnick JC, Fantuzzo JJ, Troost T: Simultaneous intranasal procedures to improve chronic obstructive nasal breathing in patients undergoing maxillary (Le Fort I) osteotomy. *J Oral Maxillofac Surg* 65: 2273–2281, 2007.
186. Posnick JC, Agnihotri N: Consequences and management of nasal airway obstruction in the dentofacial deformity patient. *Curr Opin Otolaryngol Head Neck Surg* 18:323–331, 2010.
187. Posnick JC, Agnihotri N: Managing chronic nasal airway obstruction at the time of orthognathic surgery: A twofer. *J Oral Maxillofac Surg* 69:695–701, 2011.
188. Raghavan U, Jones NS, Romo R III: Immediate autogenous cartilage grafts in rhinoplasty after alloplastic implant rejection. *Arch Facial Plast Surg* 6:192–196, 2004.
189. Rakovar Y, Rosen G: A comparison of partial inferior turbinectomy and cryosurgery for hypertrophic inferior turbinates. *J Laryngol Otol* 110:732–735, 1996.
190. Reber M, Rahm F, Monnier P: The role of acoustic rhinometry in the pre- and postoperative evaluation of surgery for nasal obstruction. *Rhinology* 36:184–187, 1998.
191. Rees TD, Krupp S, Wood-Smith D: Secondary rhinoplasty. *Plast Reconstr Surg* 46:332, 1970.
192. Rees TD, La Trenta GS: The long face syndrome and rhinoplasty. *Perspect Plast Surg* 3:24, 1989.
193. Rethi A: Operation to shorten an excessively long nose. *Rev Chir Plast* 2:85, 1934.
194. Rettinger G, Kirsche H: Complications in septoplasty. *Arch Otolaryngol Head Neck Surg* 121:681–684, 1995.
195. Rhee JD, Poetker DM, Smith TL, et al: Nasal valve surgery improves disease specific quality of life. *Laryngoscope* 115:437–440, 2005.
196. Rinder CS: Fire safety in the operating room. *Curr Opin Anaesthesiol* 21:790–795, 2008.
197. Rizvi SS, Gauthier MG: Lateralizing the collapsed nasal valve. *Laryngoscope* 113:259–269, 2003.
198. Rohrich RJ, Gunter JP, Friedman RM: Nasal tip blood supply: An anatomic study validating the safety of the transcolumellar incision in rhinoplasty. *Plast Reconstr Surg* 95:795–799; discussion 800–801, 1995.
199. Rohrich RJ, Adams WP Jr: The boxy nasal tip: Classification and management based on alar cartilage suturing techniques. *Plast Reconstr Surg* 107:1849–1863, 2001.
200. Rohrich RJ, Janis JE, Kenkel JM: Male rhinoplasty. *Plast Reconstr Surg* 112: 1071–1085, 2003.
201. Rohrich RJ, Kurkjian TJ, Hoxworth RE, et al: The effect of the columellar strut graft on nasal tip position in primary rhinoplasty. *Plast Reconstr Surg* 130:926, 2012.
202. Rohrich RJ, Lee MR: External approach for secondary rhinoplasty: Advances over the past 25 years. *Plast Reconstr Surg* 131(2):404–416, 2013.
203. Ronchi P, Chiapasco M: Simultaneous rhinoplasty and maxillomandibular osteotomies: Indications and contraindications. *Int J Adult Orthodon Orthognath Surg* 1392:153–161, 1998.
204. Ronda JM, Sancho M, Lafarga J, et al: Midfacial necrosis secondary to cocaine abuse. *Acta Otorrinolaringol Esp* 53:129–132, 2002.
205. Rosen HM: Lip-nasal aesthetics following Le Fort I osteotomies. *Plast Reconstr Surg* 81:171, 1988.
206. Rosenfeld RM, Andes D, Bhattacharyya N: Clinical practice guideline: Adult sinusitis. *Otolaryngol Head Neck Surg* 137(Suppl): S1–S31, 2007.
207. Sajjadian A, Guyuron B: Primary rhinoplasty. *Aesthet Surg J* 30:527–539, 2010.
208. Sajjadian A, Naghshineh N: The current status of grafts and implants in rhinoplasty: Part I. Autologous grafts. *Plast Reconstr Surg* 1:40–49, 2010.
209. Sajjadian A, Naghshineh N: The current status of grafts and implants in rhinoplasty: Part II. Homologous grafts and allogenic implants. *Plast Reconstr Surg* 125:99–109, 2010.
210. Samadi DS, Shah UK, Handler SD: Choanal atresia: A twenty-year review of medical comorbidities and surgical outcomes. *Laryngoscope* 113:254–258, 2003.
211. Sarver DM, Rousso DR: Plastic surgery combined with orthodontic and orthognathic procedures. *Am J Orthod Dentofacial Orthop* 126:305–307, 2004.
212. Sastry RC, Lee D, Har-el G: Palate perforation from cocaine abuse. *Otolaryngol Head Neck Surg* 116:565–566, 1997.
213. Schlosser RJ, Park SS: Surgery for the dysfunctional nasal valve: Cadaveric analysis and clinical outcomes. *Arch Facial Plast Surg* 1:105–110, 1999.
214. Schwab JA, Pirsig W: Complications of septal surgery. *Facial Plast Surg* 13:3–14, 1997.
215. Schwenter I, Dejakum K, Schmutzhard J, et al: Does nasal septal surgery improve quality of life? *Acta Otolaryngol* 126: 752–757, 2006.
216. Seyer BA, Grist W, Muller S: Aggressive destructive midfacial lesion from cocaine abuse. *Oral Surg Oral Med Oral Pathol* 94:465, 2002.
217. Sheen JH: Achieving more nasal tip projection by use of small autogenous vomer or septal cartilage grafts. *Plast Reconstr Surg* 56:35, 1975.
218. Sheen JH: Spreader graft: A method of reconstructing the root of the middle nasal vault following rhinoplasty. *Plast Reconstr Surg* 73:230–237, 1984.
219. Sheen JH: *Aesthetic rhinoplasty*, ed 2, St. Louis, MO, 1987, Mosby-Year Book.
220. Sheen JH: Tip graft: A 20-year retrospective. *Plast Reconstr Surg* 91:48–63, 1993.
221. Sheen JH: The ideal dorsal graft: A continuing quest. *Plast Reconstr Surg* 102:2490, 1998.
222. Siegel NS, Gliklich RE, Taghizadeh F, Chang Y: Outcomes of septoplasty. *Otolaryngol Head Neck Surg* 122:228–232, 2000.
223. Sipilä J, Suonpää J: A prospective study using rhinomanometry and patient clinical satisfaction to determine if objective measurements of nasal airway resistance can improve the quality of septoplasty. *Eur Arch Otolaryngol* 254:387–390, 1997.
224. Sittel C, Eckel HE: Nasal cocaine abuse presenting as central facial destructive granuloma. *Eur Arch Otorhinolaryngol* 255:446–447, 1998.
225. Siztzman TJ, Girotto JA, Marcus JR: Current surgical practices in cleft care: Unilateral cleft lip repair. *Plast Reconstr Surg* 121:261e, 2008.
226. Skouras A, Noussios G, Chouridis P, et al: Acoustic rhinometry to evaluate plastic surgery results of the nasal septum. *B-ENT* 5:19–23, 2009.
227. Slavin SA, Goldwyn RM: The cocaine user: The potential problem patient for rhinoplasty. *Plast Reconstr Surg* 86:436, 1990.
228. Smith JC, Kacker A, Anand VK: Midline nasal and hard palate destruction in cocaine abusers and cocaine's role in rhinologic practice. *Ear Nose Throat J* 81:172–177, 2002.
229. Smith DM, MacIssac ZM, Losee JE: Discussion: Limited evidence for the effect of presurgical nasoalveolar molding in unilateral cleft on nasal symmetry: A call for unified research. *Plast Reconstr Surg* 131:72e, 2012.

230. Song C, MacKay DR, Chait LA, et al: Use of costal cartilage cantilever grafts in Negroid rhinoplasties. *Ann Plast Surg* 27:201, 1991.
231. Spielman PM, White PS, Hussain SSM: Surgical techniques for the treatment of nasal valve collapse: A systematic review. *Laryngoscope* 119:1281–1290, 2009.
232. Stal S, Brown RH, Higuera S, et al: Fifty years of the Millard rotation-advancement: Looking back and moving forward. *Plast Reconstr Surg* 123:1364–1377, 2009.
233. Stewart MG, Witsell DL, Smith TL, et al: Development and validation of the Nasal Obstruction Symptom Evaluation (NOSE) scale. *Otolaryngol Head Neck Surg* 130: 157–163, 2004.
234. Stewart MG, Smith TL, Weaver EM, et al: Outcomes after nasal septoplasty: Results from the Nasal Obstruction Symptom Evaluation (NOSE) study. *Otolaryngol Head Neck Surg* 130:183–290, 2004.
235. Stucker FJ, Hoasjoe DK: Nasal reconstruction with conchal cartilage: Correcting valve and lateral nasal collapse. *Arch Otolaryngol Head Neck Surg* 120: 653–658, 1994.
236. Tan SPK, Greene AK, Mulliken JB: Current surgical management of bilateral cleft lip in north America. *Plast Reconstr Surg* 129:1347–1355, 2012.
237. Tardy ME: *Rhinoplasty: The art and the science*, Philadelphia, 1997, WB Saunders.
238. Tawadros AM, Prahlow JA: Death related to nasal surgery: Case report with review of therapy-related deaths. *Am J Forensic Med Pathol* 29:260–264, 2008.
239. Tebbetts JB: Shaping and positioning the nasal tip without structural disruption: A new systematic approach. *Plast Reconstr Surg* 94:61–77, 1994.
240. Thomson C, Mendelsohn M: Reducing the incidence of revision rhinoplasty. *J Otolaryngol* 36:130–134, 2007.
241. Toriumi DM, Josen J, Weinberger M, Tardy ME Jr: Use of alar batten grafts for correction of nasal value collapse. *Arch Otolaryngol Head Neck Surg* 123:801–808, 1997.
242. Turk AE, Chang J, Soroudi AE, et al: Free flap closure in complex congenital and acquired defects of the palate. *Ann Plast Surg* 45:274, 2000.
243. Uppal S, Mistry H, Nadig S, et al: Evaluation of patient benefit from nasal septal surgery for nasal obstruction. *Auris Nasus Larynx* 32:129–137, 2005.
244. Van de Heijden P, Dijkstra PU, Stellingsma C, et al: Limited evidence for the effect of presurgical nasoalveolar molding in unilateral cleft on nasal symmetry: A call for unified research. *Plast Reconstr Surg* 131:62e, 2013.
245. Veale D, De Haro L, Lambrou C: Cosmetic rhinoplasty in body dysmorphic disorder. *Br J Plast Surg* 56(6):546–561, 2003.
246. Vilensky W: Illicit and licit drugs causing perforation of the nasal septum. *J Forensic Sci* 27:958–962, 1982.
247. Vuyk HD, Langenhuijsen KJ: Aesthetic sequelae of septoplasty. *Clin Otolaryngol Allied Sci* 22:226–232, 1997.
248. Waite PM, Matukas VJ, Sarver DM: Simultaneous rhinoplasty and orthognathic surgery. *Int J Oral Maxillofac Surg* 17:298, 1987.
249. Webster RC, Davidson TM, Smith RC: Importance of the columellar-labial junction in rhinoplasty. *Head Neck Surg* 1:423, 1979.
250. Weir R: On restoring sunken noses. *NY Med Jour* 499, 1892.
251. Wheeler ES, Kawamoto HK, Zarem HA: Bone grafts for nasal reconstruction. *Plast Reconstr Surg* 69:9, 1982.
252. Wolfe SA: A pastiche for the cleft lip nose. *Plast Reconstr Surg* 114:1, 2004.
253. Woolf R, Snow J, Walker JH, Broadbent TR: Correction of saddle nose deformity with the upper-lateral turnover procedure. *Plast Reconstr Surg* 35:310, 1965.
254. Yeow VK, Chen PK, Chen YR, Noordhoff SM: The use of nasal splints in the primary management of unilateral cleft nasal deformity. *Plast Reconstr Surg* 103: 1347–1354, 1995.
255. Zijlker TD, Quaedvlieg PC: Lateral augmentation of the middle third of the nose with autologous cartilage in nasal valve insufficiency. *Rhinology* 32:34–41, 1994.

39

Aesthetic Alteration of Prominent Ears: Evaluation and Surgery

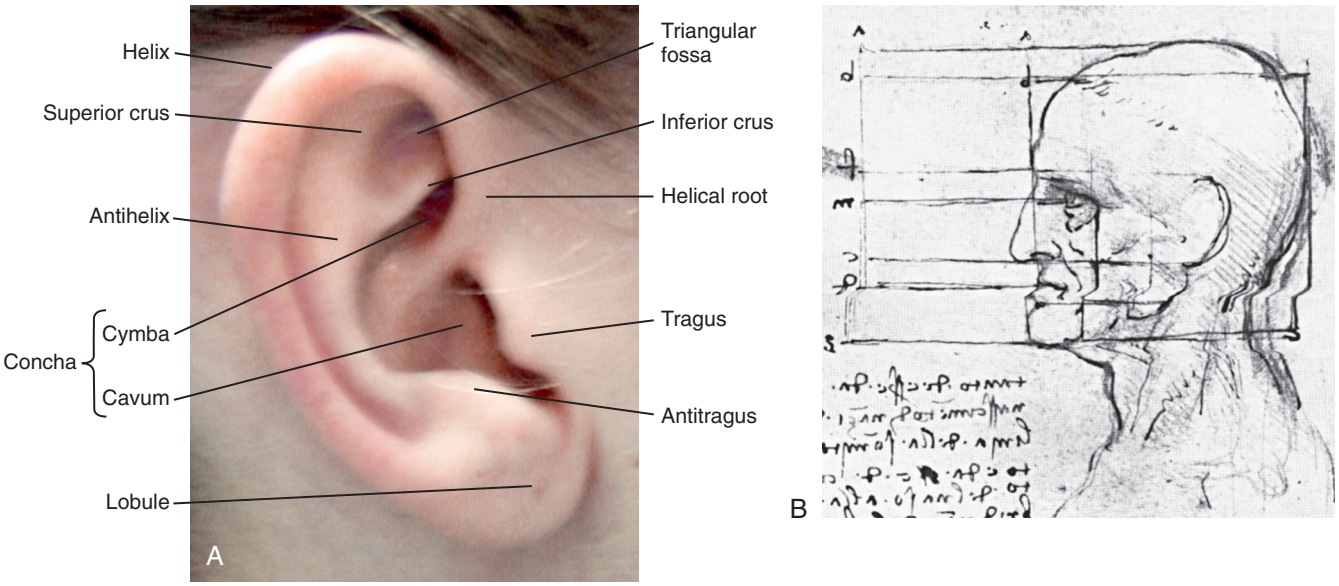
JEFFREY C. POSNICK, DMD, MD

- Historic Perspective
- Embryology and Anatomy
- Timing of Surgery
- Aesthetic Objectives
- Surgical Principles
- Surgical Technique
- Otoplasty for Prominent Ears: Step-by-Step Approach
- Assessment of Results
- Complications and Suboptimal Aesthetic Results
- Non-Surgical Correction of Prominent Ears in the Newborn
- Conclusions
- Patient Education Materials

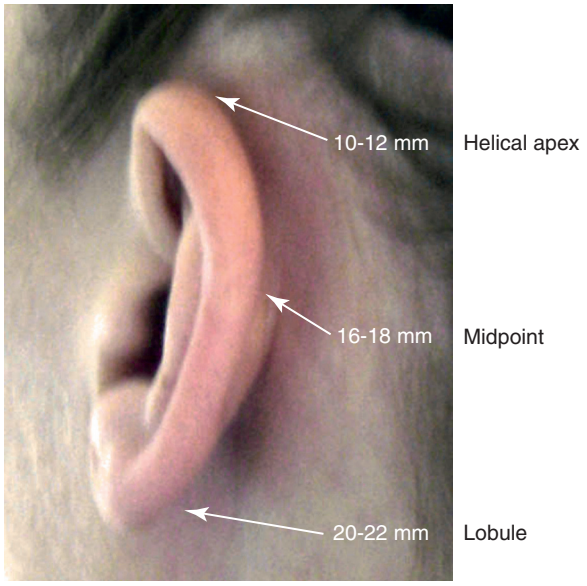
Aside from the excision of auricular tags and the repair of traumatically clefted ear lobes, the *prominent ear* is the most commonly treated external ear deformity. Approximately 5% of the Caucasian population has protruding ears, thereby making this the most frequent congenital anomaly of the external ear. *Otoplasty* is a common aesthetic operation that is carried out among children and adolescents to address this issue. There is a wide variation with regard to what is considered normal for ear size, prominence, and shape (Fig. 39-1). An "outstanding" ear is said to be present when the distance of the helical rim from the temporomastoid surface of the skull is excessive. As a general rule, a measurement of more than 16 to 21 mm is said to result in a prominent ear (Fig. 39-2). The average ear is 6.5 cm long and 3.5 cm wide, but significant variations are considered acceptable. After the completion of normal ear growth and with ongoing age, the external ear changes in that the elongation of the soft tissue covering and the earlobe is to be expected. Provided that the ear has a morphology that is in balance with the other facial features and that there is reasonable symmetry from one ear to the other, these variations are considered to be in an acceptable aesthetic range. However, when ear shape, size, and proportion are perceived as distracting to the casual observer at conversational distance, the ears may become a source of ridicule and personal anguish. Affected children are frequently stigmatized by their peers and commonly ask their parents if a solution for their visible abnormal ear shape can be found. In these circumstances, the surgical correction of the prominent ear is likely to have a beneficial effect on the person's self-esteem and body image.

A *failure of scaphal folding* (i.e., limited antihelical fold) as well as *conchal hypertrophy* (i.e., increased depth of the conchal wall), *prominent lobules* (i.e., protruding ear lobes); and *anterolateral rotation of the concha* (i.e., cupping of the ear) frequently create prominence of the external ear.[5,13,14,30,52,57,67] Noticeable ear protrusion often results from a combination of these deformities. An accurate assessment of the ear dysmorphology is essential to the achievement of an unobtrusive look through surgery. Adjusting the scaphal (antihelical) folding with sutures, reducing the conchal wall depth via conchal excision and conchal mastoid sutures, and performing lobe set-back with sutures and excision are typical components of the surgical correction that is undertaken to achieve a favorable result.[3,4,6,11,26,50,55,60,70,72]

Overemphasis on the surgical creation of an antihelical fold without paying proper attention to conchal hypertrophy often results in an oversharpened antihelix and a buried helical rim (i.e., "telephone ear"). Some surgeons advocate cartilage-cutting (i.e., scoring) procedures rather than sutures to achieve a preferred antihelical fold.[11] These cartilage-scoring techniques generally require dissection of the soft tissues off of the anterior surface of the cartilage. This can result in hematoma, skin necrosis, and chondritis, which may result in irreversible cartilage or external ear irregularities. Surgical correction of the outstanding ear



• **Figure 39-1 A,** The topographic anatomy of the external ear is shown. Key anatomic landmarks are demonstrated and labeled. **B,** Drawing by Leonard da Vinci that confirms the importance of achieving Euclidian proportions of the external ear within the face.



• **Figure 39-2** Ear protrusion is measured as the distance of the helical rim from the cranium; it is measured in millimeters. The specific locations where measurements are taken include the helical apex, the midpoint, and the lobe.

should in general be uncomplicated and result in reliable, long-lasting, favorable aesthetics with minimal downtime for the patient and minimal chance of relapse.

When considering the ideal timing for external ear surgery, it is important to balance what we know about ear and facial growth, psychosocial development, and the ability of the child to comply with the necessary routines after surgery.[1,20,21,39] The goals of otoplasty for the management of congenitally prominent ears, as outlined by McDowell, remain universal despite a multitude of etiologies and suggested surgical techniques.[42] According to McDowell, the surgical goals should include the following:

1. Correcting ear protrusion, by addressing the over prominent helix and antihelix
2. Achieving a smooth antihelical fold without a "sharp" crease
3. Limiting any disturbance to the postauricular sulcus
4. Avoiding a "plastered down" appearance of the ear

Historic Perspective

Dieffenbach is credited with reporting the first otoplasty in the medical literature in 1845.[15] This was performed to reconstruct a traumatically deformed ear. The ear surgery consisted of excising skin from the postauricular sulcus and suturing the conchal cartilage to the mastoid periosteum (i.e., conchal–mastoid suturing). In 1881, Ely was the first surgeon to report a procedure for the aesthetic correction of a congenitally prominent ear.[19] He resected both the skin and the conchal cartilage, but he did so as a two-stage procedure (i.e., one ear at a time).[56] Moristin further developed these skin-excision and cartilage-resection otoplasty techniques.[44] In 1910, Luckett realized that a prominent ear frequently resulted from the congenital failure to form an antihelical fold.[38] He was the first to describe setting the ear back via the removal of postauricular skin and by simultaneously completing an incision through the length of the ear cartilage in an attempt to create the deficient antihelical fold; he also placed sutures to hold the new fold in place. In 1960, Stenstrom used the known principles of cartilage biology as described by Gibson and Davis in 1958 to propose the scoring and abrading the anterior cartilage surface to weaken it and thereby create an antihelical

fold.[61,62,25] In 1963, Mustarde introduced the placement of mattress sutures in the posterior cartilage surface of the ear as a technique for the creation of a new antihelical fold.[46-48]

In 1967, Kaye combined the anterior scoring technique of Stenstrom and the posterior suture placement technique of Mustarde to limit the relapse that surgeons complained about when using either technique alone; he also advocated the use of nonresorbable suture.[32] In 1968, Furnas reintroduced the technique of conchal–mastoid suturing to set back the prominent conchal bowl.[22-24] He also advocated cleaning out the soft tissue in the postauricular groove (i.e., postauricular muscle and fibrofatty tissue) so that the concha could be rotated in a sagittal plane from posterior to anterior (clockwise rotation) before anchoring it to the mastoid fascia (conchal–mastoid sutures) with nonresorbable sutures. In 1969, Webster advocated paying special attention to the prominent earlobe to achieve a successful otoplasty.[69] He noted that the "tail" of the helix could be repositioned with suture to change the lobe's orientation. In 1972, Elliott combined and refined all of the previously described techniques to manage, the deep conchal wall, the limited antihelical fold, and the abnormal earlobe that were frequently seen in the prominent ear.[18] He advocated the use of elliptical partial conchal wall excision when conchal–mastoid suturing alone was felt to be insufficient to correct the ear prominence. He used an anterior incision placed within the "shadow zone" of the conchal margin when conchal resection was required. Bauer advocated the use of the Elliott anterior conchal cartilage excision but preferred to also excise anterior skin to avoid postoperative skin folding or bunching.[4]

Embryology and Anatomy

Embryologically, the ear arises from six hillocks of the first and second branchial arches during the second to fourth month of gestation. Hillocks 1 through 3, from the first arch, form the anteromedial aspects of the auricle (i.e., the tragus, the helical crus, and the helix, respectively). Hillocks 4 through 6, from the second arch, develop into the posterolateral aspects of the ear (i.e., the antihelix and the antitragus, respectively).[34,35] During the third month of gestation, protrusion of the auricle increases. By the end of the sixth month, the helical margin curves, the antihelix forms its folds, and the antihelical crura appear at the superior aspect of the forming external ear. Congenital ear deformities result from the disruption of one or more of the auricular hillocks during embryogenesis. Any deforming forces to the side of the head of the fetus may also alter the shape of the external ear.

The ear is supplied by branches from the superficial temporal artery anteriorly and the posterior auricular and occipital arteries posteriorly. The superficial temporal, posterior auricular, and retromandibular veins provide venous drainage. The lymphatic drainage of the anterior three hillocks is mainly to the anterior triangle of the neck, whereas the posterior three hillocks ultimately drain into the posterior triangle of the neck. Lastly, the ear is supplied by the great auricular nerve on its lower lateral and inferior cranial surfaces, by the lesser occipital nerve on its superior cranial surface, and by the auriculotemporal nerve on its superior lateral surface as well as the anterior superior surface of the external acoustic meatus. The Arnold nerve, which is an auricular branch of the vagus nerve, supplies the posterior inferior external auditory canal and meatus as well as the inferior conchal bowl.[34,35]

The most frequently seen deformity or malformation of the external ear arises from the failure of the antihelix to fold. The conchoscaphal angle widens and results in a flat superior crus, antihelical body, and inferior crus. The helical roll (i.e., the antihelical fold) may be absent altogether or only partially formed, with a wide variation of deformity. Conchal widening (i.e., the presence of a deep conchal bowl) may also occur either as an isolated deformity or in conjunction with antihelical fold deformities. These congenital abnormalities are usually bilateral and often asymmetric, and they frequently demonstrate a familial pattern.

Bauer pointed out that the external ear may be thought of as consisting of a three-tiered cartilaginous framework with tightly adherent skin on the anterior surface and loosely applied skin on the posterior surface.[4] The three tiers of sculpted cartilage form are the helical–lobular complex, the antihelical–antitragal complex, and the conchal complex. Key anatomic (aesthetic) landmarks of the normal ear include the scapha, the concha, the helix, the antihelix, the tragus, and the lobule (see Figs. 39-1 and 39-2).

Timing of Surgery

Auricular deformations are best suited for ear molding correction techniques. Molding therapy is preferably carried out during the first few days of life, as described later in this chapter. In general, the surgical correction of a malformation of the external ear is planned with an appreciation of normal ear maturation. As a result of differing interpretations of ear growth and the effect of an operation on residual growth of the auricle, opinions vary with regard to the preferred timing of definitive external ear reconstruction.

In a study by Farkas, two anthropometric surface measurements, ear width and ear length, were taken directly from the ears of normal subjects with the use of an anthropometric sliding caliper.[20,21] At the age of 1 year, ear width is highly developed in both sexes (mean, 93.5%), and it is mostly completed at the age of 5 years (mean, 97%). The width of the ear reaches its full mature size in boys at the age of 7 years and in girls at the age of 6 years. At 1 year of age, the length of the ear has already attained 75% of its eventual adult size. By 5 years of age, 87% of its eventual development is observed. Ear length achieves its full adult size in 13-year-old boys and 12-year-old girls. Adamson and colleagues reviewed the growth patterns of the external ear and concluded that the ear reaches 85% of its overall adult size by the age of 3 years and that little change in ear width

or the distance of the ear from the scalp occurs after 10 years of age.[1] The authors concluded that, for all practical purposes, the normal ear is almost fully developed by the age of 6 years.

Although children notice differences in each other beginning very early in life, they are rarely self-conscious about differences in ear protrusion before 5 years of age.[39] From this point on, however, social interactions with peers frequently elicit negative comments that can affect self-esteem and body image. The correction of external ear deformities before this age is possible, but the postoperative course may be compromised if the ear bandage is removed early by the uncooperative child or with disruption of the repair by pulling on the ears after the bandage is removed. Consideration is given to setting back the protruding ears as early as 3 years of age, although many children display improved cooperation and parents are more confident in their decision when children are between the ages of 5 and 6 years.

Aesthetic Objectives

It is recognized that there are wide variations with regard to ear proportions, projection, and position among individuals. When feasible, the surgically reconstructed (previously protruding) ear should be of ideal size and shape and have a good relationship with the other facial features and the contralateral ear. Loose guidelines for the assessment of preferred ear shape and position have been summarized by Tolleth.[67]

Ear Axis

A line that passes through the longest dimension of the ear is called its *axis*. In the majority of people, an angle of 20 degrees formed between the axis of the nasal bridge and the axis of the ear is considered the most aesthetically pleasing. The axis of an aesthetically pleasing ear is rarely vertical.

Ear Position

When the head is in the true horizontal (natural head) position, the top of the ear will generally be level with the eyebrow, whereas the bottom of the ear will generally be on a line with the base of the columella of the nose or slightly lower. In the anteroposterior dimension, the ear is roughly positioned one ear length (i.e., 6.5 to 7.5 cm) posterior to the lateral rim of the orbit.

Ear Size

A wide variation exists with regard to what is considered normal ear size in terms of length and width. The length of a "normal" ear varies from 5.5 to 7.5 cm, whereas a normal ear width varies from 3.0 to 4.5 cm. The normal width-to-length ratio is 50% to 65%.

Ear Protrusion

Although the spectrum of protrusion of the helical rim from the scalp varies widely, a measurement of 1.5 to 2.0 cm is aesthetically acceptable.[2]

Patient and family satisfaction should be anticipated if the reconstructed ears are no longer a facial feature that draws attention when viewed at conversational distance by the casual observer. Sharp angles, unnatural contours, overcorrection and "eye-catching" asymmetry from ear to ear are not ideal. In the operating room, the surgeon should view the reconstructed ear from several feet of distance in frontal and profile views and from the rear. From the rear, the helical contour should form a relatively straight line; this will indicate that the antihelical fold (the upper third), the conchal depth (the middle third), and the lobe (the lower third) are in sync. From the front, the helical rim above and the lobe below should be seen to an extent just beyond the antihelix without any cupping of the upper and lower thirds of the ear. In profile, the contours should be gentle and the folds rounded.

Surgical Principles

A reconstructed prominent ear should be reasonably symmetrical with its counterpart and without a sharply angulated antihelical fold. If the upper third of the ear protrudes as a result of an absent or weak antihelical fold, then the fold should be improved (i.e., Mustarde and superior helix–mastoid stitches). If the middle third of the ear is too prominent (i.e., outstanding and away from the side of the head), then the concha must be recessed (i.e., conchal wall excision and conchal–-mastoid stitches) and the antihelical fold improved (i.e., Mustarde stitches). In the lower third of the ear, if the lobule protrudes, the surgeon must resect or reposition the cartilaginous tail, excise the retrolobular skin, or both.

Historically, the creation of an *antihelical fold* was carried out by techniques that involved either scoring or abrasion of the cartilage to control the direction and extent of folding.[16,25,28] Later, suture techniques (i.e., Mustarde sutures) were added to reshape the cartilage. Many surgeons combine these methods. Interestingly, some "cartilage scorers" recommend partial-thickness scoring, whereas others score the full thickness of the cartilage.[11] This author prefers to avoid scoring the cartilaginous surface all together, because this requires separation of the anterior skin from the cartilage surface with increased risk of hematoma, flap necrosis, and visible sharp edges on the anterior (cartilage) surfaces. The careful placement of Mustarde mattress sutures in a gentle curve will form a smooth antihelical fold. The Mustarde sutures are placed along the posterior surface of the cartilage as needed in the upper, middle, and lower thirds to accomplish aesthetic antihelical folding.[46-48]

Conchal hypertrophy is a frequent component of the ear deformity. The correction of this aspect is usually

accomplished through a combination of techniques, including the following: 1) the repositioning of the concha into the surgically "cleaned out" mastoid recess; 2) conchal setback with conchal–mastoid sutures; and 3) conchal reduction through direct elliptical cartilage excision. If the anterior approach is selected for conchal wall resection, an incision (scar) must be placed on the anterior surface, but the folding of redundant skin is limited.[4] If a posterior approach is used, there is a risk that the redundant anterior skin that is left behind may not shrink sufficiently and thus result in a visible fold in the conchal floor. Some surgeons believe that a redundant anterior skin fold may be more noticeable than a "fine" anterior conchal scar. This author prefers to excise conchal cartilage with the use of a posterior approach. The anterior skin is then released from the residual conchal floor cartilage to avoid bunching, as discussed later in this chapter.

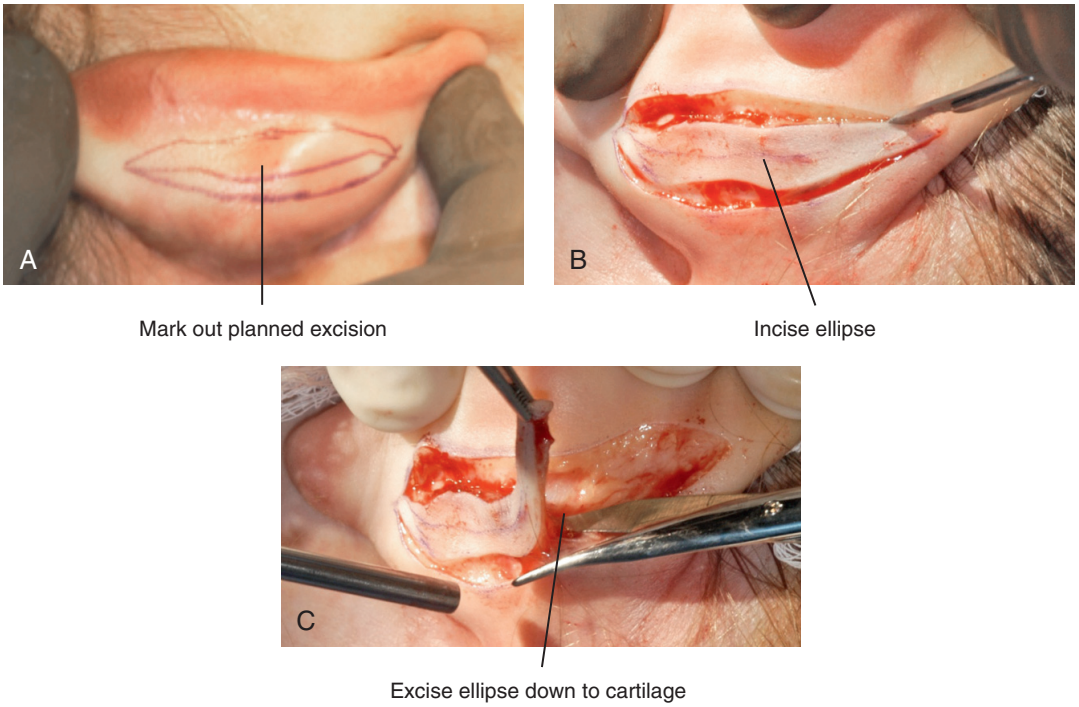
To review, the *ear set-back procedure* will generally require the resection of conchal (wall) cartilage, the cleaning out of excess soft tissues in the retroauricular sulcus, and three rows of deep sutures. These three rows of sutures include the following: 1) Mustarde horizontal mattress sutures to create an antihelical fold; 2) conchal sutures for reconstructing after elliptical (conchal wall) resection; and 3) Furnas-type sutures, including placement in the conchal–mastoid region and the superior helix–mastoid region. Nonresorbable sutures should be used to limit relapse (4-0 Mersilene versus 4-0 clear nylon).

Surgical Technique
(Figs. 39-3 through 39-9)

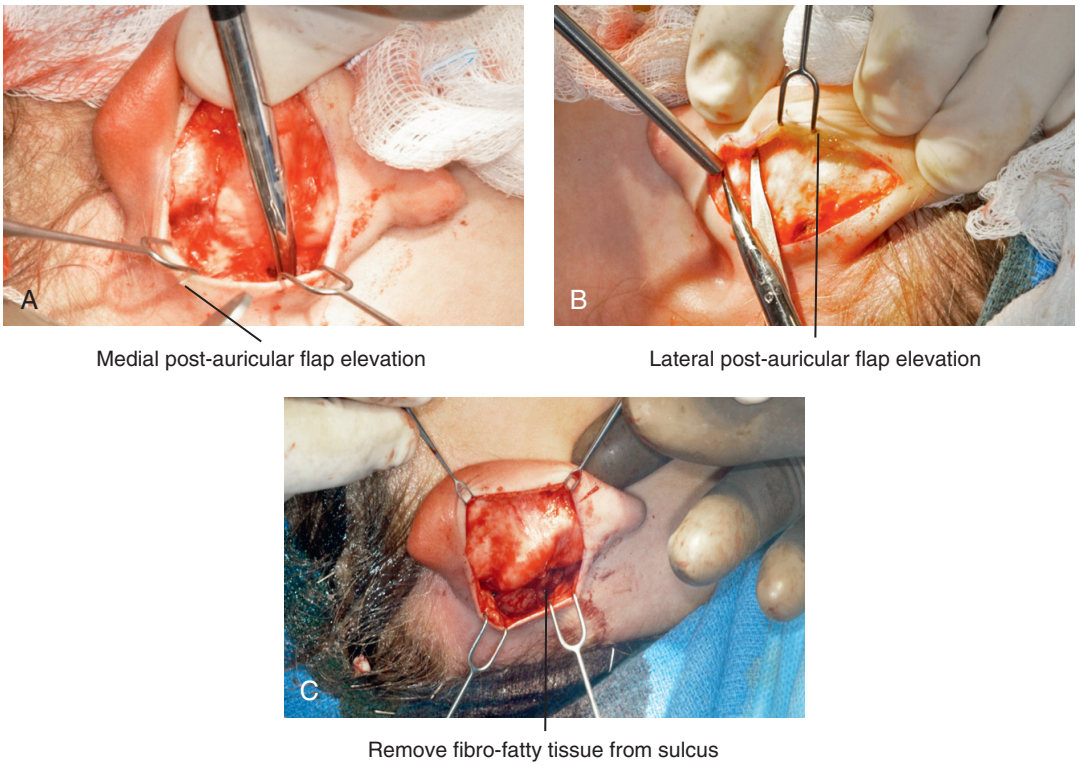
The intraoperative draping of the patient within the sterile field should provide visualization of both ears and the entire forehead, face, and neck as well as the ability to safely turn the patient's head from side to side. An orotracheal tube with a natural curve at the level of the lower lip is used and secured to the skin of the chin with a minimal amount of tape (e.g., an Oral RAE endotracheal tube). Ophthalmic eye ointment and eye protectors (corneal shields) are placed below the eyelids to limit corneal injury and to prevent the need for tape or dressing material on the face. The patient's head is placed on the Mayfield horseshoe head ring in the neutral neck position. The Mayfield provides stability and adequate exposure and allows for the turning of the head without disrupting the surgical field. The majority of the hair is wrapped in a sterile towel, and prophylactic intravenous antibiotics are given. A mouth pack is placed to limit saliva contamination of the surgical field.

The ears are marked on the anterior surface for the creation of a new or improved antihelical fold and on the posterior surface for an elliptical skin excision. Lidocaine (Xylocaine) 1% with 1:100,000 epinephrine is injected into the postauricular skin fold.

The postauricular incisions (i.e., elliptical skin excision) are completed with a scalpel (see Fig. 39-3). All soft tissue



• **Figure 39-3** Intraoperative views of postauricular skin incisions and excision as part of otoplasty. **A,** The location of the postauricular elliptical skin excision is demonstrated. The planned long axis of the ear excision extends from the superior helical rim to the earlobe. **B,** With the use of a knife (No. 15 blade), the full thickness of the skin ellipse is incised. **C,** With the use of blunt-tip Stevens scissors, the full thickness of the skin ellipse is removed, down to the surface of the cartilage.



• **Figure 39-4** Intraoperative views of postauricular flap elevation and then subcutaneous tissue excision as part of otoplasty. **A,** Elevation of the medial postauricular flap is completed down to the mastoid fascia. **B,** Elevation of the lateral postauricular flap is extended to the outer edge of the helical rim. The flaps are also elevated inferiorly to the tail of the helix and superiorly to the upper aspect of helix. **C,** All soft tissue is cleaned out in the sulcus down to mastoid fascia (i.e., muscle and fibrofatty tissue).

down to the posterior surface of the cartilage is removed. The postauricular medial and lateral skin flaps are elevated (see Fig. 39-4). The resection and removal of postauricular muscle and fibrofatty tissue that is between the conchal cartilage and the mastoid fascia is completed. This allows the concha to rest in a deep pocket so that the bending of the cartilage over a soft-tissue mound does not occur when tying conchal–mastoid sutures (Fig. 39-6A).

When scaphal folding is needed to improve the antihelical fold, the placement of postauricular horizontal mattress (Mustarde-type) sutures is carried out (see Figs. 39-5 and 39-6). Before the creation of the improved antihelical fold, the posterior cartilaginous surface is cleaned of all soft tissue. The Mustarde sutures are placed in a radial fashion to form a curved scaphal fold that extends from the superior crus down to the lobule. Antihelical reconstruction usually requires three to five Mustarde sutures to achieve a smooth roll. Each suture is tested for its effectiveness after it is placed, but none are tied until all are in position. If the position of the superior helix remains protruded, one or two additional superior helix–mastoid sutures can be placed to further accomplish the ear set-back.

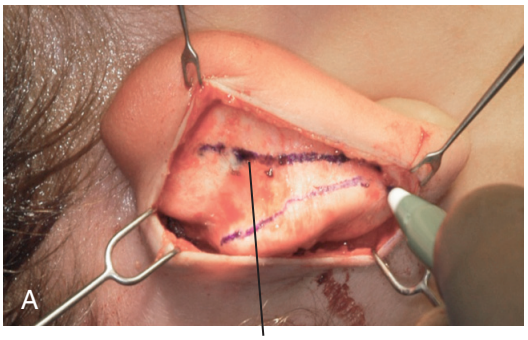
A conchal set-back with sutures (conchal–mastoid sutures) is useful but generally inadequate for the management of even moderate degrees of conchal hypertrophy. Partial conchal wall resection is generally indicated (see Figs. 39-7 through 39-9). This author prefers to complete the resection through a posterior approach. An ellipse of the conchal wall that extends superiorly and inferiorly is excised to allow the bowl to reposition itself without a cartilaginous "dog ear" (i.e., puckering). If only minor degrees of conchal prominence exist, management just with the placement of conchal–mastoid sutures may be possible.

The position of the lobule can be further managed by the setting back of the tail of the helix. The helical tail is exposed on its posterior surface. The tail is sutured back to the concha or the mastoid fascia. Each placed suture is tested to determine if the desired position of the lobule will be achieved. In some patients, it is useful to excise cartilage at the lobule to achieve adequate positioning. In others, the excision of a postauricular wedge of skin is helpful.

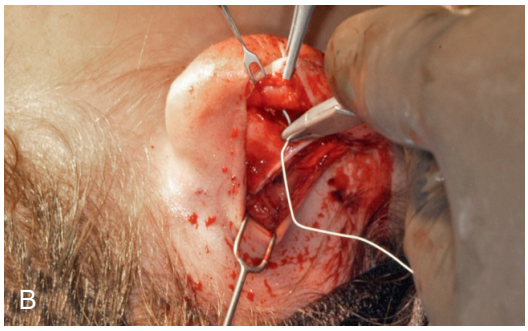
Otoplasty for Prominent Ears: Step-by-Step Approach

Positioning on the Operating Room Table

- Place the patient supine on the operating table with his or her head resting on the Mayfield horseshoe. The neck should be in the neutral position.



Mark out anti-helical fold



Placement of (3-5) horizontal mattress "Mustarde" sutures (4-0 merseline)

Confirm new anti-helical fold prior to tying down

• **Figure 39-5** Intraoperative views of the creation or deepening of the preferred antihelical fold as part of the otoplasty. **A,** The preferred antihelical fold is located and marked on the posterior surface of cartilage with a surgical pen or methylene blue. **B,** The new antihelical fold is created or deepened by placing three to five carefully located horizontal mattress (Mustarde-type) sutures. The sutures are placed in a natural curve like the spokes of a wheel. Each placed Mustarde suture is tested for effectiveness with a single throw, after which it is loosened. **C,** After all sutures have been placed and tested, each will be carefully tied and confirmed for the preferred aesthetic effect. Note that the conchal wall excision is completed prior to placement of Mustarde stitches. Only after all of the Mustarde stitches have been secured, are the edges of the conchal walls (after elliptical excision) approximated with the use of interrupted sutures.

General Anesthesia Intubation

- Place the orotracheal (Oral RAE) tub.
- Tape the endotracheal tube to the chin.
- Be sure that the anesthesia ventilation tubing extends over the anterior chest in the midline and then goes off to the side.

Corneal Protection

- Place the ophthalmic eye ointment.
- Place the corneal shields deep to the eyelids.

Medications

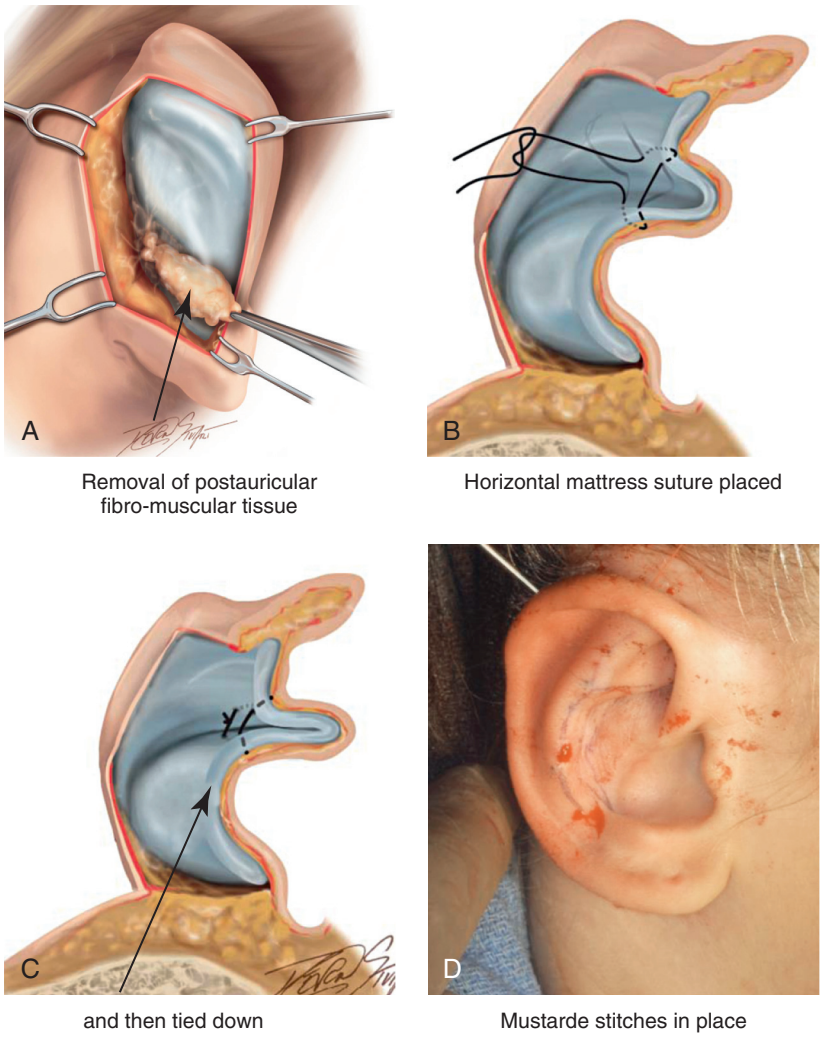
- Administer intravenous antibiotics (Cefazolin 17 mg/kg/dose, max: 1 gm).
- Administer steroids via an intravenous bolus (Dexamethasone 0.5 mg/kg/dose, max: 8 mg).

Preparation and Draping

- If the patient has long hair, tie it in a ponytail; this will keep the hair out of the surgical field.
- Prep the patient's entire scalp, forehead, external ears, face, and neck.
- Use Betadine solution (do not use soap, alcohol, Hibiclens, and so on) to avoid irritation of the mucous membranes and the corneas of the eyes.
- Drape the patient, leaving the anterior scalp, forehead, external ears, face, and neck exposed in the operative field.

Surgical Markings

- Before preparation, use a surgical pen to locate and mark the preferred location of the antihelical fold on the anterior aspect of the ear, including the superior and inferior crus.



• **Figure 39-6** Illustrations and patient example of the surgical correction of an antihelical fold. **A,** Illustration that demonstrates the removal of redundant fibromuscular tissue from the postauricular fold. **B,** Illustration that demonstrates the placement of a Mustarde stitch before tie down (cross section view). **C,** Illustration of a tied-down Mustarde stitch (cross section view). **D,** Intraoperative view of the anterior surface of the ear just after the placement of five Mustarde stitches that were used to create the preferred antihelical fold.

- Before preparation, use a surgical pen to locate and mark the postauricular elliptical skin excision. Center the ellipse at the long axis of the ear from the superior helical rim to the earlobe. Stop the ellipse approximately 1 cm short of the superior and inferior margins of ear.

Local Anesthesia

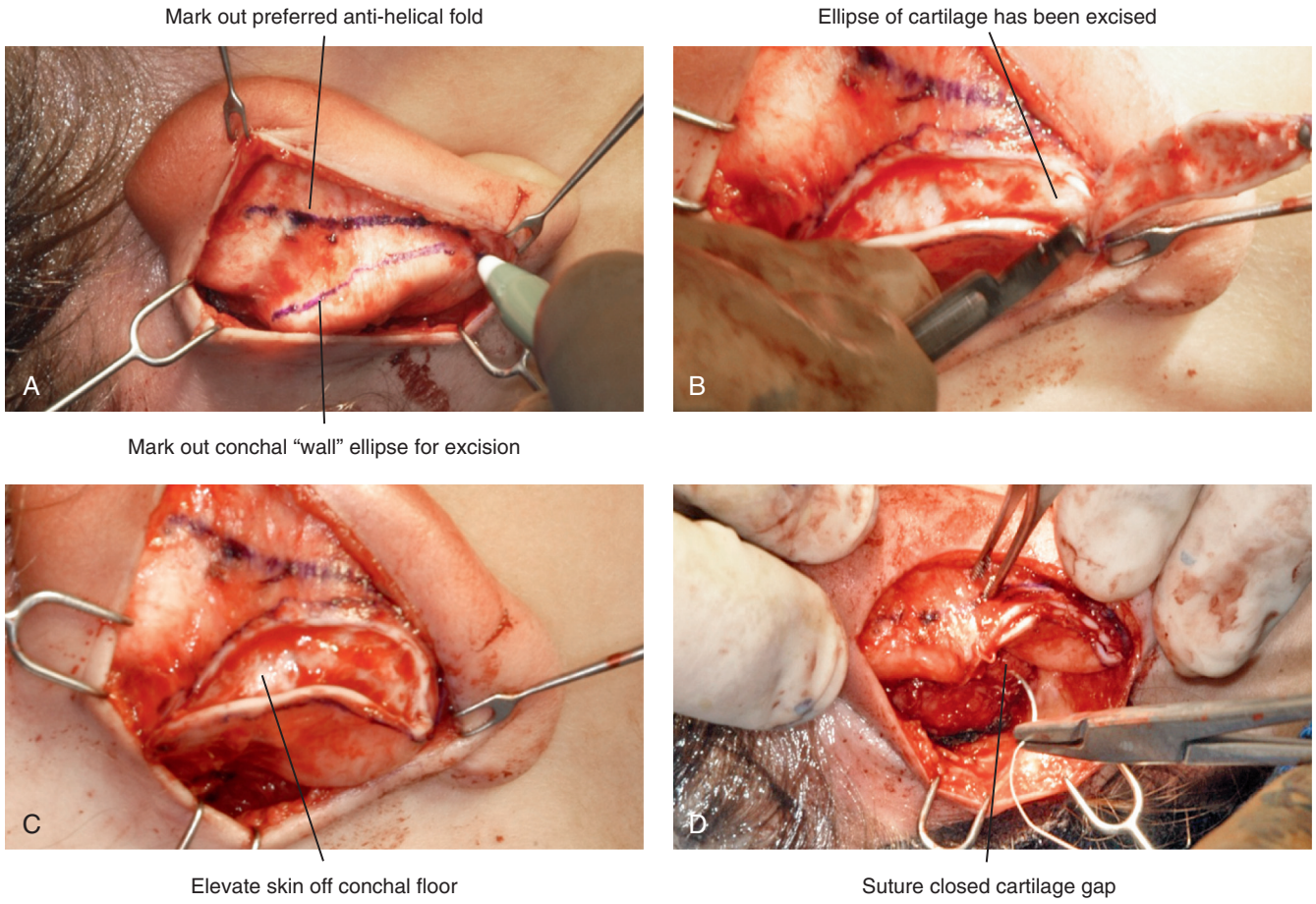
- Inject Xylocaine (1% with 1:100,000 epinephrine) into the postauricular sulcus above the mastoid fascia and superficial to the ear cartilage.

Postauricular Skin Excision (see Fig. 39-3)

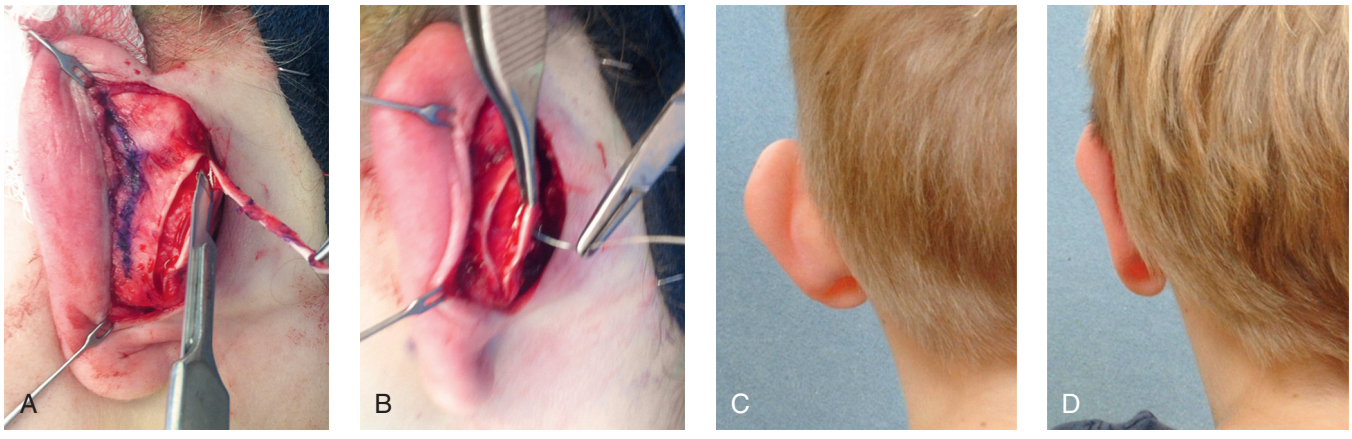
- With a knife (no. 15 blade), incise the full thickness of the skin ellipse.
- With blunt-tipped Stevens scissors, remove the full thickness of the skin ellipse down to the surface of the cartilage.

Elevation of the Postauricular Flaps (see Fig. 39-4)

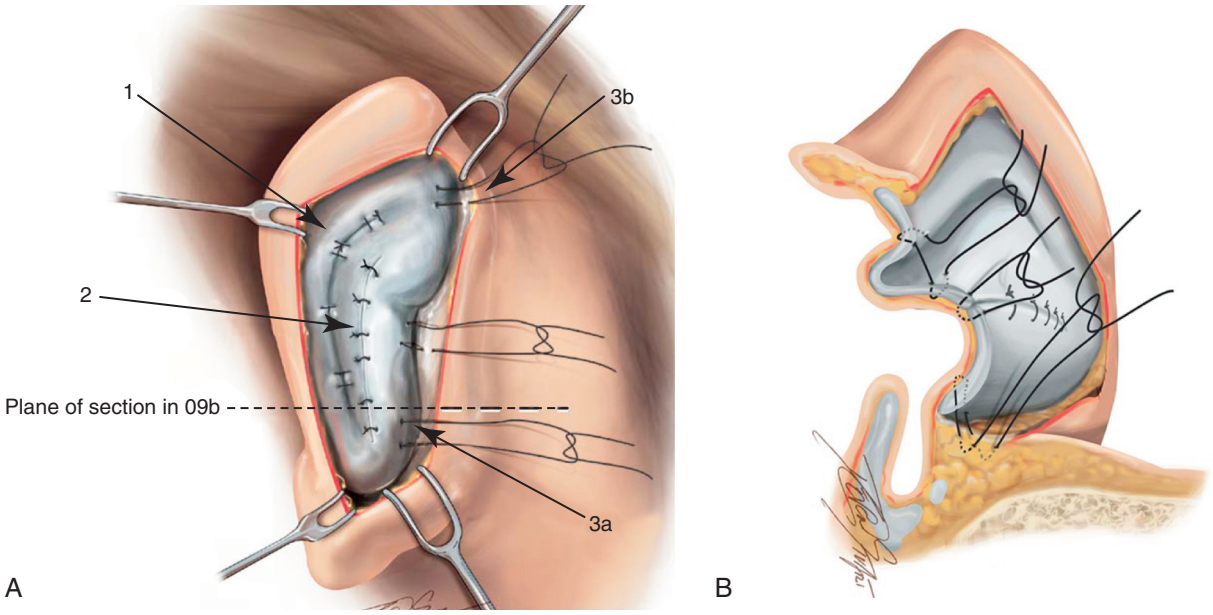
- Elevate the lateral postauricular skin flap near the outer edge of the helical rim.
- Elevate the medial postauricular skin flap down to the mastoid fascia.
- Clean out all soft tissue (i.e., muscle and fibrofatty tissue) in the sulcus down to the mastoid fascia.
- Confirm the adequate elevation of the flaps inferiorly (to the tail of the helix) for later control of the earlobe position and superiorly (the upper aspect of the helix)



• **Figure 39-7** Intraoperative views of conchal wall resection and reconstruction as part of otoplasty. **A,** The preferred antihelical fold is located on the posterior surface of the cartilage and marked with a surgical pen or methylene blue. In addition, the long axis for the planned elliptical excision of the conchal wall is located and marked on the posterior surface of the cartilage. **B,** The full thickness of the conchal wall ellipse for excision is incised through the cartilage with a knife (No. 15 blade) but without perforation through the anterior skin. The cartilage is then removed with the use of a Cottle elevator and Stevens scissors. This image was created after the ellipse of cartilage had been excised. **C,** Next, the anterior ear skin is elevated off of the residual conchal floor toward the ear canal. This is accomplished to limit bunching of the skin during the healing phase after the conchal wall edges are reapproximated with suture. **D,** The edges of the cut conchal wall are sutured together to limit overlap during healing. Note that it is best to suture the conchal edges together only after the Mustarde sutures are placed and tied.



• **Figure 39-8** Illustration and patient example of deep conchal bowl correction. **A,** Illustration of the postauricular approach for access and then for the excision of the ellipse of the conchal wall. **B,** Intraoperative view after the elliptical excision of the conchal wall to "set-back" the ear. The anterior soft tissues of the ear remain intact. **C** and **D,** Clinical example of a child with a deep conchal bowl before and after surgical correction.



• **Figure 39-9** Illustrations of a postauricular approach to the management of a prominent ear. Frequently, three rows of sutures are required for successful ear set-back. **A,** The illustration demonstrates the three rows of sutures being placed, but they are not yet tied down (cross section view). **B,** Illustration of the three rows of sutures (post auricular view) after they are tied down. The three rows of sutures include the following: 1) Mustarde (scaphoconchal) sutures; 2) conchal wall resection and then suture reconstruction of the cartilage defect; and 3) conchal–mastoid and superior helix–mastoid sutures.

for later control of the position of the upper third of the helix.

Marking of the New Antihelical Fold on the Posterior Surface of the Cartilage

- With the use of a 27-gauge needle (i.e., a tuberculin syringe filled with methylene blue), puncture through the anterior surface of the skin along the previously marked location of the new antihelical fold.
- Visualize the needle on the posterior surface of the cartilage, and use a surgical pen or methylene blue to mark the location of the new fold along the full length from the superior crus to the tail. This will allow for the accurate placement of the Mustarde-type sutures.
- Mustarde sutures are not placed until after conchal wall resection is complete.

Conchal Wall Resection for Set-Back (see Figs. 39-7 and 39-8)

- The planned elliptical excision of the conchal wall (superior to inferior) is marked out with a surgical pen or methylene blue on the posterior surface of the cartilage.
- The conchal excision should include a portion of the conchal wall. The full thickness of the ellipse of concha for excision is incised with a scalpel (no. 15 blade) but without perforation through the anterior skin.
- With the use of a Cottle elevator, the incised ellipse of cartilage is freed from the anterior ear skin and then removed.
- The anterior ear skin is further elevated off of the residual concha floor toward the ear canal. This will limit bunching of the skin during the healing phase after the conchal wall edges are reapproximated with suture.
- The edges of the cut conchal wall will be sutured together with 4-0 Vicryl to limit overlap. Wait to suture the edges together until the Mustarde sutures are placed and tied.

Deepening or Creation of the Antihelical Fold (see Figs. 39-5 and 39-6)

- The new antihelical fold is created or deepened by placing three to five carefully located horizontal mattress (Mustarde-type) sutures. The sutures are placed in a natural curve like the spokes of a wheel.
- Experience has taught that cartilage scoring is not necessary and can cause complications (e.g., hematoma, skin flap necrosis, sharp edges).
- Each placed Mustarde suture is tested for effectiveness with a single throw, after which it is loosed.
- When all sutures have been placed and tested, each is carefully tied and confirmed for the preferred aesthetic effect.
- Experience has taught that use of non-resorbable suture (4-0 Mersilene or 4-0 clear nylon) is useful to limit or

prevent late cartilage relapse with the loss of the antihelical fold.

Conchal Wall Resection for Set-Back (Continued)

- Now that the Mustarde sutures have been secured, the cut edges of the conchal wall are approximated with the use of interrupted 4-0 Vicryl sutures to limit overlap.

Placement of Conchal–Mastoid Sutures to Further the Set-Back of the Middle Third of the Ear (see Fig. 39-9)

- Conchal–mastoid sutures are placed as needed only after the conchal wall is resected and reconstructed with sutures and the Mustarde sutures have been placed. The conchal–mastoid sutures should be made of a non-resorbable material (4-0 Mersilene or 4-0 clear nylon) to limit cartilage relapse. They are placed to establish the preferred clockwise rotation of the ear and to further set back the middle concha, as needed.
- If more than one conchal–mastoid suture is required, each is placed and tested with a single throw and then loosened.
- When all of the conchal–mastoid sutures have been placed and tested, each one is tied to confirm the preferred aesthetic effect.

Placement of Superior Helix–Mastoid Sutures to Further the Set-Back of the Upper Third of the Ear

- The superior helical rim may remain "outstanding" more than is preferred even after the placement of Mustarde sutures. A superior helical–mastoid suture (4-0 Mersilene or 4-0 clear nylon) is often useful to further improve the aesthetic result. The combination of these surgical maneuvers will limit a sharp edge in the antihelical fold that may otherwise occur as a result of the overtightening of the Mustarde sutures.

Set-Back of the Earlobe (Lower Third of Ear)

- Excision of the postauricular skin and the subcutaneous tissue of the lobe is sometimes helpful to set back the lower third of the ear.
- The placement of a tail of helix–mastoid or tail of helix–conchal base suture with the use of non-resorbable material (4-0 Mersilene or 4-0 clear nylon) is sometimes helpful to control the lobe position.

Skin Closure

- The placement of several interrupted sutures and then a running and locking suture of resorbable material (5-0 chromic) is used for skin closure. This prevents the need for postoperative suture removal.

Placement of Dressing (Fig. 39-10)

- A thick coat of antibiotic ointment is placed in the postauricular fold and over the anterior ear skin surface.
- A sheet of gauze (Xeroform) is placed in the postauricular groove and also along the anterior ear surface.
- Two or three pieces of dry fluff gauze are placed over the anterior skin surface of each ear.
- A gauze roll (Kerlix) is used as a wrap to apply gentle pressure and to hold the gauze fluffs in place. The Kerlix wrap is also secured across the anterior neck to prevent the head dressing from loosening.
- An Ace wrap is then loosely placed as an outer layer of the head and ear dressing.
- The whole dressing is removed 5 days later.

Postoperative Care

- After the ear dressing is removed, the patient is encouraged to shower and shampoo the hair and to wash the external ears, face, and neck one or two times daily. (Overly hot water should be avoided, because the ears will have diminished sensation.)
- The ears are dabbed dry after washing. (Excess rubbing of the ears should be avoided.)
- Ear protection is used when sleeping for 2 to 3 months to prevent accidental anterior folding of the external ears. A skier's ear warmer or a tennis sweatband may be used for this purpose.
- No vigorous sports activities are allowed for 4 to 6 weeks.
- No strong sun (ultraviolet) exposure to the ears is allowed for approximately 3 months.
- When the patient has returned to more regular physical or sports activities after 4 to 6 weeks, additional precautions are encouraged, depending on type of sport activity (e.g., wrestling, basketball), during the initial 3 months after surgery.

Assessment of Results

Although prominent ears do not have any physiologic disadvantages, they play an important role in social life. Prominent ears often provoke teasing by others, especially by children; they can lead to emotional stress with a decline in the patient's health-related quality of life; they may negatively affect school or job performance; and they may result in social avoidance and a loss of self-confidence (Figs. 39-11 through Fig. 39-27). Today, health-related quality of life is considered by many to be the most important parameter for the evaluation of aesthetic procedures such as the correction of protruding ears.[7,64]

In a recent study by Braun and colleagues, a consecutive series of patients (N = 84) who underwent otoplasty (suture technique) were given a health-related quality-of-life survey.[6]

The survey used was either the Glasgow Children's Benefit Inventory (N = 41; 73.8% of study group) or the Glasgow Benefit Inventory (N = 21; 33.9% of study group). Both of these quality-of-life surveys have been validated for measuring the effect of plastic (aesthetic) operations on health-related quality of life. Sixty-two patients (73.8%) returned a valid questionnaire for analysis. For the study group, the main reasons for carrying out the ear set-back operations as reported by the patient and the family included teasing (42.6%); aesthetic impairment (39.3%); and reduced self-confidence (23%). The results of the study indicate that 100% of the adults were satisfied with their aesthetic results and that 90.5% would again decide in favor of the operation. Ninety-five percent of the parents of the children and 95.1% of the children themselves were satisfied with the aesthetic results. Ninety-seven percent of the parents and 92.7% of the children then also stated that they would again decide in favor of the operation. The authors also speculated that an individual's reluctance to participate in certain social activities, if caused by reduced self-confidence as a result of the protruding ears, was more likely to remain constant with increased age. In other words, adults that had lived through the social ridicule of prominent ears for many years were more likely to retain reduced self-confidence even after successful ear set-back.

Complications and Suboptimal Aesthetic Results

Although the majority of patients state that they are satisfied with their otoplasty results and say that they would undergo such procedures again, complications and suboptimal results will always occur in a minority of cases.[10,17,31,37,53,54,63]

Pinching of the External Auditory Canal

Conchal-mastoid sutures that are placed too far forward on the mastoid or too far back on the concha may exaggerate the conchal rotation. In theory, the created angular edge of the auditory canal may diminish the diameter of the canal. This problem can be minimized by managing conchal hypertrophy with elliptical excision rather than with the overaggressive use of conchal–mastoid sutures.

Inadequate Management of Conchal Hypertrophy

Many patients who are dissatisfied with the aesthetic results of an otoplasty demonstrate residual conchal hypertrophy that was missed, ignored, or undertreated. When conchal hypertrophy is part of the deformity, it should be adequately addressed. This author prefers the elliptical resection of the outstanding conchal wall in most cases. The placement of conchal–mastoid sutures alone is often inadequate and results in either immediate or late relapse.

A "Too-Sharp" Antihelical Fold

An unnaturally sharp antihelical fold may be created as a result of overaggressive scoring of the cartilage or overtightening of the Mustarde sutures in the presence of undercorrected conchal hypertrophy.

Text continued on p. 1730

Just prior to otoplasty

5 days after otoplasty, day of dressing removal

• **Figure 39-10** A 5-year-old with prominent ears underwent reconstruction. He is shown before and 5 days after reconstruction at the time of bandage removal. After bandage removal, the patient was allowed to wash the hair, face, and ears in the shower with soap and water. Other than avoiding direct trauma or forward folding of the ears while sleeping, the ears are then left open to the air.

©Elsevier Inc.
Orthognathic Surgery; J.C. Posnick

• **Figure 39-11** A 4-year-old girl with asymmetric prominent ears that are characterized by conchal hypertrophy and deficient scaphal folding. She underwent elliptical conchal wall resection (posterior approach); conchal rotation and stabilization (conchal–mastoid and superior helix–mastoid sutures); and the creation of improved antihelical folds (Mustarde sutures). **A,** Frontal views before and after reconstruction. **B,** Oblique facial views before and after reconstruction.

Continued

• **Figure 39-11, cont'd C,** Profile views before and after reconstruction. **D,** Posterior facial views before and after reconstruction.

©Elsevier Inc.
Orthognathic Surgery; J.C. Posnick

• **Figure 39-12** A 7-year-old girl with asymmetric prominent ears that are characterized by both conchal hypertrophy and deficient scaphal folding. She underwent elliptical conchal wall resection (posterior approach); conchal rotation and stabilization (conchal–mastoid and superior helix–mastoid sutures); and the improvement of the antihelical folds (Mustarde sutures). **A,** Frontal views before and after reconstruction. **B,** Oblique facial views before and after reconstruction. **C,** Profile views before and after reconstruction.

©Elsevier Inc.
Orthognathic Surgery; J.C. Posnick

• **Figure 39-13** A 5-year-old boy with asymmetric prominent ears that are characterized by both conchal hypertrophy and deficient scaphal folding. He underwent elliptical conchal wall resection (posterior approach); conchal rotation and stabilization (conchal–mastoid and superior helix–mastoid sutures); and the improvement of the antihelical folds (Mustarde sutures). **A,** Frontal views before and after reconstruction. **B,** Oblique facial views before and after reconstruction.

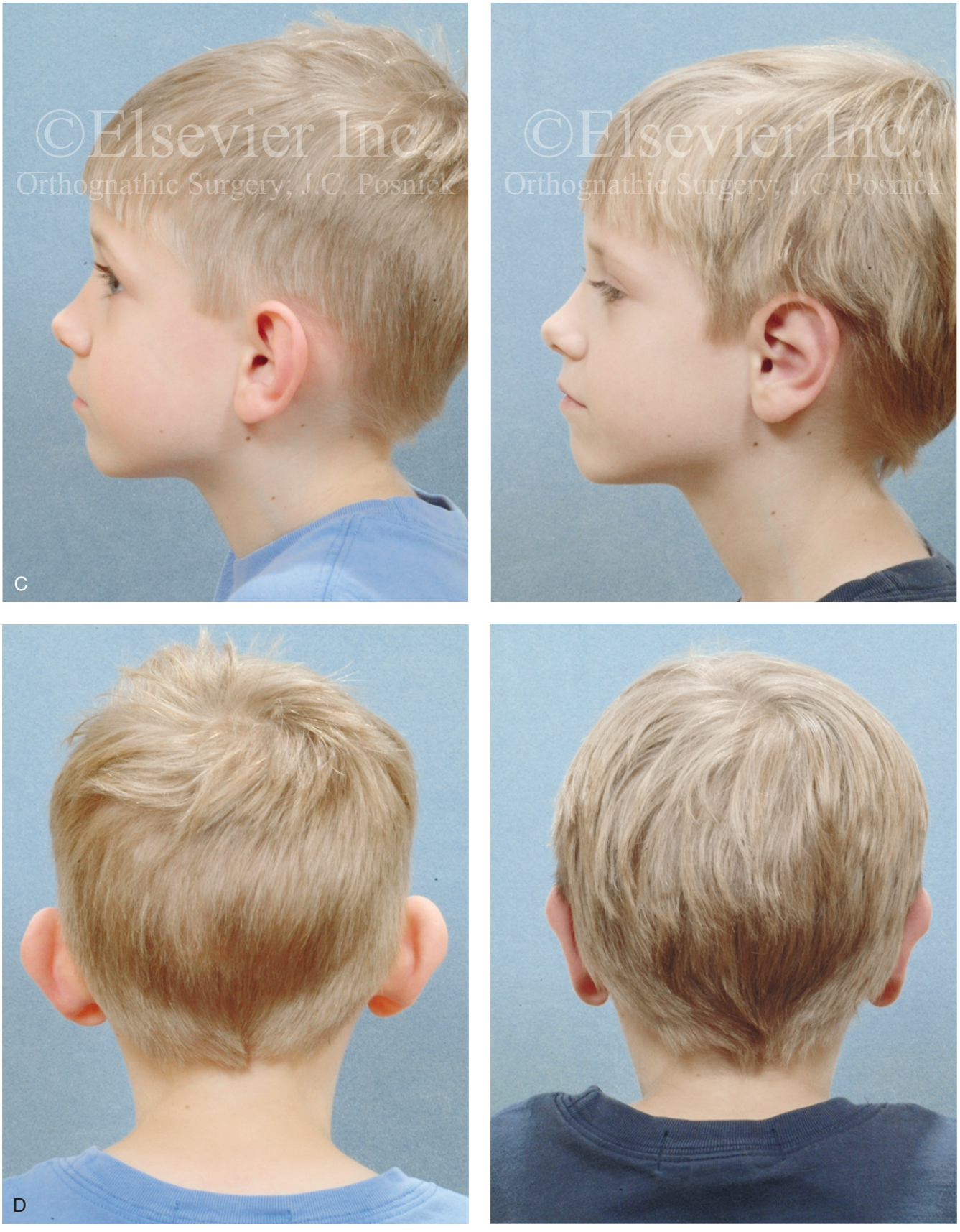
• **Figure 39-13, cont'd** **C,** Profile views before and after reconstruction. **D,** Posterior views before and after reconstruction.

©Elsevier Inc.
Orthognathic Surgery; J.C. Posnick

• **Figure 39-14** A 5-year-old boy with asymmetric prominent and cupped ears that are characterized by conchal hypertrophy, deficient scaphal folding, and a prominent earlobe. He underwent conchal wall resection (posterior approach); conchal rotation and stabilization (conchal–mastoid sutures); improvement of the antihelical folding (Mustarde sutures); and earlobe set-back. **A,** Frontal views before and after reconstruction. **B,** Oblique facial views before and after reconstruction.

• **Figure 39-14, cont'd** **C,** Profile views before and after reconstruction. **D,** Posterior facial views before and after reconstruction.

• **Figure 39-15** A 5-year-old boy with asymmetric prominent ears that are characterized by conchal hypertrophy and inadequate scaphal folding. He underwent conchal wall resection (posterior approach); conchal rotation and set-back (conchal–mastoid sutures); and deepening of the antihelical folding (Mustarde sutures). **A,** Frontal views before and after reconstruction. **B,** Oblique facial views before and after reconstruction.



• **Figure 39-15, cont'd** **C,** Profile views before and after reconstruction. **D,** Posterior views before and after reconstruction.

• **Figure 39-16** A 5-year-old boy with small and prominent ears that are characterized by the absence of scaphal folding and deep conchal hypertrophy. He underwent elliptical conchal wall resection (posterior approach); conchal rotation and stabilization (conchal–mastoid sutures); and the creation of antihelical folds (Mustarde sutures). He returned 6 months after surgery with chronic irritation in the right postauricular fold. A stitch granuloma was debrided, and he went on to develop a localized hypertrophic scar in the same region. One year after surgery, the scar and inflammatory issue were excised, with primary closure. Healing was uneventful, and no other treatment was required. **A,** Frontal views before and after reconstruction. **B,** Profile views before and after reconstruction.

• **Figure 39-16, cont'd C,** Posterior views before and after reconstruction. **D,** Right ear postauricular view that shows the inflammatory tissue hypertrophic scar just before excision. **E,** Facial views 3 years after reconstruction and 2 years after scar tissue excision.

• **Figure 39-17** A 7-year-old boy with asymmetric prominent and cupped ears underwent reconstruction. This required limited elliptical conchal wall resection (posterior approach); conchal rotation and stabilization (conchal–mastoid and superior helix–mastoid sutures); improvement of the antihelical folding (Mustarde sutures); and earlobe set-back (tail of the helix–mastoid sutures). **A,** Frontal views before and after reconstruction. **B,** Oblique facial views before and after reconstruction. **C,** Profile views before and after reconstruction.

©Elsevier Inc.
Orthognathic Surgery; J.C. Posnick

• **Figure 39-18** An 11-year-old girl with asymmetric prominent ears that are characterized by conchal hypertrophy and inadequate scaphal folding underwent reconstruction. The procedures included conchal rotation and set-back (conchal–mastoid sutures) and the creation of improved antihelical folding (Mustarde sutures). **A,** Frontal views before and after reconstruction. **B,** Oblique facial views before and after reconstruction. **C,** Profile views before and after reconstruction. Note that there is a degree of incomplete correction or relapse of both the conchal and superior helix set-back of the right ear.

©Elsevier Inc.
Orthognathic Surgery; J.C. Posnick

• **Figure 39-19** A high-school student with prominent ears that are characterized by conchal hypertrophy and deficient scaphal folding. She underwent elliptical conchal wall resection (posterior approach); conchal rotation and stabilization (conchal–mastoid sutures); and the creation of an improved antihelical fold (Mustarde sutures). **A,** Frontal views before and after reconstruction. **B,** Oblique facial views before and after reconstruction. **C,** Profile views before and after reconstruction. Note the overly prominent antihelical crease of the right ear.

©Elsevier Inc.
Orthognathic Surgery; J.C. Posnick

• **Figure 39-20** A high school student with prominent ears that are characterized by conchal hypertrophy and deficient scaphal folding. She underwent elliptical conchal resection (posterior approach); conchal rotation and stabilization (conchal–mastoid sutures); and the creation of an improved antihelical fold (Mustarde sutures). **A,** Frontal views before and after reconstruction. **B,** Oblique facial views before and after reconstruction. **C,** Profile views before and after reconstruction.

• **Figure 39-21** A 9-year-old girl with a unilateral (right-side) prominent ear that is characterized by conchal hypertrophy and a lack of scaphal folding. She underwent a unilateral otoplasty. The ear cartilage was extremely thin and flexible. She underwent conchal rotation and set-back (conchal–mastoid sutures) and the creation of antihelical folding (Mustarde sutures). **A,** Frontal views before and after reconstruction. **B,** Right profile views before and after reconstruction.

Excessively Prominent Earlobe

After the traditional surgical management of the antihelical fold, one area of the prominent ear that may require additional attention is the earlobe. At times, this problem can be addressed with additional posterior skin excision. If the lobe still projects forward and laterally, consideration should be given to the removal of cartilage deep to the antitragus (cavum conchalis) to allow the lobe of the ear to be aesthetically retropositioned. This may be carried out as an extension of the conchal bowl excision. For others, the prominent earlobe is managed with suturing of the tail of the helix to either the mastoid fascia or the conchal base.

Failure (Relapse) of Scaphal (Upper Helix) Folding

In some patients, the created antihelical fold will open (relapse) with recurrence of the prominent ear appearance. This may occur for one of several reasons. If a Mustarde stitch becomes inflamed or infected, it may loosen, which results in the unfolding of the helix. In other situations, an

• **Figure 39-22** A 9-year-old boy with malformed and asymmetric external ears who underwent reconstruction. His procedures included asymmetric elliptical conchal wall resection (posterior approach); conchal rotation and stabilization (conchal–mastoid and superior helix–mastoid sutures); the creation of the antihelical fold (Mustarde sutures); and earlobe set-back (soft-tissue sutures). **A,** Frontal views before and after reconstruction. **B,** Profile views before and after reconstruction.

uncooperative child may pull on the ears, thereby causing shearing of the sutures with a recurrence of ear prominence. For others, the sutures may not have a sufficient "bite" into the cartilage and thus not withstand the recoil forces. In this author's experience, the use of non-resorbable (4-0 Mersilene or 4-0 clear nylon) suture is an important aspect for the limiting of relapse tendency.

In some patients, despite the placement of Mustarde stitches, the upper helix still protrudes. If residual protrusion of the upper third of the helix is observed early after surgery, it is most likely an intraoperative technical error rather than cartilage relapse. To prevent this problem during operation, rather than overtightening the Mustarde stitches, it is best to place a superior helix–mastoid suture.

Residual Ear Asymmetry

When managing unilateral or bilateral ear prominence, the preoperative assessment should clarify any baseline asymmetry of the auricles. Each ear should be treated indepen-

Text continued on p. 1739

• **Figure 39-23** A woman in her mid 20s with prominent ears that are characterized by a mild degree of conchal hypertrophy and deficient scaphal folding. She underwent elliptical conchal wall resection (posterior approach); conchal rotation (conchal–mastoid sutures); and the creation of the antihelical fold (Mustarde sutures). She is shown before and just 2 weeks after surgery. At this stage of healing, residual edema and skin discoloration remain. **A,** Frontal views before and 2 weeks after surgery. **B,** Oblique facial views before and 2 weeks after surgery. **C,** Profile views before and 2 weeks after surgery.

• **Figure 39-24** A high school student with prominent ears that are characterized primarily by conchal hypertrophy requested setback. He underwent conchal wall resection (posterior approach); conchal rotation and stabilization (conchal–mastoid sutures); and stabilization of the antihelical folding (Mustarde sutures). **A,** Frontal views before and after reconstruction. **B,** Oblique facial views before and after reconstruction. *Continued*

• **Figure 39-24, cont'd** **C,** Profile views before and after reconstruction. **D,** Posterior views before and after reconstruction.

• **Figure 39-25** A middle-aged man with a lifelong history of prominent ears that are characterized by limited antihelical folding and a degree of conchal hypertrophy. He underwent conchal wall resection (posterior approach); conchal stabilization (conchal–mastoid sutures); and deepening of the scaphal folding (Mustarde sutures). **A,** Frontal views before and after reconstruction. **B,** Oblique facial views before and after reconstruction. **C,** Profile views before and after reconstruction.

• **Figure 39-26** A high school student who was born with hemifacial microsomia underwent simultaneous jaw and external ear reconstruction (see Fig. 28-15). Although the ears are asymmetric in morphology and position, they were simply set back. The left ear underwent both conchal wall resection (posterior approach) and improvement of the scaphal fold (Mustarde stitches). The right ear required improvement of the scaphal fold (Mustarde stitches) and the placement of superior helix–mastoid sutures. **A,** Frontal views in repose before and after reconstruction. **B,** Frontal views with smile before and after reconstruction.

• **Figure 39-26, cont'd C,** Left oblique facial views before and after reconstruction. **D,** Right oblique facial views before and after reconstruction.

• **Figure 39-27** A 5-year-old child with Treacher Collins syndrome underwent zygomatic–orbital reconstruction and bilateral external otoplasty (see Fig. 27-11). The external ear procedures included elliptical conchal wall resection (posterior approach); conchal rotation and stabilization (conchal–mastoid suture); and the improvement of the antihelical folds (Mustarde sutures). **A,** Frontal views before and after reconstruction. **B,** Oblique facial views before and after reconstruction. **C,** Worm's-eye views before and after reconstruction.

dently but with consideration of overall facial symmetry and balance. Ear asymmetry is easily glossed over preoperatively; however, when scrutinizing the ears after surgery, even minor asymmetry may take on greater importance. Good-quality facial photographs before and soon after surgery should be reviewed with the patient and the family to avoid misunderstandings.

Postsurgical "Telephone Ear" Appearance

The "telephone ear" deformity is caused by overaggressive set-back (antihelical folding) in the middle third of the helical rim in combination with the undercorrection of the earlobe and the superior third of the helix. This generally occurs when conchal hypertrophy has not been appropriately addressed.

Postsurgical "Flat Ear" Appearance

The postsurgical "plastered back" appearance of the external ears occurs when the antihelical fold is sharp and excessive in combination with the overreduction of the conchal wall.

Infection of the Cartilage (Chondritis)

Infection after ear surgery is not common. Chondritis is more likely after aggressive anterior flap elevation and cartilage scoring, especially when contamination occurs.

Necrosis with Loss of Skin

Necrosis is more likely to occur with wide anterior flap undermining when inadequate attention has been given to circulation requirements. It may also occur as a result of an overly tight ear dressing or bandage, a hematoma beneath the dressing, or a combination of these factors.

Abnormal Scar Formation

Hypertrophic scarring can occur at any wound closure. It is more likely in the presence of infection or poor circulation or with excessive scratching or rubbing by the patient postoperatively. Keloid formation can occur and may be differentiated from hypertrophic scarring by a positive personal or family history and scar enlargement that extends outside of the operative site. Current options for the management of a keloid scar are often suboptimal but may include intralesional steroid injections, cutaneous irradiation, or re-excision.

Suture Granuloma ("Stitch Abscess")

Occasionally a buried suture will develop a granulomatous reaction around it. The offending suture will either spontaneously exfoliate or require surgical removal. This seems to be more frequent with Mersilene suture material. Removal under local anesthesia can be difficult in a child.

Non-Surgical Correction of Prominent Ears in the Newborn

Although the idea of "binding" the external ears in an attempt to correct abnormalities is not a new one, Matsuo and colleagues can be credited with examining the efficacy of doing so in newborns.[40] Byrd and colleagues convincingly demonstrated that there are particular auricular deformities in the newborn that are amenable to molding back toward normal.[9] In their publication, the types of deformities that can be most effectively treated and details concerning methods of molding are discussed. Auricular deformations are characterized by a misshapen but fully developed pinna.[65,66] Ear molding techniques are best suited for deformations rather than malformations.[8,12,29,36,41,43,45,59,68,71] Interestingly, approximately one third of the ear deformations that are present at the time of birth self-correct during the first week of life. The patterns of deformation that are frequently seen include the following: prominent/cupped ears; lidded/lopped ears; Stahl (or Spock) ears; helical rim deformities (with an absent or deficient rim); and conchal crus deformities.[49,51,58] Byrd and colleagues found that birth deformations of the auricle more often than not involve the upper third of the ear, with incomplete formation of the superior limb of the triangular fossa being a frequent finding. This leads to a helical rim scapha deformation. The authors stressed the importance of accomplishing three key molding forces to correct the majority of these ear deformations in newborns:

1. A stent or conformer that rests along the retroauricular sulcus and that is in alignment with the antihelix is placed.
2. A second conformer that is curved to match the natural shape of the helical rim is used. It is placed anteriorly but with force directed against the scapha. The anterior conformer is carefully located to avoid excess direct pressure against the posterior conformer for the avoidance of skin necrosis.
3. As the helix is retracted, the molding technique is also used to expand the rim to its full dimension.

The authors described good to excellent results in 90% of the newborns whose external ear deformities were treated in this way. Complications were seen in only approximately 5% of patients and were mostly characterized as localized skin excoriations. The authors recommend withholding earmolding therapy until the end of the first week to identify those infants whose ears spontaneously self-correct soon after birth. It is believed by most that, for treatment to be effective, it should be instituted by the second or (at most) third week

of life. Only approximately half of the infants treated by Byrd and colleagues after 3 weeks of age had good outcomes. The reason for this is believed to be related to the level of hyaluronic acid, which is an important component of ear cartilage.[27] Hyaluronic acid is increased by estrogen and believed to be responsible for the malleable nature of the neonatal ear. The circulating estrogen levels are known to decrease rapidly to levels that are similar to those of older children by 6 weeks of age.[33] It is also known that, with breast-feeding, the maternal transfer of estrogen extends the time frame of higher hyaluronic acid levels, which likely explains the variations in the effectiveness of ear molding in some children; this may also leave the ears prone to relapse after molding treatment is stopped unless the result is maintained.

Conclusions

There are wide variations with regard to what is considered normal for ear size, prominence, and shape. However, when the ears stick out excessively, it may become a source of ridicule and personal anguish for the affected individual. The successful surgical alteration of the prominent ear requires accurate diagnosis; the setting of realistic expectations and the clear communication of those expectations; meticulous operative technique; and careful postoperative management. Attention to detail and a sense for facial aesthetics helps the surgeon to achieve a natural appearance for the reconstructed ear with an acceptable incidence of complications and high level of patient satisfaction.

Patient Education Materials

Otoplasty (Reshaping of the Ears) Surgery

Although a wide variation in the shape and size of the ears naturally occurs from person to person, ears that stick out make individuals easy targets for teasing and ridicule. For most individuals, after this occurs, the sooner this problem is corrected, the better. *Otoplasty* is the name of the surgical procedure that is carried out to diminish ear projection or to change the appearance of the ears. It may be carried out as early as the ears approach their adult size (i.e., around the age of 3 to 5 years) or at any time thereafter. Each individual who is seeking otoplasty has unique concerns. It is important to fully discuss your expectations with your surgeon before surgery.

In general, the technical aspects of the otoplasty procedure include an incision that is made behind each ear to provide access to the ear cartilage. The cartilage of the ear is then reshaped to create improved curvature and position. The procedure is carried out on an outpatient basis with the patient under anesthesia in the operating room. With the ears reshaped and set back, a bandage is used to secure the new ear position and to limit swelling. The bandage generally remains in place for 5 days. After this, return to school or work is rapid. An extra level of care of the ears is required during the initial 4 to 6 weeks of healing.

INSTRUCTIONS FOR OTOPLASTY SURGERY

The purpose of these instructions is to help you prepare for and then recover from otoplasty with as little discomfort and inconvenience as possible.

Preoperative Instructions

1. Do not take any aspirin or aspirin-like products (e.g., Advil) for at least 2 weeks before surgery. Aspirin and aspirin-like products tend to increase bleeding during surgery and bruising postoperatively.
2. Starting 2 days before surgery, we request that you shower and shampoo your hair and wash your face at least once per day. The morning of surgery, shower and shampoo again, if feasible.
3. Do not eat or drink anything after midnight the night before surgery, not even a sip of water.
4. We will give you a prescription for pain control and another for antibiotics to be used after surgery.
5. Be sure to make arrangements with someone to accompany you home after surgery, because you will not be allowed to drive.

Postoperative Instructions

1. It is preferred that you keep the head of your bed elevated when resting and sleeping for the first week after surgery.

2. Take the prescribed antibiotics as ordered and the pain medication as needed.
3. You may eat whatever you wish, but start out slowly, because the anesthesia may leave your stomach unsettled for several hours.
4. Call the office for an appointment on the date specified at the time of discharge.
5. If there are any concerns while you are at home, contact your surgeon's office at any time.
6. Before you arrive for your first office visit after surgery, purchase and bring along an elastic cloth ear band to be used at night when you are sleeping. Your surgeon will instruct you with regard to its use.

Jeffrey C. Posnick, DMD, MD
Director, Posnick Center for Facial Plastic Surgery

Consent for Otoplasty Surgery

_____ Initial here

INSTRUCTIONS

This document has been prepared to help your surgeon to educate and inform you about your otoplasty surgery and its risks as well as alternative treatment options. It is important that you read this document carefully and completely. Please initial each section on each page to indicate that you have read and understand it. In addition, please sign the consent for surgery on the last page as proposed by your surgeon.

_____ Initial here

INTRODUCTION

Otoplasty is a surgical process that is used to reshape the ears. A variety of different techniques and approaches may be used to reshape congenitally prominent ears or to restore damaged ears. Each individual who is seeking otoplasty is unique both in terms of the appearance of his or her ears and his or her expectations of the results that will be obtained with surgery. It is important that you fully discuss your expectations with your surgeon before surgery.

_____ Initial here

ALTERNATIVE TREATMENTS

Alternative forms of management consist of not undergoing an otoplasty operation at all or proceeding with other more limited or more extensive surgical options.

_____ Initial here

RISKS OF SURGERY

Every surgical procedure involves a certain amount of risk. An individual's choice to undergo an otoplasty procedure is based on the comparison of the risk to the potential benefit. Although the majority of patients do not experience the complications detailed below, you should discuss each of them with your surgeon. Make sure that you understand all of the possible consequences of the otoplasty procedure that is planned for you.

_____ Initial here

BLEEDING

It is uncommon to experience significant bleeding during or after otoplasty surgery. If postoperative bleeding does occur, it may require emergency treatment to drain accumulated blood (i.e., hematoma). Do not take any aspirin or similar anti-inflammatory medications for 2 weeks before or after surgery, because this may increase the risk of bleeding.

Continued

_____ **INFECTION**

Initial here Infection is not common after otoplasty surgery. If an infection does occur, treatment that includes the extended use of antibiotics or additional procedures may be necessary.

_____ **CHANGES IN SKIN SENSATION**

Initial here Diminished sensation in the skin of the ear will occur and may not totally resolve after otoplasty surgery.

_____ **TRAUMA**

Initial here Physical injury to the ear soon after otoplasty may disrupt the results of surgery. Care must be taken by you to protect the affected ear from injury during the healing process. Additional surgery may be possible to correct any damage that does occur.

_____ **SKIN CONTOUR IRREGULARITIES**

Initial here Contour irregularities as well as visible and palpable wrinkling of skin and ear cartilage can occur after otoplasty.

_____ **SKIN SCARRING**

Initial here In some cases, excessive scars may result after otoplasty. Scars may be unattractive and of a different color than the surrounding skin. Additional treatments or procedures may be possible to treat unfavorable scarring.

_____ **SURGICAL ANESTHESIA**

Initial here Both local and general anesthesia involve risk. Complications and injuries are possible with all forms of surgical anesthesia and sedation.

_____ **ASYMMETRY**

Initial here The human face is normally asymmetric, and this is especially true for the external ears. Typically, there are differences between ears in terms of shape and size. There will also be variations from one side to the other with regard to the results obtained with an otoplasty procedure.

_____ **DELAYED HEALING**

Initial here Wound disruption or delayed healing is possible after otoplasty. Some areas of the ear may heal abnormally or slowly. Occasionally, further procedures to manage the non-healed tissue may be required.

_____ **ALLERGIC REACTIONS**

Initial here Local allergies to tape, suture material, or topical preparations can occur. Systemic reactions, which are more serious, may also occur in response to the drugs used during surgery and prescription medicines. Allergic reactions may require additional treatment.

_____ **AGING EFFECTS**

Initial here Subsequent alterations in ear appearance may occur as a result of aging or other circumstances not related to the otoplasty surgery.

_____ **RELAPSE**

Initial here As a result of the resilient nature of ear cartilage, relapse (i.e., the springing back out of the ears) may occur after surgery. Revisional surgery may be possible to improve the results if relapse occurs.

_____ **PAIN**

Initial here Infrequently, chronic pain may occur as a result of nerves being injured or trapped in scar tissue after an otoplasty.

_____ **DEEPER SUTURES**

Initial here Deep (buried) non-absorbable sutures may spontaneously work their way through the skin, become visible, or produce irritation and inflammation and thus require removal.

_____ **UNSATISFACTORY RESULTS**

Initial here You may be disappointed with the results of surgery. If so, it may be possible to perform additional surgery to improve your satisfaction.

_____ **ADDITIONAL SURGERY**

Initial here The practice of medicine and surgery is not an exact science. Although good results are expected, there is no guarantee or warranty expressed or implied with regard to the results that may be obtained. If complications do occur, additional surgery or other treatment may be possible.

_____ **HEALTH INSURANCE**

Initial here Most health insurance companies exclude coverage for operations to change the shape of the external ears (otoplasty) or any complications that may occur in association with this surgery. Please carefully review your health insurance subscriber-information pamphlet to clarify these issues.

_____ **FINANCIAL RESPONSIBILITIES**

Initial here The cost of surgery involves several charges for the services provided, including the fees charged by your surgeon (i.e., surgeon's fees); the cost of surgical supplies and the use of the operating room (i.e., facility/hospital charges); and the costs associated with anesthesia (i.e., anesthesiologist's fees). Depending on whether the cost of surgery is covered by your medical insurance plan, you will be responsible for necessary copayments, deductibles, and all other charges that are not covered. Additional costs may occur should complications develop from the surgery. Any secondary surgeries and all charges involved with additional procedures (i.e., surgeon's fees, facility/hospital charges, anesthesiologist's fees) would also be your responsibility.

_____ **DISCLAIMER**

Initial here Informed-consent documents are used to communicate information about the proposed surgical treatment as well as to disclose information about risks and alternative forms of treatment. The informed-consent process attempts to define principles of risk disclosure that should generally meet the needs of most patients in most circumstances.

Because every patient is unique, this informed-consent document should not be considered all-inclusive with regard to defining other methods of treatment to consider and potential risks encountered. Your surgeon will provide you with additional written and verbal information that is based on all of the facts of your particular case and the current state of medical knowledge.

Informed-consent documents are not intended to define or serve as the standard of medical care. Standards of medical care are determined on the basis of all of the facts involved in an individual case and are subject to change as scientific knowledge and technology advance and practice patterns evolve.

Continued

CONSENT FOR OTOPLASTY SURGERY

1. I hereby authorize my surgeon, _____, and such assistants as may be selected by my surgeon to perform the following procedure: otoplasty.
2. I have received, read, and understand the following information sheets:
   - Otoplasty (Reshaping of the Ears) Surgery
   - Book chapter about otoplasty
   - Consent for Otoplasty Surgery
3. I recognize that, during the course of the operation and the administration of anesthesia, unforeseen conditions may necessitate different procedures than those stated above. I therefore authorize my surgeon, _____, and my surgeon's assistants or designees to perform such other procedures that are in the exercise of professional judgment necessary and desirable. The authority granted under this paragraph shall include all conditions that require treatment and that are not known to my surgeon at the time that the procedure is begun.
4. I consent to the administration of such anesthetics as are considered necessary or advisable. I understand that all forms of anesthesia involve risk and the possibility of complications, injury, and even death.
5. I acknowledge that no guarantee has been given by anyone with regard to the results that may be obtained from this procedure.
6. I consent to the photographing of me (head and neck region) before, during, and after surgery. These photographs may be used by my surgeon for medical, scientific, or educational purposes now and in the future.
7. For the purposes of advancing medical education, I consent to the admittance of observers into the operating room.
8. I consent to the disposal of any body tissues or medical devices that may be removed.
9. **THE FOLLOWING INFORMATION HAS BEEN EXPLAINED TO ME IN A WAY THAT I UNDERSTAND:**
   - **THE PREVIOUSLY DESCRIBED PROCEDURES TO BE UNDERTAKEN**
   - **ALTERNATIVE PROCEDURES AND METHODS OF TREATMENT THAT I HAVE DECIDED AGAINST**
   - **THE SPECIFIC RISKS AND POTENTIAL COMPLICATIONS OF THE PROCEDURES THAT I PLAN TO UNDERGO**

I CONSENT TO THE PROCEDURES AND THE ABOVE LISTED ITEMS (1 THROUGH 9). I AM SATISFIED WITH THE EXPLANATION GIVEN TO ME.

_____	_____
Patient (or Person Authorized to Sign for Patient)	Date
_____	_____
Witness	Date

References

1. Adamson JE, Horton CE, Crawford HH: The growth pattern of the external ear. *Plast Reconstr Surg* 36:466, 1965.
2. Adamson PA, McGraw BL, Tropper GJ: Otoplasty: Critical review of clinical results. *Laryngoscope* 101:883, 1991.
3. Barnes WE, Morris FA: Otoplasty: An improved technique. *South Med J* 59:681, 1966.
4. Bauer BS, Margulis A, Song DH: The importance of conchal resection in correcting the prominent ear. *Aesthet Surg J* 25:72–79, 2005.
5. Beam RB: Some characteristics of the external ear of American whites, American Indians, American Negros, Alaskan Eskimos, and Filipinos. *Am J Anat* 18:201, 1915.
6. Braun T, Hainzinger T, Stelter K, et al: Health-related quality of life, patient benefit, and clinical outcome after otoplasty using suture techniques in 62 children and adults. *Plast Reconstr Surg* 126:2115–2124, 2010.
7. Brent B: Panel: Aesthetic otoplasty. *Aesthet Surg J* 12:4, 1992.
8. Brown FE, Colen LB, Addante RR, Graham JM, Jr: Correction of congenital auricular deformities by splinting in the neonatal period. *Pediatrics* 78:406–411, 1986.
9. Byrd HS, Langevin CJ, Ghidoni LA: Ear molding in newborn infants with auricular deformities. *J Plast Reconstr Surg* 126:1191–1200, 2010.
10. Calder JC, Naasan A: Morbidity of otoplasty: A review of 562 consecutive cases. *Br J Plast Surg* 47:170, 1994.
11. Caouette-Laberge L, Guay N, Bortoluzzi P, Belleville C: Otoplasty: Anterior scoring technique and results in 500 cases. *Plast Reconstr Surg* 105:504, 2000.

12. Dancey A, Jeynes P, Nishikawa H: Acrylic ear splints for treatment of cryptotia. *Plast Reconstr Surg* 115:2150–2152, 2005.
13. Davis J: Prominent ears. *Clin Plast Surg* 5:471, 1978.
14. Davis J: The prominent ear: Sequel repair. In: *Aesthetic and Reconstructive Otoplasty*, New York, 1987, Springer-Verlag, pp 129–187.
15. Dieffenbach LF: *Die operative chirugiem:* FA Brockhaus, 1845.
16. Elliott RA, Hoehn JG: Otoplasty for prominent ears: A composite approach [microfiche]. *Int J Aesthet Plast Surg* 1972.
17. Elliott RA: Complications in the treatment of prominent ears. *Clin Plast Surg* 5:479, 1979.
18. Elliott RA: Otoplasty: A combined approach. *Clin Plast Surg* 17:2, 1990.
19. Ely E: An operation for prominence of the auricles. *Arch Otolaryngol* 10:97, 1881.
20. Farkas LG: Anthropometry of normal and anomalous ears. *Clin Plast Surg* 5:401, 1978.
21. Farkas LG, Posnick JC, Hreczko T: Anthropometric growth study of the ear. *Cleft Palate Craniofac J* 29(4):324–329, 1992.
22. Furnas DW: Correction of prominent ears by conchamastoid sutures. *Plast Reconstr Surg* 42:189, 1968.
23. Furnas DW: Correction of prominent ears with multiple sutures. *Clin Plast Surg* 5:491, 1978.
24. Furnas DW: Otoplasty for prominent ears. *Clin Plast Surg* 29:273, 2002.
25. Gibson TW, Davis W: The distortion of autogenous cartilage grafts: Its cause and prevention. *Br J Plast Surg* 10:257, 1958.
26. Goulian D, Conway H: Prevention of persistent deformity of the tragus and lobule by modification of Luckett's technique of otoplasty. *Plast Reconstr Surg* 26:399, 1960.
27. Hardingham TE, Muir H: The specific interaction of hyaluronic acid with cartilage proteoglycans. *Biochim Biophys Acta* 279:401–405, 1972.
28. Hinderer UT, del Rio JL, Fregenal FJ: Otoplasty for prominent ears. *Aesthetic Plast Surg* 11:63, 1987.
29. Hirose T, Tomono T, Yamamoto K: Non-surgical correction for cryptotia using simple apparatus. In Foneseca J, editor: *Transactions of the 7th International Congress of Plastic and Reconstructive Surgery, Rio de Janeiro, 1979*, Sao Paulo, Brazil, 1980, Catgraf.
30. Hirose T, Tomono T, Matsuo K, et al: Cryptotia: Our classification and treatment. *Br J Plast Surg* 38:352–360, 1985.
31. Jeffery SL: Complications following correction of prominent ears: An audit review of 122 cases. *Br J Plast Surg* 52:588, 1999.
32. Kaye BL: A simplified method for correcting the prominent ear. *Plast Reconstr Surg* 40:44, 1967.
33. Kenny FM, Angsusingha K, Stinson D, Hotchkiss J: Unconjugated estrogens in the perinatal period. *Pediatr Res* 7:826–831, 1973.
34. Klockars T, Rautio J: Embryology and epidemiology of microtia. *Facial Plast Surg* 25:145–148, 2009.
35. Kösling S, Omenzetter M, Bartel-Friedrich S: Congenital malformations of the external and middle ear. *Eur J Radiol* 69:269–279, 2009.
36. Kurozumi N, Ono S, Ishida H: Non-surgical correction of a congenital lop ear deformity by splinting with Reston foam. *Br J Plast Surg* 35:181–182, 1982.
37. Lentz AK, Plikaitis CM, Bauer BS: Understanding the unfavorable result after otoplasty: An integrated approach to correction. *Plast Reconstr Surg* 128: 536–544, 2011.
38. Luckett WH: A new operation for prominent ear based on the anatomy of the deformity. *Surg Gynecol Obstet* 10:635, 1910. Reprinted in *Plast Reconstr Surg* 43:83, 1969.
39. Macgregor F: Ear deformities: Social and psychological implications. *Clin Plast Surg* 5:347, 1978.
40. Matsuo K, Hirose T, Tomono T, et al: Nonsurgical correction of congenital auricular deformities in the early neonate: A preliminary report. *Plast Reconstr Surg* 73:38–51, 1984.
41. Matsuo K, La Rusca I, Molea G: Non-surgical correction of congenital auricular deformities. *Clin Plast Surg* 17:383–395, 1990.
42. McDowell AJ: Goals in otoplasty for protruding ears. *Plast Reconstr Surg* 41:17, 1968.
43. Merlob P, Eshel Y, Mor N: Splinting therapy for congenital auricular deformities with the use of soft material. *J Perinatol* 15:293–296, 1995.
44. Morestin MH: De la reposition et du plissement cosmetiques du pavillon de l'oreille. *Rev Orthop* 4:289, 1903.
45. Muraoka M, Nakai Y, Ohashi Y, et al: Tape attachment therapy for correction of congenital malformations of the auricle: Clinical and experimental studies. *Laryngoscope* 95:167–176, 1985.
46. Mustarde JC: The correction of prominent ears by using simple mattress sutures. *Br J Plast Surg* 16:170, 1963.
47. Mustarde JC: The treatment of prominent ears by buried mattress sutures: A ten year survey. *Plast Reconstr Surg* 39:382, 1967.
48. Mustarde JC: Correction of prominent ears using buried mattress sutures. *Clin Plast Surg* 5:459, 1978.
49. Nakajima T, Yoshimura Y, Kami T: Surgical and conservative repair of Stahl's ear. *Aesthetic Plast Surg* 8:101–107, 1984.
50. Owens N, Delgado DD: The management of outstanding ears. *South Med J* 58:32, 1965.
51. Park C: Correction of cryptotia using an external stretching device. *Ann Plast Surg* 48:534–538, 2002.
52. Posnick JC: Aesthetic alteration of prominent ears: Evaluation and surgery. In Posnick JC, editor: *Craniofacial and maxillofacial surgery in children and young adults*, Philadelphia, 2000, W.B. Saunders, 45. pp 1143–1153.
53. Reynaud JP, Gary-Bobo A, Mateu J, Santoni A: Chondrites postoperatories de l'oreille externe: 2 cases from a series of 200 cases (387 otoplasties). *Ann Chir Plast Esthet* 31:170, 1986.
54. Rigg BM: Suture materials in otoplasty. *Plast Reconstr Surg* 63:409, 1979.
55. Rodriguez-Champs S: Our procedure for integral aesthetic otoplasty. *Aesthetic Plast Surg* 21:332, 1997.
56. Rogers BO: Ely's 1881 operation for correction of protruding ears. A medical "first." *Plast Reconstr Surg* 42:584, 1968.
57. Rubin LR, Bromberg BE, Walden RH, et al: An anatomic approach to the obtrusive ear. *Plast Reconstr Surg* 29:360, 1962.
58. Schonauer F, La Rusca I, Molea G: Non-surgical correction of deformational auricular anomalies. *J Plast Reconstr Aesthet Surg* 62:876–883, 2009.
59. Smith W, Toye J, Reid A, Smith R: Nonsurgical correction of congenital ear abnormalities in the newborn: Case series. *Paediatr Child Health* 10:327–331, 2005.
60. Stark RB, Saunders DE: Natural appearance restored to the unduly prominent ear. *Br J Plast Surg* 15:385, 1962.
61. Stenstrom SJ: A "natural" technique for correction of congenitally prominent ears. *Plast Reconstr Surg* 26:640, 1960.
62. Stenstrom SJ, Heftner J: The Stenstrom otoplasty. *Clin Plast Surg* 5:465, 1978.
63. Szychta P, Orfaniotis G, Stewart KJ: Otoplasty: An algorithm. *Plast Reconstr Surg* 130:907, 2012.
64. Tan KH: Long-term survey of prominent ear surgery: A comparison of two methods. *Br J Plast Surg* 39:270, 1986.
65. Tan ST, Shibu M, Gault DT: A splint for correction of congenital ear deformities. *Br J Plast Surg* 47:575–578, 1994.
66. Tan ST, Abramson DL, MacDonald DM, Mulliken JB: Molding therapy for infants with deformational auricular anomalies. *Ann Plast Surg* 38:263–268, 1997.
67. Tolleth H: Artistic anatomy, dimensions, and proportions of the external ear. *Clin Plast Surg* 5:337, 1978.
68. Ullmann Y, Blazer S, Ramon Y, et al: Early nonsurgical correction of congenital auricular deformities. *Plast Reconstr Surg* 109:907–913, 2002.
69. Webster CV: The tail of the helix as a key to otoplasty. *Plast Reconstr Surg* 44:455, 1969.
70. Whitaker LA, Yaremchuk MJ, Posnick JC: A method for repositioning the external ear. *Plast Reconstr Surg* 86(1):128–132, 1990.
71. Yotsuyanagi T, Yokoi K, Urushidate S, Sawada Y: Nonsurgical correction of congenital auricular deformities in children older than early neonates. *Plast Reconstr Surg* 101: 907–914, 1998.
72. Young F: The correction of abnormally prominent ears. *Surg Gynecol Obstet* 78:541, 1944.

40

Aesthetic Alteration of the Soft Tissues of the Neck and Lower Face: Evaluation and Surgery

JEFFREY C. POSNICK, DMD, MD

- Aging of the Facial Soft-Tissue Envelope
- Approach to Rejuvenation Surgery of the Neck and Lower Face
- Facial Aging and Rejuvenation: Treatment Pitfalls
- Conclusions
- Patient Education Materials

I agree with Fritz Barton when he stated the following: "The shape of the human face is composed of a skeletal bony framework that is covered by a soft tissue envelope. Overall, skeletal proportions are probably the most important component of facial attractiveness."[7]

In my experience, the absence of an attractive cervical–mental (neck–mandibular) angle most often is the result of a developmental mandibular deficiency that predisposes the affected individual to loose lower cheek and neck skin, the noticeable bunching of fat in the neck area, and the eventual presence of visible platysma bands that worsens and becomes less attractive with age (see Chapters 19, 23, and 25).

This chapter is not intended to be a treatise that addresses all of the details involved in facial soft-tissue aging and rejuvenation options. Rather, the discussion that follows provides perspective concerning the fundamental importance of the maxillomandibular skeletal structures in the aging process of the neck and the lower face. An approach to the management of the anterior neck soft tissues in specific clinical settings is also described.

Aging of the Facial Soft-Tissue Envelope

Background

The layers of the face encompass the skin; the underlying soft tissues, including the fat (subcutaneous and deep); the mimetic muscles (superficial and deep); the investing fascia, which is known as the *superficial muscular aponeurotic system* (SMAS); the retaining ligaments; and the skeletal structural support system (bones, cartilage, and teeth).[4,18] The soft tissues of the face will naturally descend with age, which will result in progressive laxity and ptosis of the skin, the subcutaneous tissue, the fat, and the fascia (i.e., the SMAS–platysma layer) as well as the retaining ligaments. The facial fat (subcutaneous and deep) may either atrophy (i.e., as a result of resorption) or hypertrophy (i.e., increase in volume), and it is also affected by gravity over time. Facial aging is dynamic and cumulative, and it is also affected by hormonal and degenerative changes in the soft tissues. An understanding of the pertinent biochemical and histologic changes that tend to occur in the facial soft tissues with age and through environmental exposures is an important aspect to consider when developing a reconstruction and rejuvenation treatment plan.

Soft-tissue aging is affected by genetic aspects, hormonal changes, and environmental influences. For some individuals, the deeper soft-tissue envelope remains well preserved while the superficial surface of the skin appears weathered. For others, the surface skin remains youthful while the deeper soft-tissue envelope loses structural landmarks with cutaneous laxity. *Extrinsic (environmental) forces* that are working on the skin include dehydration, inadequate nutrition, temperature extremes, traumatic influences, chronic exposure to strong ultraviolet sunlight, toxins (e.g., cigarette smoke), and gravity. The *intrinsic (genetic) effects* have to do with the individual's basic soft tissues and their skeletal morphology.[15,73] Environmental (extrinsic) factors primarily

result in dysplasia and structural alteration of the dermal and epidermal layers of the skin, whereas intrinsic (genetic) effects on the skin may result in atrophy and the loss of structural dermal and epidermal components.[19,20,29,84] An individual's hormone levels throughout life are also known to have significant effects on the aging process.[30] Aging is associated with declining levels of several hormones, and it is gender dependent. It is known that, in women, declining estrogen levels are associated with cutaneous changes, many of which can be reversed or improved via estrogen supplementation. Studies of postmenopausal women indicate that estrogen deprivation is associated with dryness, atrophy, fine wrinkling, poor healing, and hot flashes.[30,60] Epidermal thinning, declining dermal collagen content, diminished skin moisture, tissue laxity, and impaired wound healing have been reported among postmenopausal women. The soft-tissue effects of changing hormonal levels with age in males are less well studied but presumed to be of equal importance. The cumulative *intrinsic (genetic) effects* on the skin can be seen histologically as epidermal thinning, changes in the morphology of the keratinocytes, and a decrease in the number of Langerhans cells and melanocytes. *Environmental (extrinsic) aging effects* on the skin result in keratinocytic dysplasia and the accumulation of solar elastosis, and they can result in cutaneous carcinogenesis (e.g., basal cell carcinoma, melanoma, squamous cell carcinoma).

All individuals experience some degree of both extrinsic (environmental) and intrinsic (genetic) aging simultaneously (Fig. 40-1). The genetic variability of an individual's skeletal framework and the degree and pace of soft-tissue aging in each tissue layer makes a uniform template for facial rejuvenation impractical. Visually, as the intrinsic and extrinsic aging of the skin progress, there will be pigmentary abnormalities, wrinkles (rhytids), and texture irregularities that may result in a "weathered" appearance. Current thinking about aging also involves the concept that the gain or loss of fat volume within the deep compartments leads to changes in the shape and contour of the face.[20] By contrast, soft-tissue folds occur at the transition points between the thicker and thinner superficial fat compartments. The transition points cause nasolabial folds, labiomental folds, submental creases, and preauricular folds.[65,67] In the upper third of the face, around the eyes, researchers have found that suborbicularis oculi fat is composed of two distinct anatomic compartments: the medial limbus and the lateral canthus region. These researchers have also confirmed that the lateral suborbicularis oculi fat extends from the lateral canthus to the lateral orbital thickening.[7] The deep medial cheek fat is the most medial of the periorbital deep fat compartments.[51] In current clinical practice, periorbital rejuvenation relies more on soft-tissue augmentation and rearrangement than on tissue removal. In the midface, progressive skin laxity is often combined with the resorption of subcutaneous fat and gravitational effects to result in characteristic changes in the soft-tissue envelope.[65] In the lower third of the face, there may be ptosis of the chin soft tissues, a loss of the delineation between the jawline and the neck, the development of characteristic jowls, and looseness in the neck skin (which, in the extreme, is characterized as resembling a "turkey gobble"). In many individuals, prominent vertical platysma bands develop in the submental region, and there is often fat hypertrophy in the submental and submandibular regions. These aging effects further hide or eliminate an otherwise attractive and youthful neck–jaw angle.

Frequent Soft-Tissue Facial Aging Characteristics

- Forehead (horizontal) and glabellar (vertical) creases
- Ptosis (drooping) of the lateral eyebrows
- Redundant upper eyelid skin
- Lower eyelid laxity and wrinkles
- Lower eyelid (puffy) visible bags
- Deepening of the nasojugal grooves and the palpebral malar grooves
- Ptosis (drooping) of the malar soft tissues
- Generalized facial skin laxity*
- Deepening of the nasolabial folds*
- Jowl formation*
- Loss of neck definition (neck–jaw angle) and excess fat collection (hypertrophy) in the neck*
- Deep platysmal bands that are visible in repose and often more obvious with function (e.g., facial expression)*
- Perioral wrinkles
- Downturn of the oral commissures
- Deepening of the labiomental creases

*These are facial soft-tissue aging characteristics that may be more or less improved through traditional face and neck lift techniques.

Basic Principles of Midface and Neck Aging and Rejuvenation

Achieve Facial Harmony

The goal is to help the individual look more like the way they used to look rather than severe, operated on, or unnatural.[44,79,94,95] The recognition of any longstanding maxillomandibular skeletal disharmonies is essential to the achievement of enhanced facial aesthetics. If the surgeon attempts to ignore baseline skeletal deformities and then overcompensates with excessive soft-tissue maneuvers, the outcome is likely to be suboptimal. Amateurish or overdone face lifts often look excessively taut, radically defatted, or overfilled (e.g., fat grafts, artificial fillers), or they may involve irregularities from deep layer soft-tissue manipulation. The "overoperated" face may also look startling and unnatural from too much soft-tissue surgical alteration in just one region or layer of the face while ignoring the others. The entire face and all of its layers should be analyzed and given consideration before focusing on just one aspect or technique for rejuvenation.

• **Figure 40-1** A 46-year-old woman first arrived for the surgical assessment of a long-standing bony mass in the left forehead region. She underwent a computed tomography scan that confirmed an osteoma without intracranial involvement. She elected to not undergo biopsy, recontouring, or removal at that time. She returned 13 years later, when she was 59 years old, to discuss facial aging. Her concerns included webbing (platysma bands) and loose skin of the neck; heavy jowls; marionette lines; deep nasolabial folds; puffiness and irregularities of the lower eyelids; hollowing in the temporal and cheek regions; deepening of the labiomental crease; and vertical wrinkles of the upper lip. She had undergone Restylane injections of the nasolabial folds, the marionette lines, the labiomental crease, and the lower eyelids with another clinician but without satisfactory improvement. She also had a short face growth pattern (i.e., maxillomandibular deficiency). During her teenage years, she had undergone camouflage orthodontic treatment that included upper bicuspid extractions to achieve a neutralized occlusion. There was no dental show with the upper lip in repose. With broad smile, only half of the maxillary incisors were visible. The upper lip appeared to have lengthened slightly, and the lips appeared to be more deflated (as compared with images taken when the patient was 46 years old). She stated that she received frequent comments about looking "sad" or "unhappy." She had a history of an inability to breathe well through the nose, loud snoring, restless sleeping, and excessive daytime fatigue. An attended polysomnogram was carried out and confirmed obstructive sleep apnea, with a respiratory disturbance index of 24 events/hour with desaturations of up to 85%. A comprehensive approach to improve dental health, open the upper airway, and enhance facial aesthetics was planned (see Chapters 23 and 25). **A,** Frontal facial views at 46 and 59 years of age. **B,** Oblique facial views at 46 and 59 years of age. **C,** Profile facial views at 46 and 59 years of age.

Achieve Volume Redistribution rather than Skin Tension

Throughout the body, the soft tissues external to the skeleton are anchored to the bony framework by osseocutaneous ligaments that pass directly from the dermis to the periosteum in bare areas where there are no muscles that separate them.[22,39,49,62,63,67,80] The most prominent of these retaining ligaments in the head and neck are in the cheek and along the lateral border of the mandible. The retaining ligaments that partition the cheek fat from the neck are called the *mandibular septum*. A face lift that simply undermines the cheek soft tissues followed by excessive skin resection and then wound closure under tension does not simultaneously manage the deeper soft tissues. Therefore, in these circumstances, skin undermining and resection alone will rarely achieve an attractive or youthful face. This limited "skin-only" approach to face lifting with excessive skin resection may also lead to unsightly scars, flap necrosis, or, more commonly, distortions of key landmarks with the loss of the natural facial curvatures. The use of face-lift techniques that also modestly redistribute the deep soft-tissue layers (i.e., the SMAS–platysma layer) in combination with conservative skin removal and relaxed wound closure will often result in the more pleasing contours that are associated with a youthful and attractive appearance.

Recognize the Presence of Fat Atrophy and Hypertrophy

The process of aging involves not only gravitational effects on the soft tissues and degenerative changes of the skin but, in many cases, noticeable soft-tissue volume loss (deflation) through the atrophy (resorption) of the fat.[10,29,42,66,68,70,72,81] Frequent visual effects of descent in the face include the deepening of the nasolabial folds, ptosis of the malar portion of the superficial fat, and the accumulation of fat in the jowls. The classic effects of deflation are seen most frequently in individuals who have had lifelong thinness of the face. The fat atrophy in these individuals is often visually seen in the temporal fossa, periorbital, buccal, and perioral regions. In other individuals, a pattern of simultaneous face and neck fat hypertrophy as a result of weight gain will occur with age. To avoid an amateurish face lift, a good general rule for the surgeon to follow is to only cautiously remove any fat above the inferior border of the mandible and to be sure that an appropriate amount of fat is removed below the mandibular inferior border. Although adding fat to atrophied regions of the face is beneficial in principle, in many individuals, the long-term success of these techniques (i.e., autogenous fat grafting or the use of artificial fillers) to achieve a natural-appearing rejuvenation remains a work in progress.[10]

Recognize Degenerative Changes in the Skin

Face lifting will address gravitational changes in the soft-tissue envelope, but it does not have an effect on the quality of the soft tissues themselves.[60,85] Face-lift procedures will not treat wrinkles (rhytids), sun damage, creases, or age-related pigmentation. If feasible, fine wrinkles and irregular pigmentation are treated with good-quality skin care and possibly resurfacing procedures (e.g., dermabrasion, chemical peel, laser). Preventative measures of avoiding excessive ultraviolet light exposure, cigarette smoke, temperature extremes, dehydration, and nutritional deficiencies are important.

Recognize the Negative Effects of Any Baseline Maxillomandibular Skeletal Disharmony

Significant variation occurs in the aging of each individual's face and neck. Recognition that the shape of the neck is fundamentally determined by the maxillomandibular skeleton is essential (Figs. 40-1 through 40-4).[13,16,24,32,69,71,93] The ideal neck configuration requires a well-proportioned facial skeleton; it is often described as having a cervical–mental (neck–jaw) angle of 105 to 120 degrees and a distinct mandibular inferior border. Interestingly, despite various assertions and opinions by a handful of authors, in the absence of a loss of the teeth or significant alteration of the dentition through either natural attrition or dental restorative procedures, no convincing data confirms significant changes in the maxillofacial skeleton with aging (see Chapter 25).*

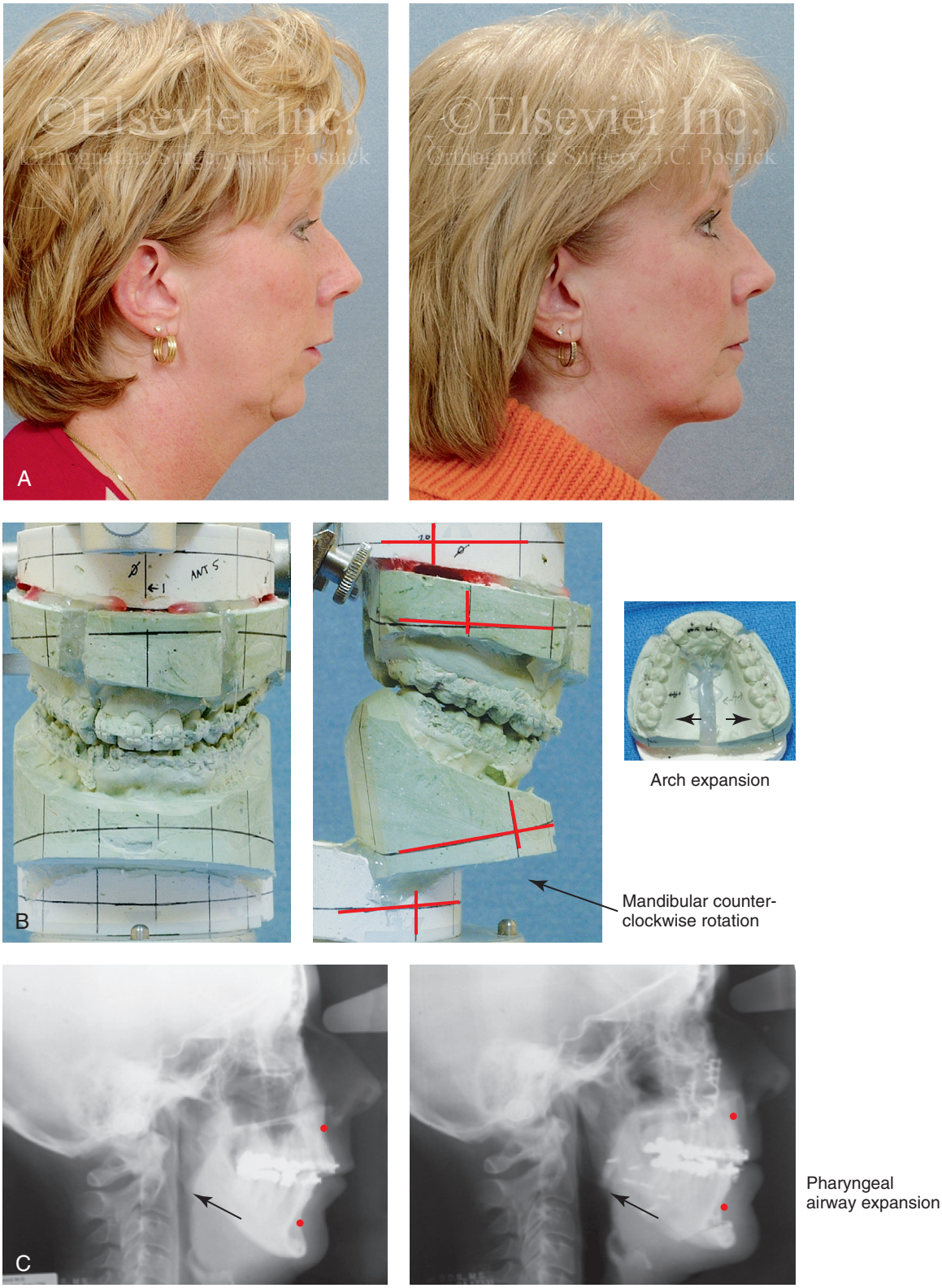
Approach to Rejuvenation Surgery of the Neck and Lower Face

Traditional Face-Lift Approach to Rejuvenation

Face lifting was first performed during the early 1900s and involved skin incisions around the ears, limited skin-flap undermining, and then skin excision before wound closure. In 1859, Gray defined a unique deep layer of tissue just below the facial skin that was described as "superficial subcutaneous fascia." Not much changed until the 1970s, when both Tessier and Skoog independently described the aesthetic benefit of manipulating this superficial subcutaneous fascia as a separate component during face lifting for rejuvenation purposes. The superficial subcutaneous fascia was recognized as investing the platysma muscles and fusing to the parotid fascia as the facial extension of the cervical investing fascia. It later came to be called the *superficial muscular aponeurotic system* or *SMAS,* as mentioned previously.[48] The SMAS envelopes the platysma muscles within the neck and the lower cheek region. Superiorly, it terminates as the investing layer of the superficial mimetic muscles. Laterally, it fuses with the parotid capsule. Superiorly, it passes over the zygomatic arch to join the temporoparietal fascia. Tessier and Skoog were the first to include as part of the face-lift technique not just the undermining and excision of the skin but also the management of the so-called SMAS–platysma layer. This required unique incisions, undermining, redraping, and tightening of the SMAS–platysma as part of the face-lifting procedure. Since the initial contributions of Tessier and Skoog, every conceivable recommendation has been made by surgeons for dissecting,

Text continued on p.1754

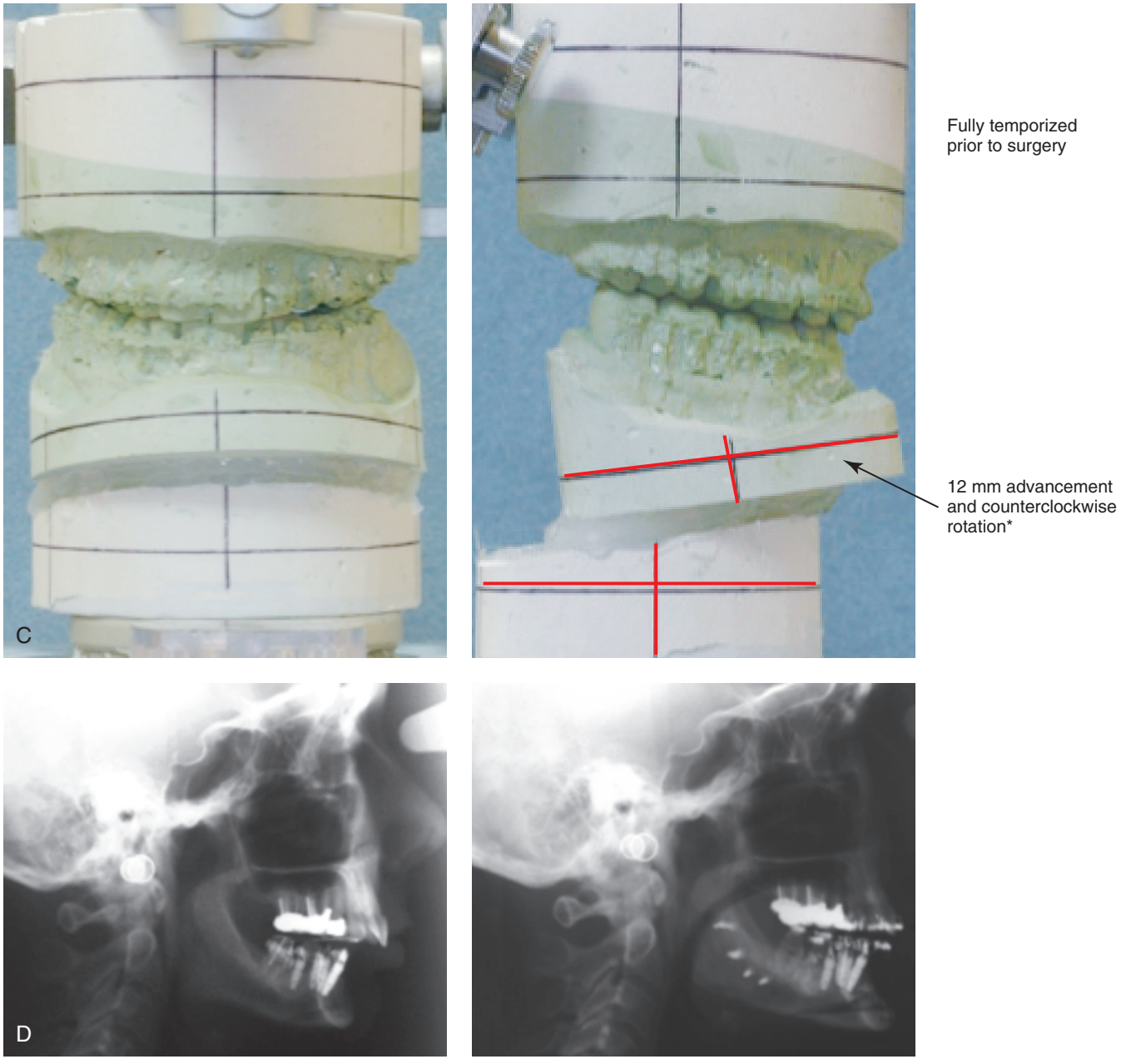
*References 6, 14, 17, 33, 37, 38, 40, 44, 45, 51-57, 64, 74, 75.



• **Figure 40-2** A woman in her mid 50s was referred from her general dentist to an orthodontist for the management of a longstanding Angle Class II excess overjet malocclusion that had gradually resulted in deterioration of the posterior dentition. The orthodontist recognized that the malocclusion resulted from a retrusive mandible and a constricted maxilla. With referral for surgical evaluation, the patient was found to have a developmental jaw deformity, chronic obstructed nasal breathing, and a sleep history that was consistent with obstructive sleep apnea. Unfavorable facial aging with a desire for an improved neck–chin angle was also discussed. An attended polysomnogram confirmed moderate obstructive sleep apnea. The patient agreed to proceed with orthodontic treatment that included lower first bicuspid extractions to relieve dental compensation in combination with jaw, intranasal, and facial surgery. The objectives were to open the upper airway, enhance the facial aesthetics, and achieve improved long-term dental health. The patient's surgical procedures included maxillary Le Fort I osteotomy in segments (horizontal advancement, counterclockwise rotation, arch expansion, and correction of the curve of Spee); bilateral sagittal split ramus osteotomies (horizontal advancement and counterclockwise rotation); osseous genioplasty (horizontal advancement); anterior approach to the soft tissues of the neck (cervical flap elevation, neck defatting, and vertical platysma muscle plication); and septoplasty and inferior turbinate reduction (see Fig. 25-1). **A,** Profile views before and after reconstruction. Note the improved A-point–to–B-point relationship that was achieved as a result of maxillomandibular counterclockwise rotation. **B,** Articulated dental casts that indicate analytic model planning. **C,** Lateral cephalometric radiographs before and after surgery. Note the improved posterior airway space with pharyngeal expansion as a result of the orthognathic procedures.

• **Figure 40-3** A woman in her early 50s was referred by a prosthodontist for surgical evaluation. There had been a gradual deterioration of the dentition, at least partially as a result of the longstanding skeletal Class II excess overjet deep-bite malocclusion. Head and neck evaluation confirmed a retrusive mandible that also resulted in retroglossal airway obstruction. A desire for improved profile aesthetics was also discussed. A comprehensive approach to dental rehabilitation, opening the upper airway, and facial rejuvenation and reconstruction was selected. Coordinated endodontic, orthodontic, periodontic, prosthodontic, and surgical care was required. Periodontal treatment, extractions, dental implant placement, restorative temporization, and orthodontic alignment were carried out. This was followed by surgery that included bilateral sagittal split ramus osteotomies (horizontal advancement and counterclockwise rotation); osseous genioplasty (vertical lengthening) with interpositional grafting; and an anterior approach to the soft tissues of the neck (cervical flap elevation, neck defatting, and vertical platysma muscle plication). This was then followed by crown lengthening and final dental restorations (see Fig. 25-6). **A,** Profile views before and after reconstruction and dental rehabilitation. **B,** Oblique facial views before and after reconstruction and dental rehabilitation.

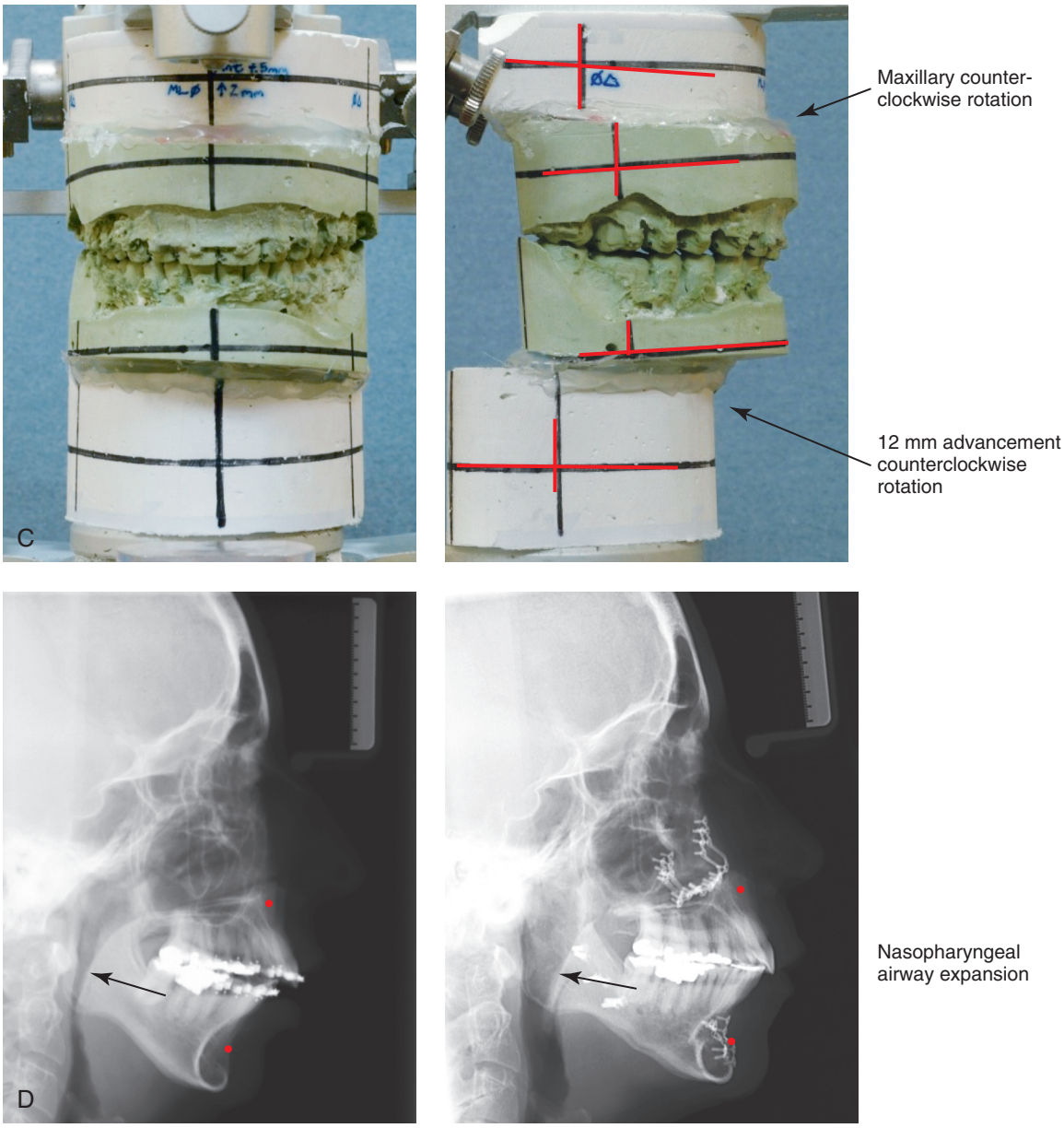
Continued



• **Figure 40-3, cont'd** **C,** Articulated dental casts that indicate analytic model planning. **D,** Lateral cephalometric radiographs before and after reconstruction.

• **Figure 40-4** A woman in her mid 50s was seen by a prosthodontist for the management of a deteriorating posterior dentition. She was confirmed to have secondary dental trauma as a result of a longstanding malocclusion. She was referred to an orthodontist and then for surgical assessment. Head and neck evaluation confirmed a short face growth pattern with a Class II excess overjet deep bite and constricted maxillary arch malocclusion. She had a lifelong history of obstructed nasal breathing, and her history was suggestive of obstructive sleep apnea (this was confirmed with an attended polysomnogram). Facial aesthetic concerns included downturned corners of the mouth, deep perioral creases, early jowl formation, weak profile, an obtuse neck–chin angle, and loose skin around the neck. The patient agreed to a comprehensive surgical and dental rehabilitative approach. Periodontal evaluation, initial restorations, and orthodontic decompensation preceded surgery. The patient's surgical procedures included maxillary Le Fort I osteotomy (horizontal advancement, counterclockwise rotation, and vertical adjustment); bilateral sagittal split osteotomies of the mandible (horizontal advancement and counterclockwise rotation); osseous genioplasty (horizontal advancement); an anterior approach to the neck (cervical flap elevation, neck defatting, and vertical platysma muscle plication); and septoplasty, inferior turbinate reduction, and nasal recontouring (see Fig. 25-7). **A,** Oblique facial views before and after treatment. **B,** Profile views before and after treatment.

Continued



• **Figure 40-4, cont'd** **C,** Articulated dental casts that indicate model planning. **D,** Lateral cephalometric radiographs before and after treatment. Note the improved posterior airway space with pharyngeal expansion as a result of the orthognathic procedures.

undermining, excising, adding to, or removing virtually all of the tissue layers and cell types in the head and neck to further enhance the aesthetic outcome.[2-5,8,18,21,31,47,50,59,77,78] Many of the "innovations" in the area of face lifting have proven to have little additional long-term value, and some have resulted in increased complications and prolonged recoveries. This was confirmed by Chang and colleagues in a recently completed systematic review and comparison of efficacy and complication rates among a variety of face-lift techniques. The authors conclude that there are pros and cons to each of a variety of face-lift techniques and that there is no clear evidence that one is routinely superior to another.[9]

A well-designed traditional face lift often confers much benefit to the jowls (i.e., the mandibular line) and the neck (Figs. 40-5 through 40-10). It can be an effective way to soften the nasolabial folds, to lift the jowls back into the face, to relieve the platysmal bands, to remove the loose skin and excess fat of the neck, and to restore the neck–jaw angle back to its baseline. This can change the overall facial shape from rectangular back to that of a heart in a way that no other soft-tissue treatment modality can provide. The basic

• **Figure 40-5** A woman in her early 50s arrived for the evaluation of facial aging. She had satisfactory skeletal proportions and no significant surface skin layer aging effects. She requested surgery to diminish her jowls, to soften her nasolabial folds, and to reduce the amount of loose skin in her neck region. She underwent a face lift that included periauricular and submental incisions; cheek and neck flap elevation; superficial musculo-aponeurotic system flap elevation with postauricular transposition; neck defatting; vertical platysma muscle plication; and skin resection and redraping. Asymmetric upper eyelid ptosis was not addressed. **A,** Oblique facial views before and after surgery. **B,** Profile views before and after surgery.

traditional face-lift approach requires the consideration of the following:

- *Skin incision placement* (i.e., preauricular with temporal extensions and postauricular with occipital extensions)
- *Skin flap elevation and undermining* (i.e., temporal, cheek, postauricular, neck, and submental regions)
- *Supraplatysmal, intraplatysmal, and subplatysmal defatting* (e.g., below the inferior border of the mandible and chin, above the thyroid cartilage, and anterior to and above the sternocleidomastoid muscles)

• **Figure 40-6** A 50-year-old woman arrived for the evaluation of facial aging. She has satisfactory skeletal proportions and minimal skin surface layer aging effects. She underwent a limited incision brow lift with suspension; blepharoplasty (elliptical excision of the upper eyelid skin and redraping of the lower lid tissue); and a face lift as described in the legend for Fig. 40-5. **A,** Oblique facial views before and after surgery. **B,** Profile views before and after surgery.

• **Figure 40-7** A woman in her late 50s arrived for the evaluation of facial aging. She had satisfactory skeletal proportions but marked surface layer aging effects. She elected to not undergo skin resurfacing procedures. She agreed to a limited incision brow lift with suspension, blepharoplasty (elliptical excision of the upper lid skin and lower lid redraping), and a face lift as described in the legend for Fig. 40-5. **A,** Frontal views before and after surgery. **B,** Oblique facial views before and after surgery. **C,** Profile views before and after surgery.

• **Figure 40-8** A woman in her early 40s requested an improved neck–chin angle and the relief of hooding of the upper eyelids. She had a short face growth pattern that resulted in mildly deficient lower anterior facial height and horizontal facial projection. She had a good airway and occlusion. There was limited surface skin layer damage and aging effects. She underwent upper blepharoplasty (elliptical skin incision) and a face lift as described in the legend for Fig. 40-5. **A,** Oblique facial views before and early after surgery. **B,** Profile views before and early after surgery.

- *Platysma muscle management* (i.e., vertical plication versus the excision of bands)
- *SMAS management* (i.e., preauricular SMAS plication versus SMAS elevation with postauricular flap transposition)
- *Redraping of the elevated skin flaps* (i.e., in a superoposterior vector)
- *Excision of redundant skin* (e.g., temporal, preauricular, postauricular, occipital)
- *Wound closure* under minimum tension

Anterior Neck (Submental) Approach to Rejuvenation

Background

Changes that occur in the neck with age can alter the natural youthful facial curvatures and angles.* As people age, characteristic changes tend to occur in the skin and throughout the soft-tissue envelope. Soft-tissue

*References 12, 18, 23, 25-27, 34-36, 41, 46, 61, 83, 94, 95

• **Figure 40-9** A woman in her early 50s with facial aging concerns. She had a mild long face growth pattern but a satisfactory occlusion. The lower anterior facial height was slightly increased, which resulted in mild lip incompetence and mentalis strain; this resulted in accentuated perioral creases. She agreed to blepharoplasty (elliptical excision of the upper lid skin and redraping of the lower lid tissue); osseous genioplasty (vertical reduction and horizontal advancement); and a face lift as described in the legend for Fig. 40-5. **A,** Frontal views before and after surgery. **B,** Profile views before and after surgery. Note the relief of the perioral folds and the mentalis strain as a result of vertical reduction and advancement genioplasty.

rejuvenation procedures that focus on the neck often include the following: 1) cervical flap elevation; 2) the selective removal of fat (above, in between, and below the platysma muscles); 3) the tightening of the platysma muscles (in the neck midline); and 4) the redraping of lax skin (i.e., skin retraction). In the presence of well-proportioned (harmonious) maxillomandibular skeletal structures, the combination of these procedures can generally be carried out to achieve a preferred geometric angle between the cylindrical neck and the straight-line inferior border of the jaw.[12,18,23,25-27,34-36,41,46,61,83,94,95]

Aging of the neck often presents in one of two classic ways; it is primarily a reflection of the underlying skeletal anatomy. One common pattern is what appears to be a *long neck with a sharp neck–jaw angle.* This occurs in an individual with preferred proportions and fullness of the jaws. With aging, these patients may develop visible platysmal bands but with the maintenance of a reasonable neck–jaw angle and chin projection. A second common pattern of presentation is seen in individuals with what appears to be a *short neck and a poorly defined neck–jaw angle.* This is the result of a baseline mandibular deficiency (e.g., combined

©Elsevier Inc.
Orthognathic Surgery; J.C. Posnick

• **Figure 40-10** A woman in her late 50s arrived for the evaluation of facial aging. She had satisfactory skeletal proportions and occlusion. Her skin was thick but did not have major ultraviolet light damage. She agreed to a limited incision brow lift with suspension; blepharoplasty (elliptical excision of the upper eyelid skin and fat removal and redraping from the lower lids); and a face lift as described in the legend for Fig. 40-5. **A,** Frontal views in repose before and after rejuvenation. **B,** Frontal views with smile before and after rejuvenation.

• **Figure 40-10, cont'd C,** Oblique facial views before and after rejuvenation. **D,** Profile views before and after rejuvenation.

maxillomandibular deficiency, primary mandibular retrognathia, or a long face growth pattern with sagittal deficiency and clockwise rotation of the maxillomandibular complex; see Figs. 40-2, 40-3, and 40-4). Individuals with this type of neck experience facial aging prematurely and in a more detrimental way. In the presence of sagittal deficiency of the mandible, when any degree of neck soft-tissue laxity occurs and even with small amounts of submental and supraplatysmal fat accumulation, further loss of the clear separation between the chin and neck (i.e., an obtuse neck–jaw angle) can be expected. Skeletal (orthognathic) procedures to reconstruct the jaws toward Euclidean proportions will have a dramatic positive effect on the facial aging of this latter group of individuals (see Figs. 40-2, 40-3, and 40-4). Completing traditional face and neck soft-tissue lifting procedures alone in the presence of a deficient maxillomandibular skeletal framework can be disappointing and, in some cases, disastrous. The surgeon should view the overlying soft-tissues as just the icing on the cake and not as the cake itself. The failure to recognize these anatomic realities is responsible for much clinician frustration and many suboptimal "face-lift" results.

A fundamental clinical observation is that the visual appearance of aging in the neck is greatly influenced by the baseline maxillomandibular skeletal anatomy. The ideal horizontal mandibular plane relative to the vertical cylindrical neck (i.e., an angle of 105 to 120 degrees) will abnormally slump downward and backward when the mandibular skeletal framework is deficient and clockwise rotated (i.e., long face growth pattern; see Chapter 21).

An example of the negative effects of the skeleton on the soft-tissue surface anatomy is seen in the presence of primary mandibular deficiency with excess overjet (see Chapter 19). Not only will the lower lip be flaccid with a deep labiomental fold (curl), but the soft tissues directly over the bony chin are bunched and ptotic, and the soft-tissue structures of the neck are cramped together to result in a double (soft-tissue) chin. In the presence of mandibular deficiency, the chin seems to fall directly into the neck without any semblance of a normal "right-angle" curvature (see Fig. 40-2, *A* and *B*).

When both the maxilla and the mandible lack vertical height and horizontal projection (i.e., short face growth pattern; see Chapter 23), the cheeks often appear to be puffy. There will be deep nasolabial folds, heavy jowls, perioral creases, and downturned oral commissures that compound the negative effects of the observed obtuse neck–jaw angle.

The deficient maxillo-mandibular skeletal anatomy seen in the adult, as described above, will have been present since the teenage years; however, it appears to be more aesthetically objectionable with aging as the overlying soft tissues progressively lose tone and become lax.

Patient Selection

The best candidates for a *neck lift through a limited submental incision* are those with a harmonious and proportionate maxillomandibular skeleton and those who do not require or request soft-tissue rejuvenation above the mandibular border or angle. A thorough evaluation by the surgeon followed by a realistic discussion with the patient is required to clarify these issues. To avoid an unhappy patient after surgery, any limitations of an anterior (submental) neck-lift approach should be clarified in advance.

Appropriate patient selection (i.e., for a traditional face lift versus an anterior submental neck lift) is based primarily on the degree of skin laxity. Zins proposed a useful grading scale for neck skin laxity: Grade I (no laxity); Grade II (mild laxity); Grade III (moderate laxity); and Grade IV (severe laxity)[95]:

- *Grade I* patients may only require closed liposuction. However, even Grade I patients frequently benefit from an open approach to fully access and then remove subplatysmal fat.
- *Grade II* patients are good candidates for an anterior (submental) approach to neck rejuvenation. Their limited lax skin should be expected to retract in a favorable way.
- *Grade III* patients can often be managed with an anterior (submental) approach to the neck. They will require wide cervical flap elevation with meticulous attention to release all septae and ligaments to achieve maximum skin retraction and to avoid unfavorable visible irregularities after surgery. Full disclosure with regard to the limitations of an anterior neck lift and the setting of realistic expectations are especially relevant for the Grade III patient to avoid later disappointment.
- *Grade IV* patients are not good candidates for an anterior-only approach to the neck and should be considered for a traditional face lift. Unless skin is excised (i.e., with a traditional face-lift approach), residual laxity will provide a suboptimal result.

Cervical Flap Elevation and Neck Defatting

Some teenagers and young adults and many middle-aged adults benefit aesthetically from neck defatting.[1] The fat removal is typically carried out in the region below the inferior border of the mandible, above the thyroid cartilage, and anterior to the sternocleidomastoid muscles. Whether by liposuction, direct scissors excision, or a combination of the two the objective is to remove appropriate adipose cells deep to the skin and superficial to the platysma muscles. Below the chin in the midline, where there is a tendency for the separation of the platysma muscles, excess fat also frequently accumulates. If the platysma muscles are in continuity (i.e., if there is no dehiscence), then surgical separation is required to expose the excess submental fat for removal.

Closed neck liposuction without skin-flap undermining is preferentially carried out only in those patients with no skin laxity and minimal excess neck fat and who do not require platysma separation to remove submental fat. This situation is more commonly seen in teenagers and young adults. In all other patients, cervical flap elevation is completed first.

Generally, the *cervical skin undermining* extends up to the inferior border of the mandible and chin, laterally to and over the surface of the sternocleidomastoid muscles, and inferiorly past the thyroid cartilage toward the clavicles. In those with only moderate skin laxity, adequate release of all cutaneous septae and cutaneous ligaments will generally allow for skin contracture during healing without the need for skin resection. After surgery, skin irregularities and adhesions will persist if inadequate undermining was accomplished. A second procedure would then be required to complete the undermining. The surgeon should also avoid uneven fat removal and excessive defatting in the neck, because this can result in contour irregularities between the skin and the platysma muscles. Ptotic submandibular salivary glands may also affect an otherwise satisfactory result. This author does not favor either partial submandibular gland resection or intracapsular gland dissection and repositioning, because the incidence of associated complications is high.[28,76,82] Close to the midline and just below the chin, overaggressive fat removal may result in an unnatural concavity, which is a stigma of an amateurish cervical defatting. In some cases, prominent hypertrophy digastric muscles may benefit from "shaving" (i.e., scissors excision) in the region just below the chin.[11]

Management of the Platysma Muscles and Cervical Fascia

The *platysma muscles* are enveloped by the cervical investing fascia on each side. The muscles run from just over the inferior border of the mandible (i.e., the lower cheek) and down to and over the clavicles across the curve of the neck. With contracture, the muscle fibers shorten and bowstring (i.e., form vertical bands) away from the midline of the neck. This bowstringing tendency is restrained by the cervical investing fascia. In most individuals, the thin midline cervical fascia tends to attenuate with age; this explains the fascial dehiscence with a pulling away of the platysma muscle on each side from the neck midline that is observed later in life. Initially, bowstrings are only present with active contraction; eventually, however, passive platysmal banding is observed.

A *platysmaplasty* can be a useful component of neck rejuvenation. Through a 3- to 4-cm incision that is made adjacent to a natural submental crease, subcutaneous undermining is performed that allows for the separation of the neck skin from the platysma muscles. The extent of cervical flap elevation should be dependent on the degree of skin laxity. Even after flap elevation, there are limits to the skin's ability to redrape during healing without irregularities or residual laxity. Those with greater skin laxity (i.e., Grade IV patient and some Grade III patients) are not good candidates for an anterior (submental) neck approach, as discussed earlier in this chapter.

Most individuals who require platysmaplasty will also benefit from neck region fat removal being carried out simultaneously. This includes *supraplatysmal defatting* out to the sternocleidomastoid muscles and down to the thyroid cartilage as well as *intraplatysmal* and *subplatysmal defatting* below the chin as described previously.

After cervical flap elevation and appropriate defatting, the medial border of each platysma muscle is identified and joined in the midline with the use of a series of interrupted sutures. The *vertical platysma plication* extends from just above the thyroid cartilage to approaching the bony chin. Some surgeons prefer a continuous running suture; however, this author believes that such a suture is more likely to result in vertical ridging. As long as the lateral cervical investing fascia remains reasonably tight, elevation of the neck sling can be accomplished through this anterior (submental) approach, without the need for a simultaneous traditional face lift (i.e., preauricular and postauricular incisions, cheek flap elevation, lateral SMAS tightening, and skin excision). Nevertheless, a traditional face-lift approach remains the best way to maximize neck tightening, because skin removal is also possible.

The *anterior (submental) neck approach* is preferred to a *traditional face lift* in individuals with reasonable skin elasticity who are not overly concerned about the aging of their cheek region (i.e., aging of the jowls and the nasolabial folds). An anterior submental approach will not satisfy individuals who need and expect improvements in the frontal view (i.e., a reduction of the jowls, a softening of the nasolabial folds, and the management of the malar and eyelid regions) or in those with moderate or greater degrees of neck skin laxity.[94,95]

Anterior Neck (Submental) Approach: Step-By-Step Surgical Technique (Fig. 40-11) (Video 16)

Positioning on the Operating Room Table

- Place the patient supine on the operating table with his or her head on the Mayfield horseshoe in the neutral neck position.

Anesthesia

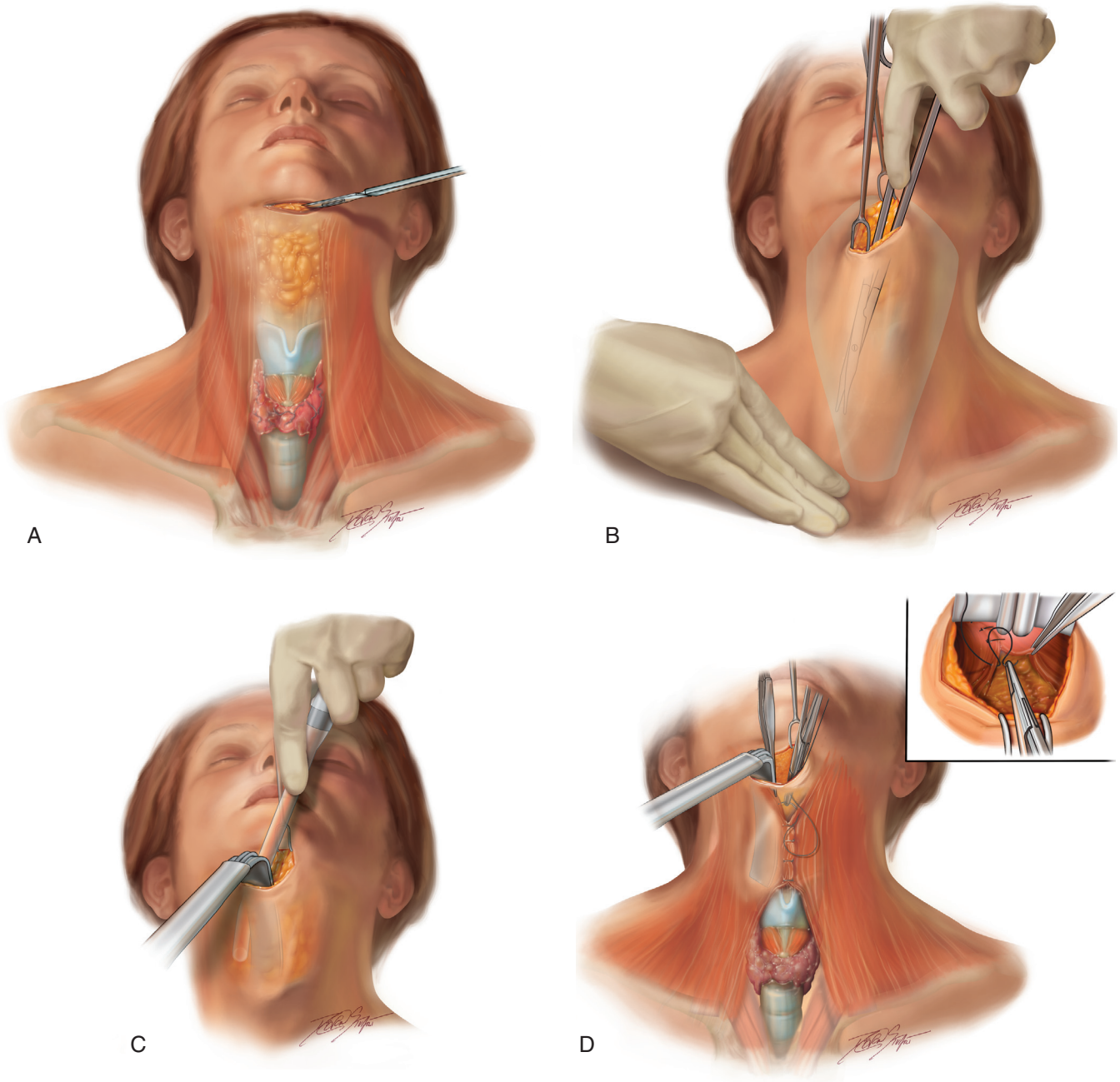
- When the procedure is carried out in conjunction with orthognathic surgery, general anesthesia is administered through a nasotracheal cuffed (RAE) tube. The nasotracheal tube will already have been secured to the cartilaginous septum with suture (#0 Ethibond).
- The application of ophthalmic eye ointment and the insertion of corneal shields deep to the eyelids will also already have been performed.

Medications

- Administer intravenous antibiotics (Cefazolin 17 mg/kg/dose, max: 1 gm).
- Administer steroids via an intravenous bolus (Dexamethasone 0.5 mg/kg/dose, max: 8 mg).

Preparation and Draping

- After the completion of the orthognathic procedures (including the removal of the throat pack and the placement of interocclusal elastics), the patient is reprepped,



• **Figure 40-11** Illustrations that demonstrate the anterior (submental) approach for neck rejuvenation as described in the text. **A,** Illustration of the neck region in the operating room in preparation for an anterior (submental) rejuvenation procedure. The submental incision is marked. A knife (no. 15 blade) is used to initiate the skin incision. Important underlying landmarks are indicated, including the thyroid cartilage; the sternocleidomastoid muscle on each side; the platysma muscles with central dehiscence; and the accumulated fat. **B,** Illustration that demonstrates the elevation of the cervical flaps after the completion of the submental incision. Instruments that are in place include double skin hooks with one at each side of the submental wound. The assistant's hands are positioned over the clavicle to provide counter tension to the double hook located at the submental incision. In this way, the neck skin in the region of the cervical flap elevation is taut. A Stevens scissors is then used to complete the dissection in the exact location where the neck skin is placed under tension. The green shaded area indicates the extent of planned supraplatysma dissection for cervical flap elevation. **C,** Illustration after cervical flap elevation that demonstrates liposuction in progress. A no. 7 curved plastic cannula is used in the supraplatysma plane laterally and in the subplatysma in the midline below the chin. **D,** Illustration that demonstrates vertical platysma muscle plication in progress. A lighted retractor is in place to provide exposure for the direct visualization of the platysma muscle dehiscence. The surgeon uses a long needle driver and a long forceps to place interrupted sutures (3-0 Vicryl) for the achievement of vertical platysma muscle plication. There is also a close-up illustration of the wound with a lighted retractor in place. The long forceps elevates the edge of the platysma muscle on the right side. A needle driver is used to place a suture through the elevated platysma edge. Several interrupted sutures are already in place from just above the thyroid cartilage.

and additional sterile drapes are placed over the ones that are already there.
- Reprep entire neck below the clavicles, the face, and the postauricular regions with the use of Betadine solution (do not use soap, alcohol, Hibiclens, and so on).
- Draping is completed with exposure of the neck just inferior to the sternal notch and clavicles. Exposure of the entire neck, the external ears, and the lower face is also accomplished.

Instrumentation
- New sterile gloves are provided to the operating team.
- New sterile instrument sets are opened.

Surgical Markings
- Locate and use a surgical pen to mark the extent of planned cervical flap elevation, including the following:
  - The inferior border of the mandible and the chin
  - The anterior border of the sternocleidomastoid muscles
  - The thyroid cartilage and below toward the clavicles
- Locate and use a surgical pen to mark the proposed 3- to 4-cm submental incision.

Local Anesthesia
- Injection 50 mL of tumescent solution (epinephrine 1:1000, 1 mL; bupivacaine plain, 10 mL; 0.9% sodium chloride, 500 mL) within the designated surgical field. The injection is made below the skin and above the platysma muscles.

Submental Incision
- Use a knife (no. 15 blade) to complete the 3- to 4-cm submental incision.

Elevation of the Cervical Flaps
- A wide double skin hook is placed on either side of the complete submental incision. With the use of the skin hooks spreading in opposing directions, tension separates the wound.
- A long Stevens scissors is used to elevate the cervical flaps. Stretch-tension is achieved on the skin of the cervical flap to be elevated; this is accomplished with the double skin hook at the submental wound and the assistant's hands over the neck skin above the clavicle. The Stevens scissors is then used to blindly separate the skin and the subcutaneous flap from the underlying platysma muscles throughout all planned locations on both sides of the neck.
- The dissection continues down past the thyroid cartilage toward the clavicles and laterally just above the anterior border of each sternocleidomastoid muscle.
- The dissection continues superiorly to the inferior border of the mandible. Caution is used to avoid injury to the marginal mandibular branch of cranial nerve VII.
- The lighted retractor is then used to confirm the complete release of the cervical flaps from all cutaneous septae and cutaneous ligaments.
- The lighted retractor is also used to confirm adequate hemostasis. A guarded electrocautery device is used to control any bleeding vessels.

Defatting Above, in Between, and Below the Platysma Muscles
- Supraplatysma defatting is carried out via open liposuction with the use of a no. 7 curved plastic cannula.
- With the use of a lighted retractor under direct vision, any remaining clumps of fat are extracted via either liposuction or sharp scissors removal.
- If the platysma muscles are intact (i.e., without dehiscence), they are separated in the midline just below the chin to expose the submental fat pad.
- Subplatysmal fat is removed with the use of open liposuction via a no. 7 curved plastic cannula or sharp scissors excision.
- In the interdigastric space, fat is removed or resected to be flush with the anterior digastric bellies.
- If there is prominent elevation of the digastric muscles, the edges are resected with a scissors.

Platysmaplasty
- Consideration is given to tightening the platysma muscle sling. Dehiscence of the muscles in the midline may be seen from the thyroid cartilage below to the bony chin above.
- The lighted retractor is useful to directly visualize the platysma muscles.
- Vertical platysma muscle plication is carried out with interrupted sutures (3-0 Vicryl).
- Typically, eight to ten interrupted sutures are placed.

Skin Closure
- Before wound closure, the redraped cervical flaps are inspected to confirm the absence of irregularities caused by residual attached cutaneous septae or cutaneous ligaments.
- The wounds are again inspected with the lighted retractor and the electrocautery device to confirm adequate hemostasis.
- If oozing is poorly controlled, consideration is given to the placement of a suction drain.
- The fascia layer of the submental wound is closed with interrupted sutures (5-0 Vicryl).
- The skin edges are approximated with running suture (6-0 nylon).

Placement of Dressing
- A mildly compressive chin and neck dressing is placed (e.g., a Jobst neck/chin strap).

Postoperative Care
- Two days after surgery, the chin/neck dressing is removed, and the patient is allowed to shower (including the head and neck region) and shampoo the hair. The dressing is replaced by the patient for an additional 3 days.

- The dressing is removed daily for showering and then replaced until 5 days after surgery.
- Submental sutures are removed on the fifth postoperative day.
- Antibiotics are discontinued after 5 days.
- No sports participation or direct facial trauma is allowed for 5 weeks.

Complications and Unfavorable Results

Hematoma

The incidence of hematoma with an anterior (submental) neck approach using the techniques described in this chapter is not common. The use of a lighted retractor confirms satisfactory hemostasis under direct vision at operation. Proper patient selection (i.e., non-hypertensive, not taking platelet-inhibiting medications or supplements) and a smooth emergence from anesthesia (i.e., control of blood pressure and vomiting) are also essential. A compression dressing as described is helpful to limit swelling after surgery. This author has not found it necessary to routinely place drains.

Skin Necrosis

The incidence of skin necrosis in properly selected patients and with the use of sound flap elevation techniques is uncommon. The occurrence of flap necrosis may be significantly higher in those with vascular occlusive disorders and in smokers (see Chapters 16 and 25).

Infection

Wound infection is not common after neck lift surgery as described in this chapter. The use of perioperative antibiotics is indicated. Close postoperative communication between the surgeon and the patient is essential to ensure prompt treatment if infection does occur. Infection is treated with appropriate antibiotics and drainage procedures.

Nerve Injury

The incidence of facial nerve injury (i.e., of the marginal mandibular branch) is low as long as the field of dissection remains below the inferior border of the mandible and above the platysma muscle laterally.

Skin Irregularities

Skin irregularities are primarily the result of inadequate cervical flap undermining with the incomplete release of all cutaneous septae and cutaneous ligaments extending the borders of flap dissection. In properly selected cases and with the complete release of septae and ligaments, neck skin has the unique ability to contract without the need for resection. However, those individuals with moderate or greater degrees of laxity before surgery cannot be expected to achieve optimal results without skin excision (i.e., a traditional face-lift approach). Irregularities may also result from a hematoma that is slow to resolve. Skin irregularities related to inadequate undermining that persists are managed secondarily by extending the degree of undermining.

Contour Irregularities

Contour irregularities may result from underresection, overresection, or the removal of supraplatysmal subcutaneous fat. When this does occur, it is generally seen either low in the neck toward the thyroid cartilage or laterally toward the sternocleidomastoid muscles. The removal of retained fat is managed secondarily with surgery; this is easier to deal with than areas of overresection that require grafting.

Midline Ridging

Midline ridging after vertical platysma muscle plication may occur with the extensive use of non-resorbable sutures in the presence of very thin overlying skin. This author believes that the placement of a continuous running suture (rather than individual interrupted sutures) may be more likely to result in ridging. In those few circumstances in which significant midline ridging occurs, secondary surgery with suture removal and platysma recontouring may be beneficial.

Residual Submental Fullness or Hollowing

Subplatysma midline defatting is carried out to reduce excess fullness. After the separation of the platysma muscles, defatting down to the level of the digastric muscles is usually beneficial.[11] If residual fullness remains as a result of inadequate fat removal, this can be managed secondarily to finish the job. Excessive defatting in this region often creates a submental hollow with a resulting "operated" look. Secondary grafting may be beneficial.

Visible Unsightly Digastric Muscle Ridging

Partial resection (shaving) of the digastric muscles is not typically necessary but should always be considered on the basis of the presurgical evaluation and the intraoperative findings.

Submandibular Gland Ptosis

Prominent submandibular glands are seen in a subgroup of patients. Gland ptosis should be noted during the preoperative office evaluation. In these cases, a thoughtful preoperative discussion with the patient about his or her expectations and then limiting surgery appropriately may be best. This author does not favor either partial submandibular gland resection or intracapsular release and repositioning. The risk of complications (i.e., salivary gland drainage, hematoma, nerve damage, contour irregularities) may be greater than any cosmetic advantage. This explains why the surgical manipulation of the submandibular gland has not achieved general acceptance.

Direct Anterior Neck Skin Excision

In general, this author does not favor direct skin removal from the anterior neck with the use of Z-plasty, W-plasty, or other similar procedures. The resulting scars are frequently objectionable, although some surgeons favor these approaches for selected older men. It would be very difficult to justify such procedures for a young person, especially a

younger woman. If skin removal is required, a traditional face lift is more likely to result in patient satisfaction (i.e., less-visible scars and preferred neck rejuvenation).

Facial Aging and Rejuvenation: Treatment Pitfalls

Pitfalls of a "Soft-Tissue–Only Approach" to Facial Rejuvenation

When a middle-aged woman consults a cosmetic surgeon to discuss aging in the lower face and neck, she may typically ask about the following:

- A weak-appearing chin
- A double (soft-tissue) chin and excess neck fat
- Loose neck skin and a poor neck–jaw angle
- Deep nasolabial folds
- Heavy jowls
- Marionette lines and perioral lines, creases, or folds

The surgeon may discuss with the patient non-surgical and surgical options that include the following:

- The injection of non-autogenous "fillers"
- The injection of autogenous fat grafts
- Skin care and resurfacing techniques
- Anterior neck surgical procedures
- A spectrum of face-lift surgical options

If baseline maxillomandibular skeletal disproportions are pronounced, the surgeon is likely to suggest the following:

- The injection of fillers and fat grafts to augment the recognized skeletal deficiencies
- The placement of "hard" implants (e.g., Silastic, porous polyethylene) to augment the recognized skeletally deficient regions (i.e., the chin; the angles of the mandible; and the perinasal, infraorbital, and zygomatic regions)

The surgeon may neglect to consider patient-specific *smile aesthetics* or *jaw dysmorphology* when discussing treatment options. Interestingly, the patient is not likely to intuitively equate these aspects. In addition, neither the patient nor the surgeon is likely to consider the *potential cause-and-effect relationship between skeletal dysmorphology and baseline breathing difficulties (i.e., maxillomandibular deficiency and obstructive sleep apnea;* see Chapter 26).

The reasons why a surgeon may not fully consider the interrelationships of soft-tissue facial aging and jaw dysmorphology; baseline breathing difficulty and jaw dysmorphology; and smile aesthetics and dental disharmony may relate to a tendency for clinicians to suggest treatment options that they are most familiar with and personally able to perform.

A preferred approach is to educate the patient about pertinent aspects that may go beyond the original chief complaint and the surgeon's immediate expertise.[43,58,78,79,86-92] To best ensure a successful outcome and a satisfied patient, a comprehensive discussion of pertinent findings should precede any compromised treatment delivered (Figs. 40-12 and 40-13).

When a clinician attempts to achieve facial rejuvenation by overcompensating with a soft-tissue–only approach (i.e., the excessive injection of fillers or overtightening of the facial soft tissues) without addressing baseline skeletal deformities and disproportions, a suboptimal result is more likely.

A fundamental principle is that both smile aesthetics and optimal neck rejuvenation are highly dependent on acceptable dental harmony and reasonable maxillofacial skeletal proportions, respectively.

In addition, overlooking baseline upper airway obstruction as a result of maxillomandibular deficiency is not ideal for two reasons. First, the clinician has the obligation to inform patients of their health needs. Second, if a surgical procedure is undertaken (i.e., face lifting under anesthesia), then any baseline airway compromise (e.g., obstructive sleep apnea) may increase perioperative risk.

Pitfalls of a "Dental-Only Approach" to Facial Rejuvenation

When a middle-aged woman consults a restorative dentist or an orthodontist with a chief complaint of an unattractive smile, it is tempting for the clinician to only consider the alignment, shape, and color of the teeth (see Fig. 40-3). If the dentition and occlusion cannot be adequately improved through a combination of restorative dentistry and orthodontic mechanics, a dentofacial deformity should be suspected. The consulted orthognathic surgeon may then be tempted to limit the discussion to the "simplest way" to correct the occlusion (i.e., one-jaw surgery) without fully considering skeletal dysmorphology, facial aesthetics, or any baseline upper airway issues (e.g., chronic nasal obstruction, obstructive sleep apnea). For example, the individual with a long face Class II anterior open-bite growth pattern may arrive to the surgeon with a request from the orthodontist to "close the bite." It may be possible to achieve an acceptable occlusion with surgically assisted rapid palatal expansion and further orthodontic alignment. Doing so may resolve the open bite, but it is unlikely to address the following:

- Gummy smile
- Lip incompetence
- Chronic nasal obstruction
- Baseline obstructive sleep apnea
- Appearance of a "big" nose, "flat" cheekbones, and "weak" chin
- Negative-vector globe/eyelid relationship

Full disclosure to the individual regarding the limitations of an isolated dental or occlusion-only approach and the potential advantages of selecting a more comprehensive reconstruction best ensures patient satisfaction.

Difficulty achieving an optimal outcome may also occur when the restorative dentist or the orthodontist has overoptimistic expectations of what can be achieved through limited "surgical cosmetic" procedures. For example, the dental team may incorrectly assume that, if they are able to improve the occlusion in an individual with mandibular deficiency, then a chin implant will resolve the aesthetic concerns (see Fig. 38-1). It should be within the collaborative skill set of the treating orthodontist and the consulting orthognathic surgeon to broadly evaluate the patient's dental rehabilitative, jaw reconstruction, and rejuvenation needs.

Pitfalls of a Segmental Soft-Tissue Approach to Facial Rejuvenation

At times, a surgeon will be asked to perform a limited "segmental" soft-tissue approach to an individual's facial aging. For example, the patient shown in Fig. 40-14 presents with

• **Figure 40-12** A woman in her 50s requested evaluation for a "weak chin" and unfavorable neck aesthetics. Since her teenage years, she was known to have significant mandibular deficiency and a degree of maxillary hypoplasia. She underwent orthodontic treatment only and has a stable occlusion. Maxillary and mandibular osteotomies with advancement would best restore Euclidean proportions, open the airway, and rejuvenate the face (see Chapters 23 and 25). She was not prepared to undergo further orthodontics or an orthognathic approach. A compromised treatment plan was agreed to that included osseous genioplasty (vertical lengthening and minimal horizontal advancement) with interpositional bloc hydroxyapatite grafting and an anterior approach to the neck (cervical flap elevation, neck defatting, and vertical platysma muscle plication). **A,** Frontal views before and after surgery. **B,** Oblique facial views before and after surgery.

• **Figure 40-12, cont'd C,** Profile views before and after reconstruction. **D,** Lateral cephalometric radiographs before and after reconstruction.

a degree of brow ptosis; asymmetric upper eyelid ptosis and hooding; lower eyelid fat protrusion; deep nasolabial and perioral folds; heavy jowls; and neck submental soft-tissue fullness that involves all of the layers (i.e., skin, platysma, and fat). There is also notable surface-layer skin aging and a degree of maxillomandibular deficiency.

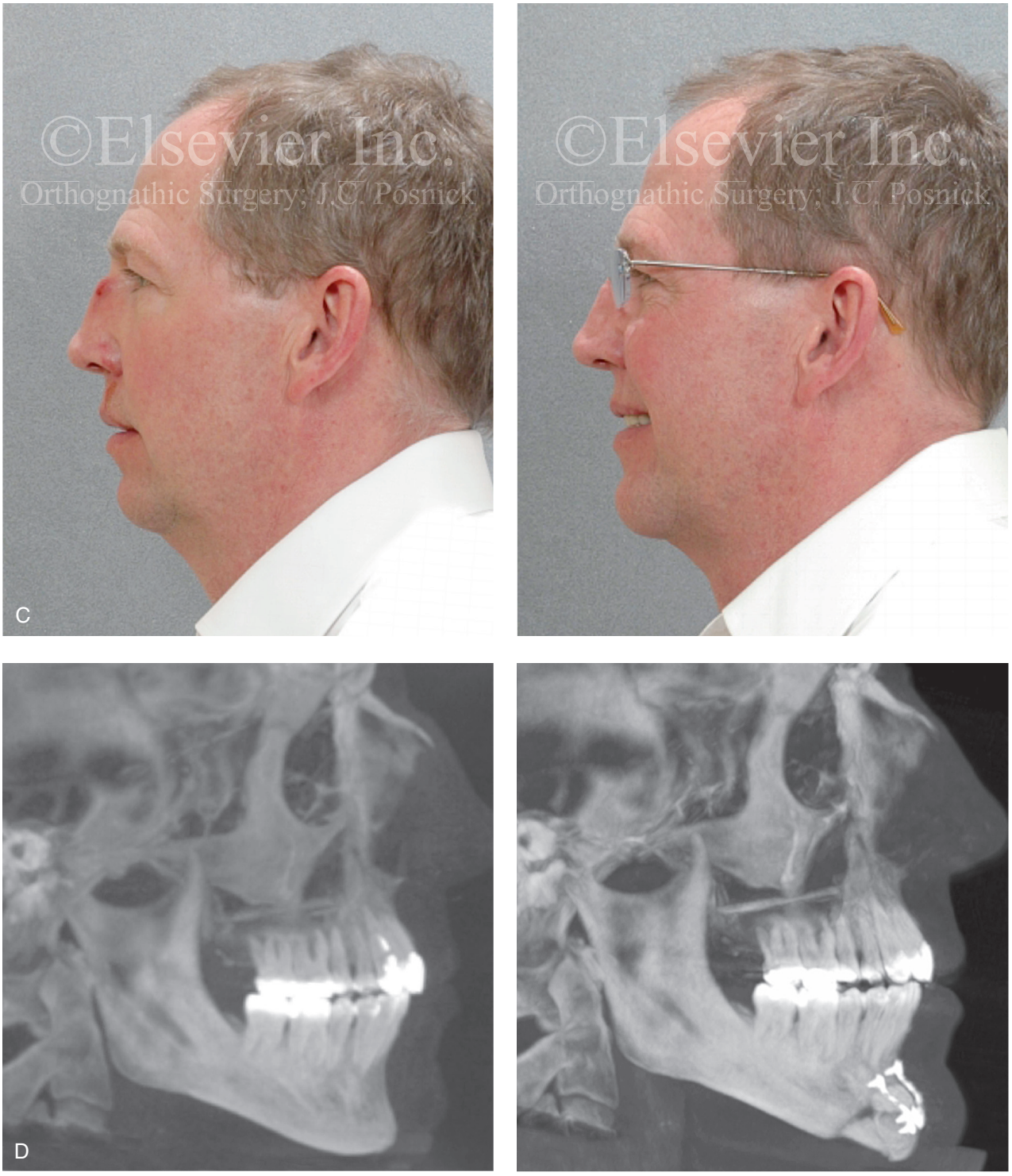
The patient requested the management of only the soft-tissue aging issues in the neck and jowls. This limited goal was reasonably achieved with a face lift. However, residual aging affecting the upper face (i.e., the brow and eyelids) was not addressed, which explains the suboptimal result. The surgeon's agreeing to a compromised soft-tissue "segmental" rejuvenation approach should be with full disclosure to the patient to avoid any misunderstandings.

Conclusions

The layers of the face include the skin, the underlying soft tissues and the skeletal structural support system (i.e., the bone, the cartilage, and the teeth). The soft tissues of the face will naturally descend with age. These and other soft-tissue changes occur throughout the face and neck and will alter the natural youthful facial curvatures. Nevertheless, an unavoidable fact is that the visual appearance of aging in the neck is greatly influenced by the baseline maxillomandibular skeletal framework. Consideration should be given to reconstructing the jaws toward Euclidian proportions, when feasible, before extensive soft-tissue procedures are undertaken.

Standard soft-tissue neck rejuvenation procedures remain an important part of the surgeon's armamentarium and include cervical skin flap undermining and elevation; defatting below the mandible platysmaplasty; and traditional face lifting. The clinician's knowledge of common patterns of dentofacial skeletal dysmorphology and their expected negative effects on soft-tissue aging—in combination with up-to-date methods for reconstruction and rejuvenation—best ensures optimal patient care.

• **Figure 40-13** A man in his early 50s was in a bicycle accident and sustained a displaced nasal fracture and an upper lip laceration. During the consultation, he also discussed a lifelong concern about a weak profile and, with age, the progressive laxity of the neck soft tissues. A lateral cephalometric and Panorex radiograph confirmed the nasal fracture and the microgenia. The patient had a stable occlusion without excess overjet. The nasal fracture required closed reduction with packing, and the upper lip laceration was approximated with suture for primary closure. The patient also underwent an osseous genioplasty (vertical lengthening and horizontal advancement) with interpositional bloc hydroxyapatite grafting and an anterior approach to the soft tissues of the neck (cervical flap elevation, neck defatting, and vertical platysma muscle plication). He is shown before and 3 months after the procedures. **A,** Frontal views before and after surgery. **B,** Oblique facial views before and after surgery.



• **Figure 40-13, cont'd** **C,** Profile views before and after surgery. **D,** Lateral cephalometric radiographs before and after surgery.

• **Figure 40-14** A woman in her mid 50s arrived with a request for neck rejuvenation. Over the years, excess loose skin had developed around her neck. She had a mild degree of maxillomandibular deficiency that was characterized as diminished lower anterior facial height and limited horizontal projection. The surface layer of the skin was thin with spotty pigmentation and wrinkling. She also had significant brow and upper eyelid ptosis. She did not wish to undergo an orthognathic correction, and she refused rejuvenation of the brow and eyelids at this time. She requested a segmental soft-tissue surgical approach that was restricted to the neck and jowls. Her face lift was carried out as described in the legend for Fig. 40-5. The aesthetic results are limited as a result of the inability to manage upper-face soft-tissue aging and the maxillomandibular skeletal deficiency. **A,** Frontal views before and after surgery. **B,** Oblique facial views before and after surgery. **C,** Profile views before and after surgery.

Patient Education Materials

Neck Liposuction Surgery

Liposuction and direct excision are surgical techniques that are used for the removal of excess fatty tissue. They are appropriate procedures to use in the neck region, and they may be carried out through a small incision below the chin. The presence of firm, elastic skin in the neck region will result in the most favorable contours after fat removal.

During liposuction, a cannula (i.e., a thin tube with an opening near the end) is inserted, and the surgeon moves it back and forth under the skin while high-pressure suction removes the unwanted fat. Fat removal in the neck can be done alone or in conjunction with other procedures (e.g., platysma muscle tightening, genioplasty, jaw-straightening surgery).

After the neck liposuction procedure, a tight-fitting dressing is applied to limit swelling and to help your skin conform smoothly to the new shape of your underlying tissue. Within 1 week, the stitches are removed. Depending on whether other procedures were carried out simultaneously, most people are then ready to go back to their work environment.

INSTRUCTIONS FOR NECK LIPOSUCTION SURGERY

The purpose of these instructions is to help you to prepare for and then recover from your operation with as little discomfort and inconvenience as possible.

Preoperative Instructions

1. Do not take any aspirin, aspirin-containing products, or aspirin-like products for at least 2 weeks before and 2 weeks after surgery. Aspirin and aspirin-like products tend to increase bleeding during surgery and bruising postoperatively.
2. You should not smoke for at least 2 weeks before surgery and 2 weeks afterward. Smoking jeopardizes wound healing, it may result in lung congestion, and it will hinder optimal results.
3. Starting at least 2 days before surgery, we request that you shower and shampoo your hair at least once per day. The morning of surgery, shower and shampoo your hair again.
4. Do not eat or drink anything after midnight the night before surgery, not even a sip of water.
5. Be sure to make arrangements for someone to accompany you home after surgery, because you will not be allowed to drive.
6. We will give you a prescription for pain control and another for antibiotics. You may wish to fill them before the date of surgery.

Postoperative Instructions

1. Applying a cold compress to the anterior neck region during the initial 36 hours after surgery will reduce swelling and discomfort.
2. Keep your head and upper back elevated when resting and sleeping for the first week after surgery.
3. You may eat whatever you wish, but start out slowly, because the anesthesia may leave your stomach unsettled for several hours.
4. Maintaining some compression in the neck region for at least 1 week after surgery may be helpful to minimize swelling and for the healing of the skin to the deeper structures of the neck. This is achieved with a specialized neck pressure garment that is applied during surgery. Your surgeon will also provide you with specific instructions about the neck dressing.
5. For men, shaving with an electric razor maybe preferred until you become comfortable with the changes in the sensation of the neck skin region.
6. Get lots of rest and perform minimal physical activity during the first week after surgery. You may then gradually increase your activity back to normal by 4 to 6 weeks after your operation as per instructions from your surgeon.
7. Palpable small lumps and a tight feeling in the neck just below the chin are common findings. These will gradually improve during the final phase of healing.
8. Call the office for an appointment to see your surgeon on the date specified at the time of discharge.
9. If there are concerns at any time, contact your surgeon's office. After hours, the answering machine message will advise you about how to proceed.

Jeffrey C. Posnick, DMD, MD
Director, Posnick Center for Facial Plastic Surgery

Consent for Neck Liposuction Surgery

_____ **INSTRUCTIONS**

Initial here This is a document that has been prepared by your surgeon to help educate and inform you about your suction-assisted lipectomy (liposuction) surgery and its risks as well as alternative treatment options. It is important that you read this information carefully and completely. Please initial each section on each page to indicate that you have read and understand it. When all of your questions are answered, please also sign the consent for surgery on the last page as proposed by your surgeon.

_____ **INTRODUCTION**

Initial here Liposuction and direct excision are surgical techniques that are used for the removal of excess fatty tissue. They are appropriate procedures to use in the neck region, and they are carried out through a small incision below the chin. The presence of firm, elastic skin will result in the most favorable neck contour after fat removal. During liposuction, a cannula (i.e., a thin tube with an opening near the end) is inserted, and the surgeon moves it back and forth under the skin while high-pressure suction removes the unwanted fat.

Fat removal in the neck can be done alone or in conjunction with other procedures (e.g., platysma muscle tightening, genioplasty, jaw-straightening surgery). After the liposuction procedure, a tight-fitting dressing is usually applied to the neck to limit swelling and to help the skin conform smoothly to the new shape of the underlying tissue. Within 1 week, the stitches are removed. Depending on whether other procedures were carried out simultaneously, most people are then ready to return to their work environment.

_____ **ALTERNATIVE TREATMENT**

Initial here Alternative forms of management include the decision to not treat the fatty deposits with surgery. Diet and exercise regimens may be of benefit for the overall reduction of excess body fat. In some patients, the removal of excess skin in the neck with the use of a more extensive neck lift or face lift technique may be required in addition to liposuction. Risks and potential complications are also associated with these and other alternative forms of treatment.

_____ **RISKS OF NECK LIPOSUCTION SURGERY**

Initial here Every surgical procedure involves a certain amount of risk. An individual's choice to undergo a neck liposuction procedure is based on the comparison of the risk to the potential benefit. Although the majority of patients do not experience the complications described below, you should discuss each of them with your surgeon.

_____ **PATIENT SELECTION**

Initial here Individuals with poor skin tone, medical problems, obesity, or unrealistic expectations may not be candidates for neck liposuction.

_____ **BLEEDING**

Initial here It is possible, although unusual, to have a significant bleeding episode during or after neck liposuction. If postoperative bleeding does occur, urgent treatment to drain accumulated blood or to control ongoing bleeding may be required. Do not take any aspirin or aspirin-like medications for 2 weeks before surgery, because this may increase the risk of bleeding. Individuals with poorly controlled hypertension are also at risk.

_____ **INFECTION**

Initial here Infection is not common after neck liposuction surgery. If an infection does occur, treatment that includes a change in antibiotics or additional surgery may be necessary.

_____ **SKIN SCARRING**

Initial here Although good wound healing after neck liposuction is expected, abnormal scarring may occur within the skin and the deeper tissues. Additional treatments, including surgery, may be helpful to treat abnormal scarring.

_____ **CHANGES IN SKIN SENSATION**

Initial here A decrease in skin sensation after neck liposuction may occur. This usually resolves over a period of time.

_____ **SKIN DISCOLORATION AND SWELLING**

Initial here Skin discoloration and swelling normally occur after neck liposuction. In rare situations, swelling and skin discoloration may persist.

_____ **SKIN CONTOUR IRREGULARITIES**

Initial here Contour irregularities, palpable wrinkling, and depressions in the skin may occur after liposuction. Additional treatments, including surgery, may be helpful to manage skin contour irregularities.

_____ **NECK ASYMMETRY**

Initial here It may not be possible to achieve a symmetric appearance with neck liposuction procedures. Factors such as skin or muscle tone and bony prominences also contribute to the noticeable asymmetry of the facial features.

_____ **LONG-TERM EFFECTS**

Initial here Subsequent alterations in neck contour may occur as a result of aging, weight loss or gain, or other circumstances not related to liposuction.

_____ **SURGICAL ANESTHESIA**

Initial here Both local and general anesthesia involve risk. Complications are possible with all forms of anesthesia and sedation.

_____ **ALLERGIC REACTION**

Initial here Local allergies to tape, suture materials, or topical preparations have been reported. Systemic reactions, which are more serious, may occur in response to drugs that are used during surgery or prescription medications taken afterward.

_____ **HEALTH INSURANCE**

Initial here Most health insurance companies exclude coverage for cosmetic procedures such as liposuction or for any related complications that may occur. Please carefully review your health insurance subscriber information.

_____ **ADDITIONAL SURGERIES THAT MAY BE NECESSARY**

Initial here There are many variable conditions in addition to risks and potential surgical complications that may influence the long-term results of neck liposuction. Although complications occur infrequently, the risks cited are those that are particularly associated with neck liposuction. The practice of medicine and surgery is not an exact science. Although good results are expected, there is no guarantee or warranty expressed or implied with regard to the results that may be obtained.

Continued

_____ **FINANCIAL RESPONSIBILITIES**

Initial here The cost of surgery involves several charges for the services provided, including the fees charged by your surgeon (i.e., surgeon's fees); the cost of surgical supplies and the use of the operating room (i.e., facility/hospital charges); and the costs associated with anesthesia (i.e., anesthesiologist's fees). Depending on whether the cost of surgery is covered by your medical insurance plan, you will be responsible for necessary copayments, deductibles, and all other charges that are not covered. Additional costs may occur should complications develop from the surgery. Any secondary surgeries and all charges involved with additional procedures (i.e., surgeon's fees, facility/hospital charges, anesthesiologist's fees) would also be your responsibility.

_____ **DISCLAIMER**

Initial here Informed-consent documents are used to communicate information about the proposed surgical treatment as well as to disclose information about risks and alternative forms of treatment. The informed-consent process attempts to define principles of risk disclosure that should generally meet the needs of most patients in most circumstances.

Informed-consent documents should not be considered all-inclusive with regard to defining other methods of treatment and potential risks encountered. Your surgeon will provide you with additional written and verbal information that is based on all of the facts of your particular case and the state of medical knowledge.

Informed-consent documents are not intended to define or serve as the standard of medical care. Standards of medical care are determined on the basis of all of the facts involved in an individual case and are subject to change as scientific knowledge and technology advance and as practice patterns evolve.

CONSENT FOR NECK LIPOSUCTION SURGERY

1. I hereby authorize my surgeon, _____, and such assistants as may be selected by my surgeon to perform the following procedure:
   - Neck region liposuction.
2. I have received, read, and understand the following information sheets:
   - Neck Liposuction Surgery
   - Consent for Neck Liposuction Surgery
3. I recognize that, during the course of the operation and the administration of anesthesia, unforeseen conditions may necessitate different procedures than those stated above. I therefore authorize my surgeon, _____, and my surgeon's assistants or designees to perform such other procedures that are in the exercise of professional judgment necessary and desirable. The authority granted under this paragraph shall include all conditions that require treatment and that are not known to my surgeon at the time that the procedure is begun.
4. I consent to the administration of such anesthetics as are considered necessary or advisable. I understand that all forms of anesthesia involve risk and the possibility of complications, injury, and even death.
5. I acknowledge that no guarantee has been given by anyone with regard to the results that may be obtained from this procedure.
6. I consent to the photographing of me (head and neck region) before, during, and after surgery. These photographs may be used by my surgeon for medical, scientific, or educational purposes now and in the future.
7. For the purposes of advancing medical education, I consent to the admittance of observers into the operating room.
8. I consent to the disposal of any body tissues or medical devices that may be removed.
9. **THE FOLLOWING INFORMATION HAS BEEN EXPLAINED TO ME IN A WAY THAT I UNDERSTAND:**
   - **THE PREVIOUSLY DESCRIBED PROCEDURES TO BE UNDERTAKEN**
   - **ALTERNATIVE PROCEDURES AND METHODS OF TREATMENT THAT I HAVE DECIDED AGAINST**
   - **THE SPECIFIC RISKS AND POTENTIAL COMPLICATIONS OF THE PROCEDURES THAT I PLAN TO UNDERGO**

I CONSENT TO THE PROCEDURES AND THE ABOVE LISTED ITEMS (1 THROUGH 9). I AM SATISFIED WITH THE EXPLANATION GIVEN TO ME.

_____	_____
Patient (or Person Authorized to Sign for Patient)	Date
_____	_____
Witness	Date

Anterior Neck Lift Surgery

Liposuction and direct excision are surgical techniques that are used for the removal of excess fatty tissue. They are appropriate procedures to use in the neck region, and they may be carried out through a small incision below the chin. During liposuction, a cannula (i.e., a thin tube with an opening near the end) is inserted, and the surgeon moves it back and forth under the skin while high-pressure suction removes the unwanted fat.

Depending on the quality of your skin and underlying muscles, *an anterior neck lift* procedure may be beneficial. An anterior neck lift is carried out through the same small incision as liposuction. By elevating the neck skin away from the deeper structures, there is a tendency for the skin to tighten in a positive way during the healing process. If skin laxity is extensive, then a more traditional face lift procedure (i.e., with incisions in front of and behind the ears) would be beneficial to directly remove the excess skin.

Just deep to the skin of the neck, there is a thin, sheet-like muscle layer (i.e., the platysma muscles) that may also become lax and benefit from surgical tightening. Platysma muscle tightening (i.e. platysmaplasty) can be performed in conjunction with cervical (neck) skin undermining as described to further improve the shape and contour of the neck.

After the *anterior neck lift* procedures, (i.e., cervical flap undermining, fat removal, and platysmaplasty), a tight-fitting dressing is usually applied to limit swelling and to help the skin conform more smoothly to the new shape of the underlying tissue. Within 1 week, the stitches are removed. Depending on whether other procedures are carried out simultaneously, most people then return back to their work environment.

INSTRUCTIONS FOR ANTERIOR NECK LIFT SURGERY

The purpose of these instructions is to help you to prepare for and then recover from your operation with as little discomfort and inconvenience as possible.

Preoperative Instructions

1. Do not take any aspirin, aspirin-containing products, or aspirin-like products for at least 2 weeks before and 2 weeks after surgery. Aspirin and aspirin-like products tend to increase bleeding during surgery and bruising postoperatively.
2. You should not smoke for at least 2 weeks before surgery and 2 weeks afterward. Smoking jeopardizes wound healing, it may result in lung congestion, and it will hinder optimal results.
3. Starting at least 2 days before surgery, we request that you shower and shampoo your hair at least once per day. The morning of surgery, shower and shampoo your hair again.
4. Do not eat or drink anything after midnight the night before surgery, not even a sip of water.
5. Be sure to make arrangements for someone to accompany you home after surgery, because you will not be allowed to drive.
6. We will give you a prescription for pain control and another for antibiotics. You may wish to fill them before the date of surgery.

Continued

Postoperative Instructions

1. Applying a cold compress to the anterior neck region during the initial 36 hours after surgery will reduce swelling and discomfort.
2. Keep your head and upper back elevated when resting and sleeping for the first week after surgery.
3. You may eat whatever you wish, but start out slowly, because the anesthesia may leave your stomach unsettled for several hours.
4. Maintaining some compression in the neck region after surgery may be helpful to minimize and reduce swelling and for the healing of the skin to the deeper structures of the neck. Each situation is unique, and your surgeon will advise you with regard to this matter.
5. For men, shaving with an electric razor may be preferred until you become comfortable with the changes in sensation in the neck skin region.
6. Get lots of rest and perform only minimal physical activity during the first week after surgery. You may then gradually increase your activity back to normal by 4 to 6 weeks after your operation as per instructions from your surgeon.
7. Palpable small lumps and a tight feeling in the neck are common findings. These will gradually improve during the final phase of healing.
8. Call the office for an appointment to see your surgeon on the date specified at the time of discharge.
9. If there are concerns at any time, contact your surgeon's office. After hours, the answering machine message will advise you about how to proceed.

Jeffrey C. Posnick, DMD, MD
Director, Posnick Center for Facial Plastic Surgery

Consent for Anterior Neck Lift Surgery

_____ Initial here

INSTRUCTIONS

This is a document that has been prepared by your surgeon to help educate and inform you about your anterior neck lift surgery and its risks as well as alternative treatment options. It is important that you read this document carefully and completely. Please initial each section to indicate that you have read and understand it. In addition, please sign the consent for surgery on the last page as proposed by your surgeon.

_____ Initial here

INTRODUCTION

Liposuction is a surgical technique to remove excess fatty tissue. It is an appropriate procedure to use in the neck region, and it is carried out through a small incision below the chin. During liposuction, a cannula (i.e., a thin tube with an opening near the end) is inserted, and the surgeon moves it back and forth under the skin while high-pressure suction removes the unwanted fat.

Depending on the quality of your skin and underlying muscles an anterior neck lift procedure may be beneficial. The anterior neck lift is carried out through the same small incision just below the chin. By elevating the neck skin away from the deeper structures, there is a tendency for the skin to tighten in a positive way. If skin laxity is extensive, then a more traditional face lift procedure may be required to directly remove the excess skin.

Just deep to the skin of the neck, there is a thin, sheet-like muscle layer (i.e., the platysma muscles) that may also become lax and benefit from surgical tightening. Platysma muscle tightening (platysmaplasty) can be done in conjunction with cervical (neck) skin undermining and fat removal as described to further improve the shape and contour of the neck.

_____ Initial here

ALTERNATIVE TREATMENT

Alternative forms of management may include the decision to not treat the neck soft-tissue concerns with surgery at all. Diet and exercise regimens may be of benefit for the overall reduction of excess body fat. The direct removal of excess skin (i.e., a traditional face lift) may be advantageous in addition to the fat removal and skin redraping procedures (i.e., the anterior neck lift) that you are currently planning to undergo. Risks and potential complications are also associated with these and other alternative forms of treatment.

_____ **RISKS OF NECK REJUVENATION PROCEDURES**

Initial here Every surgical procedure involves a certain amount of risk. It is important that you understand the risks involved with the anterior neck rejuvenation procedure that is planned for you. An individual's choice to undergo a surgical procedure is based on the comparison of the risk to the potential benefit. Although the majority of patients do not experience these complications, you should discuss each of them with your surgeon.

_____ **PATIENT SELECTION**

Initial here Individuals with poor skin tone, medical problems, obesity, or unrealistic expectations may not be candidates for a neck rejuvenation procedure.

_____ **BLEEDING**

Initial here It is possible, although not usual, to have a significant bleeding episode during or after surgery. If postoperative bleeding occurs, it might require urgent treatment to drain the accumulated blood or to control any ongoing bleeding. Do not take any aspirin or anti-inflammatory medications for 2 weeks before surgery, because this may increase the risk of bleeding. Individuals with poorly controlled hypertension are also at risk for postsurgical bleeding.

_____ **INFECTION**

Initial here Infection is not usual after this type of surgery, and you will be given perioperative antibiotics to limit this risk. If an infection occurs, a change in antibiotics and additional surgery may be necessary.

_____ **SKIN SCARRING**

Initial here Although good wound healing after neck rejuvenation is expected, abnormal scars may occur within the skin and the deeper tissues. Additional treatments, including surgery, may be beneficial to treat abnormal scarring.

_____ **CHANGE IN SKIN SENSATION AND LIP FUNCTION**

Initial here A decrease in skin sensation after neck rejuvenation may occur. Diminished neck skin sensation may not totally resolve.

_____ **SKIN DISCOLORATION AND SWELLING**

Initial here Skin discoloration and swelling normally occur after neck rejuvenation. In rare situations, swelling and skin discoloration may persist.

_____ **SKIN CONTOUR IRREGULARITIES**

Initial here Contour irregularities, or palpable wrinkling may occur in the skin after neck rejuvenation. Additional treatments, including surgery, may be beneficial to manage skin contour irregularities.

_____ **NECK ASYMMETRY**

Initial here It may not be possible to achieve a symmetric appearance with neck rejuvenation procedures. Factors such as skin or muscle tone and bony prominences may also contribute to a noticeable asymmetries.

_____ **SEROMA**

Initial here Fluid accumulation infrequently occurs in areas where neck rejuvenation has been performed. Additional treatments or surgery to drain accumulations of fluid may be necessary.

Continued

_____ **LONG-TERM EFFECTS**

Initial here Subsequent alterations in facial contour may occur as a result of aging, weight loss or gain, or other circumstances not related to the specific procedures that have been carried out.

_____ **SURGICAL ANESTHESIA**

Initial here Both local and general anesthesia involve risk. Complications are possible with all forms of anesthesia and sedation.

_____ **ALLERGIC REACTION**

Initial here Local allergies to tape, suture materials, or topical preparations may also occur. Systemic reactions, which are more serious, may occur in response to drugs that are used during surgery or prescription medication taken afterward.

_____ **HEALTH INSURANCE**

Initial here Most health insurance companies exclude coverage for cosmetic operations such as neck rejuvenation or any related complications that may occur. Please carefully review your health insurance subscriber information.

_____ **ADDITIONAL SURGERIES THAT MAY BE NECESSARY**

Initial here There are many variable conditions in addition to risks and complications that may influence the long-term results of a neck rejuvenation procedure. Although complications occur infrequently, the risks cited are those that are particularly associated with a neck rejuvenation procedure. Other complications and risks can occur but are even more uncommon. If complications do occur, additional surgeries or other treatments may be necessary. The practice of medicine and surgery is not an exact science. Although favorable results are expected, there is no guarantee or warranty expressed or implied with regard to the results that may be obtained.

_____ **FINANCIAL RESPONSIBILITIES**

Initial here The cost of surgery involves several charges for the services provided, including the fees charged by your surgeon (i.e., surgeon's fees); the cost of surgical supplies and the use of the operating room (i.e., facility/hospital charges); and the costs associated with anesthesia (i.e., anesthesiologist's fees). Depending on whether the cost of surgery is covered by your medical insurance plan, you will be responsible for necessary copayments, deductibles, and all other charges that are not covered. Additional costs may occur should complications develop from the surgery. Any secondary surgeries and all charges involved with additional procedures (i.e., surgeon's fees, facility/hospital charges, anesthesiologist's fees) would also be your responsibility.

_____ **DISCLAIMER**

Initial here Informed-consent documents are used to communicate information about the proposed treatment of a disease or condition as well as to disclose information about risks and alternative forms of treatment. The informed-consent process attempts to define principles of risk disclosure that should generally meet the needs of most patients in most circumstances.

However, informed-consent documents should not be considered all-inclusive with regard to defining other methods of care and potential risks encountered. Your surgeon may provide you with additional written and verbal information that is based on all of the facts of your particular case and the state of medical knowledge.

Informed-consent documents are not intended to define or serve as the standard of medical care. Standards of medical care are determined on the basis of all of the facts involved in an individual case and are subject to change as scientific knowledge and technology advance and as practice patterns evolve.

CONSENT FOR ANTERIOR NECK LIFT SURGERY

1. I hereby authorize my surgeon, _____, and such assistants as may be selected by my surgeon to perform the following procedure: neck rejuvenation surgery that includes the removal of fat, cervical (neck skin) undermining, and platysma muscle plication.
2. I have received, read, and understand the following information sheets:
   - Anterior Neck Lift Surgery
   - Consent for Anterior Neck Lift Surgery
3. I recognize that, during the course of the operation and the administration of anesthesia, unforeseen conditions may necessitate different procedures than those stated above. I therefore authorize my surgeon, _____, and my surgeon's assistants or designees to perform such other procedures that are in the exercise of professional judgment necessary and desirable. The authority granted under this paragraph shall include all conditions that require treatment and that are not known to my surgeon at the time that the procedure is begun.
4. I consent to the administration of such anesthetics as are considered necessary or advisable. I understand that all forms of anesthesia involve risk and the possibility of complications, injury, and even death.
5. I acknowledge that no guarantee has been given by anyone with regard to the results that may be obtained from this procedure.
6. I consent to the photographing of me (head and neck region) before, during, and after surgery. These photographs may be used by my surgeon for medical, scientific, or educational purposes now and in the future.
7. For the purposes of advancing medical education, I consent to the admittance of observers into the operating room.
8. I consent to the disposal of any body tissues or medical devices that may be removed.
9. **THE FOLLOWING INFORMATION HAS BEEN EXPLAINED TO ME IN A WAY THAT I UNDERSTAND:**
   - **THE PREVIOUSLY DESCRIBED PROCEDURES TO BE UNDERTAKEN**
   - **ALTERNATIVE PROCEDURES AND METHODS OF TREATMENT THAT I HAVE DECIDED AGAINST**
   - **THE SPECIFIC RISKS AND POTENTIAL COMPLICATIONS OF THE PROCEDURES THAT I PLAN TO UNDERGO**

I CONSENT TO THE PROCEDURES AND THE ABOVE LISTED ITEMS (1 THROUGH 9). I AM SATISFIED WITH THE EXPLANATION GIVEN TO ME.

_____	_____
Patient (or Person Authorized to Sign for Patient)	Date
_____	_____
Witness	Date

References

1. Bach DE, Newhouse RF, Boice GW: Simultaneous orthognathic surgery and cervicomental liposuction: Clinical and survey results. *Oral Surg Oral Med Oral Pathol* 71:262, 1991.
2. Baker DC: Lateral SMASectomy. *Plast Reconstr Surg* 100:509–513, 1997.
3. Baker DC: Minimal incision rhytidectomy (short scar facelift) with lateral SMAS-ectomy: Evolution and applications. *Aesthet Surg J* 21:14–26, 2001.
4. Baker DC, Nahai F, Massiha H, Tonnard P: Panel discussion: Short scar face lift. *Aesthet Surg J* 25:607–617, 2005.
5. Baker T, Stuzin J: Personal technique for facelifting. *Plast Reconstr Surg* 100:502–513, 1997.
6. Barlett SP, Grossman R, Whitaker LA: Age-related changes of the craniofacial skeleton: An anthropometric and histological analysis. *Plast Reconstr Surg* 90:592–600, 1992.
7. Barton FE: Aesthetic surgery of the face and neck. *Aesthet Surg J* 29:449–463, 2009.
8. Biggs TM: Excision of neck redundancy with single Z-plasty closure. *Plast Reconstr Surg* 98:1113–1114, 1996.
9. Chang S, Pusic A, Rohrich RJ: A systematic review of comparison of efficacy and complication rates among face-lift techniques. *Plast Reconstr Surg* 127:423, 2011.
10. Coleman SR, Grover R: The anatomy of the aging face: Volume loss and changes in 3 dimensional topography. *Aesthet Surg J* 26:S4–S9, 2006.
11. Connell BF, Shamoun JM: The significance of digastric muscle contouring rejuvenation of the submental area of the face. *Plast Reconstr Surg* 99:1586–1590, 1997.
12. Courtiss EH: Suction lipectomy of the neck. *Plast Reconstr Surg* 76:882–889, 1985.
13. Donofrio LM: Fat distribution: A morphologic study of the aging face. *Dermatol Surg* 26:1107–1112, 2000.
14. Doual JM, Ferri J, Laude M: The influence of senescence on craniofacial and cervical morphology in humans. *Surg Radiol Anat* 19:175–183, 1997.
15. El Domyati M, Attia S, Saleh F, et al: Intrinsic aging vs photoaging: A comparative histopathological, immunohistochemical, and ultrastructural study of skin. *Exp Dermatol* 11:398–405, 2002.
16. Enlow DH: *Handbook of facial growth*, Philadelphia, 1982, Saunders.
17. Farkas LG, Eiben OG, Sivkov S, et al: Anthropometric measurements of the facial framework in adulthood: Age-related changes

in eight age categories in 600 healthy white North Americans of European ancestry from 16 to 90 years of age. *J Craniofac Surg* 15:288–298, 2004.
18. Feldman JJ: *Neck lift*, St. Louis, MO, 2007, Quality Medical Publishing.
19. Fisher GJ, Varani V, Voorhees JJ: Looking older: Fibroblast collapse and therapeutic implications. *Arch Dermatol* 144:666–672, 2008.
20. Fitzgerald R, Graivier MH, Kane M, et al: Update on facial aging. *Aesthet Surg J* 30(Suppl 1):11S–24S, 2010.
21. Fuente del Campo A, Feldman JJ, Guyuron B, Hoeffin SM: Panel discussion: Treatment of the difficult neck. *Aesthet Surg J* 20:495–501, 2000.
22. Furnas DW: The retaining ligaments of the cheek. *Plast Reconstr Surg* 83:11–16, 1989.
23. Goddio AS: Skin retraction following suction lipectomy by treatment site: A study of 500 procedures in 458 selected subjects. *Plast Reconstr Surg* 87:66–75, 1991.
24. Gonzalez-Ulloa M, Flores ES: Senility of the face: Basic study to understand its causes and effects. *Plast Reconstr Surg* 36:239–246, 1965.
25. Gradinger GP: Anterior cervicoplasty in the male patient. *Plast Reconstr Surg* 106:1146–1154; discussion 1155, 2000.
26. Guerrerosantos J, Espaillat L, Morales F: Muscular lift in cervical rhytidoplasty. *Plast Reconstr Surg* 54:127, 1974.
27. Guyuron B: Problem neck, hyoid bone, and submental myotomy. *Plast Reconstr Surg* 90:830–837; discussion 838–840, 1992.
28. Guyuron B, Jackowe D, Iamphongsai S: Basket submandibular gland suspension. *Plast Reconstr Surg* 122:938–943, 2008.
29. Guyuron B, Rowe DJ, Weinfeld AB, et al: Factors contributing to the facial aging of identical twins. *Plast Reconstr Surg* 123: 1321–1331, 2009.
30. Hall G, Phillips TJ: Estrogen and skin: The effects of estrogen, menopause, and hormone replacement therapy on the skin. *J Am Acad Dermatol* 53:555–568, 2005.
31. Hamra S: The deep-plane rhytidectomy. *Plast Reconstr Surg* 86:53–61, 1990.
32. Hayes RJ, Sarver DM, Jacobson AJ: Quantification of cervicomental angle changes with mandibular advancement. *Am J Orthod Dentofacial Orthop* 105: 383–391, 1994.
33. Kahn DM, Shaw RB: Aging of the bony orbit: A three-dimensional computed tomographic study. *Aesthet Surg J* 28:258–264, 2008.
34. Kesselring UK: Direct approach to the difficult anterior neck region. *Aesthetic Plast Surg* 16:277–282, 1992.
35. Knize DM: Limited incision submental lipectomy and platysmaplasty. *Plast Reconstr Surg* 101:473–481, 1998.
36. Knize DM: Limited incision submental lipectomy and platysmaplasty. *Plast Reconstr Surg* 113:1275–1278, 2004.
37. Lambros V: Observations on periorbital and midface aging. *Plast Reconstr Surg* 120: 1367–1376, discussion 1377, 2007.
38. Lambros V: Models of facial aging and implications for treatment. *Clin Plast Surg* 35:319–327, discussion 317, 2008.
39. Le Louarn CL, Buthiau D, Buis J: Structural aging: The facial recurve concept. *Aesthetic Plast Surg* 31:213–218, 2007.
40. Levine RA, Garza JR, Wang PT, et al: Adult facial growth: Application to aesthetic surgery. *Aesthetic Plast Surg* 7:265–283, 2003.
41. Levy PM: The "Nefertiti lift": A new technique for specific re-contouring of the jawline. *J Cosmet Laser Ther* 9:249–252, 2007.
42. Little JW: Volumetric perceptions in midfacial aging with altered priorities for rejuvenation. *Plast Reconstr Surg* 105:252, 2000.
43. Marten TJ, Feldman JJ, Connell BF, Little WJ: Panel discussion: Treatment of the full obtuse neck. *Aesthet Surg J* 25:387–397, 2005.
44. Mendelson BC, Freeman ME, Wu W, Huggins RJ: Surgical anatomy of the lower face: The premasseter space, the jowl, and the labiomandibular fold. *Aesthetic Plast Surg* 32:185–195, 2008.
45. Mendelson BC, Hartley W, Scott M, et al: Age-related changes of the orbit and midcheek and the implications for facial rejuvenation. *Aesthetic Plast Surg* 31:419–423, 2007.
46. Millard DR, Pigott R, Hedo A: Submental lipectomy. *Plast Reconstr Surg* 41:513, 1968.
47. Miller T: Face lift: Which technique? *Plast Reconstr Surg* 100:501, 1997.
48. Mitz V, Peyronie M: The superficial musculo-aponeurotic system (SMAS) in the parotid and cheek area. *Plast Reconstr Surg* 58:80–88, 1976.
49. Moss JC, Mendelson BC, Taylor GI: Surgical anatomy of the ligamentous attachments in the temple and periorbital regions. *Plast Reconstr Surg* 105:1475–1490, 2000.
50. Nahai F: Neck lift. In Nahai F, editor: *The art of aesthetic surgery: Principles and techniques,* Vol II, St Louis, MO, 2005, Quality Medical Publishing Inc, pp 1239–1283.
51. Pessa JE: The tear trough and lid/cheek junction: Anatomy and implications for surgical correction. *Plast Reconst Surg* 123:1332–1340, 2009.
52. Pessa JE: An algorithm of facial aging: Verification of Lambros's theory by three-dimensional stereolithography, with reference to the pathogenesis of midfacial aging, scleral show, and the lateral suborbital trough deformity. *Plast Reconstr Surg* 106:479–488, 2000.
53. Pessa JE, Chen Y: Curve analysis of the aging orbital aperture. *Plast Reconstr Surg* 109: 751–755, discussion 756–760, 2002.
54. Pessa JE, Desvigne LD, Zadoo VP: The effect of skeletal remodeling on the nasal profile: Considerations for rhinoplasty in the older patient. *Aesthetic Plast Surg* 23:239–242, 1999.
55. Pessa JE, Slice DE, Hanz KR, et al: Aging and the shape of the mandible. *Plast Reconstr Surg* 121:196–200, 2008.
56. Pessa JE, Zadoo VP, Mutimer KL, et al: Relative maxillary retrusion as a natural consequence of aging: Combining skeletal and soft-tissue changes into an integrated model of midfacial aging. *Plast Reconstr Surg* 102: 205–212, 1998.
57. Pessa JE, Zadoo VP, Yuan C, et al: Concertina effect and facial aging: Nonlinear aspects of youthfulness and skeletal remodeling, and why, perhaps, infants have jowls. *Plast Reconstr Surg* 103:635–644, 1999.
58. Pitman G, Aston SJ, Feldman JJ, LaFerriere K: Panel discussion: Revisional neck surgery. *Aesthet Surg J* 27:527–538, 2007.
59. Pontes R: Extended dissection of the mental area in face-lift operations. *Ann Plast Surg* 27:439, 1991.
60. Rabe JH, Mamelak AJ, McElgunn PJ, et al: Photoaging: Mechanisms and repair. *J Am Acad Dermatol* 55:1–19, 2006.
61. Ramirez OM: Cervicoplasty: Nonexcisional anterior approach. *Plast Reconstr Surg* 99:1576–1585, 1997.
62. Raskin E, LaTrenta GS: Why do we age in our cheeks? *Aesthet Surg J* 27:19–28, 2007.
63. Reece EM, Pessa JE, Rohrich RJ: The mandibular septum: anatomical observations of the jowls in aging—implications for facial rejuvenation. *Plast Reconstr Surg* 121: 1414–1420, 2008.
64. Richard MJ, Morris C, Deen B, et al: Analysis of the anatomic changes of the aging facial skeleton using computer-assisted tomography. *Ophthal Plast Reconstr Surg* 25:382–386, 2009.
65. Rohrich RJ, Arbique GM, Wong C, et al: The anatomy of suborbicularis fat: implications for periorbital rejuvenation. *Plast Reconstr Surg* 124:946–951, 2009.
66. Rohrich RJ, Pessa JE: The fat compartments of the face: Anatomy and clinical implications for cosmetic surgery. *Plast Reconstr Surg* 119:2219–2227, discussion 2228–2231, 2007.
67. Rohrich RJ, Pessa JE: The anatomy and clinical implications of perioral submuscular fat. *Plast Reconstr Surg* 124:266–271, 2009.
68. Rohrich RJ, Pessa JE, Ristow B: The youthful cheek and the deep medial fat compartment. *Plast Reconstr Surg* 121:2107–2112, 2008.
69. Rosen HM: Facial skeletal expansion: Treatment strategies and rationale. *Plast Reconstr Surg* 89:798–808, 1992.
70. Sandoval S, Cox J, Koshy J, et al: Facial fat compartments: A guide to filler placement. *Semin Plast Surg* 23:283–287, 2009.
71. Sarver DM, Rousso DR: Plastic surgery combined with orthodontic and orthognathic procedures. *Am J Orthod Dentofacial Orthop* 126:305–307, 2004.
72. Schaverien MV, Pessa JE, Rohrich RJ: Vascularized membranes determine the

anatomical boundaries of the subcutaneous fat compartments. *Plast Reconstr Surg* 123: 695–700, 2009.
73. Sharabi S, Hatef D, Koshy J: Mechanotransduction: The missing link in the facial aging puzzle? *Aesthetic Plast Surg* 34:603–611, 2010.
74. Shaw RB, Katzel EB, Koltz PF, et al: Aging of the mandible and its aesthetic implications. *Plast Reconstr Surg* 125:332–342, 2010.
75. Shaw RB, Jr, Kahn DM: Aging of the midface bony elements: A three-dimensional computed tomographic study. *Plast Reconstr Surg* 119:675–681, 2007.
76. Singer DP, Sullivan PK: Submandibular gland I: an anatomic evaluation and surgical approach to submandibular gland resection for facial rejuvenation. *Plast Reconstr Surg* 112:1150–1154, 2003.
77. Singer DP, Sullivan PK: Submandibular gland I: An anatomic evaluation and surgical approach to submandibular gland resection for facial rejuvenation. *Plast Reconstr Surg* 112:1150–1154; discussion 1155–1156, 2003.
78. Stuzin JM: Restoring facial shape in face lifting: The role of skeletal support in facial analysis and midface soft tissue repositioning. *Plast Reconstr Surg* 119:362–376, discussion 377–378, 2007.
79. Stuzin JM, Baker DC, Feldman J, Marten TJ: Panel discussion: Cervical contouring in facelift. *Aesthet Surg J* 22:541–548, 2002.
80. Stuzin JM, Baker TJ, Gordon HL: The relationship of the superficial and deep facial fascias: Relevance to rhytidectomy and aging. *Plast Reconstr Surg* 89:441–451, 1992.
81. Stuzin JM, Wagstrom L, Kawamoto HK, et al: The anatomy and clinical implications of the buccal fat pad. *Plast Reconstr Surg* 85:29–37, 1990.
82. Sullivan PK, Freeman MB, Schmidt S: Contouring the aging neck with submandibular gland suspension. *Aesthet Surg J* 26:465–471, 2006.
83. Teimourian B: Face and neck suction-assisted lipectomy associated with rhytidectomy. *Plast Reconstr Surg* 72:627, 1983.
84. Wang F, Garza LA, Kang S, et al: In vivo stimulation of de novo collagen production caused by cross-linked hyaluronic acid dermal filler injections in photodamaged human skin. *Arch Dermatol* 143:155–163, 2007.
85. Yaar M, Gilchrest BA: Skin aging: Postulated mechanisms and consequent changes in structure and function. *Clin Geriatr Med* 17:617–630, 2001.
86. Yaremchuk MJ: Mandibular augmentation. *Plast Reconstr Surg* 106:697–706, 2000.
87. Yaremchuk MJ: Infraorbital rim augmentation. *Plast Reconstr Surg* 107: 1585–1592; discussion 1593–1595, 2001.
88. Yaremchuk MJ: Making concave faces convex. *Aesthetic Plast Surg* 29:141–147; discussion 148, 2005.
89. Yaremchuk MJ: Indications, evaluation and planning. In Yaremchuk MJ, editor: *Atlas of Facial Implants*, Philadelphia, 2007, Saunders-Elsevier, pp 3–22.
90. Yaremchuk MJ, Chen YC: Enlarging the deficient mandible. *Aesthet Surg J* 27:539–550, 2007.
91. Yaremchuck M, Doumit G, Thomas MA: Alloplastic augmentation of the facial skeleton: An occasional adjunct or alternative to orthognathic surgery. *Plast Reconstr Surg* 127:2021–2030, 2011.
92. Yaremchuk MJ, Israeli D: Paranasal implants for correction of midface concavity. *Plast Reconstr Surg* 102:1676–1684; discussion 1685, 1998.
93. Zadoo VP, Pessa JE: Biological arches and changes to the curvilinear form of the aging maxilla. *Plast Reconstr Surg* 106:460–466, 2000.
94. Zins JE, Fardo D: The "anterior-only" approach to neck rejuvenation: An alternative to face lift surgery. *Plast Reconstr Surg* 115:1761–1768, 2005.
95. Zins JE, Menon N: Anterior approach to neck rejuvenation. *Aesthet Surg J* 30:477–484, 2010.

Index

A
Accelerated osteogenic orthodontics, 164-165
Acetaminophen, 142-143
Achondroplasia, 91
Acid-etch techniques, 162
Activated appliances, 142. *See also* Appliances.
Active self-ligating brackets, 161. *See also* Brackets.
Active unilateral condylar hyperactivity, 860-861
Acromegaly, 102
Adequate face-mask ventilation, 319
Adhesives and cements, 162
Adult dentofacial disharmony, 945-991. *See also* Dentofacial disharmonies.
  comprehensive dental rehabilitation and, 982
  facial aging and rejuvenation, 983-986
    craniofacial morphologic changes, 985-986
    mandibular skeletal changes, 986
    midface aging, 984-985
    morphologic differentiation, 985-986
    periorbital aging, 984-985
    special considerations for, 983-984
  functional assessments for, 945-981
  previous suboptimal facial surgery and, 983
  recovery considerations, 987-988
    body dysmorphic disorder, 987
    clear objective setting strategies, 987
    dysesthesia, 987-988
    nerve injuries, inferior alveolar, 987
    nerve injuries, infraorbital, 987-988
    nerve injuries, mental, 987
    patient age, 988
    physiologic effects, 987
    psychological effects, 987
    realistic expectations and, 987
    sensory loss, 987-988
    sensory recovery, 988
  special medical considerations for, 945
  techniques for, 945, 946f-981f
Adult dentofacial disharmony *(Continued)*
  temporomandibular disorders (TMDs) and, 981-982. *See also* Temporomandibular disorders (TMDs).
  treatment considerations for, 982-983
  wound healing risk factors for, 982
Aesthetics
  aesthetic alterations
    chin, 1564-1612
    ears, prominent, 1703-1745
    fundamentals of, 337-351
    soft tissues, face *vs.* neck, 1746-1783
  aesthetic ideals, facial, 337-351. *See also* Ideal facial aesthetics.
  aesthetic objectives, prominent ears, 1706. *See also* Prominent ears.
  aesthetic units
    fronto-forehead, 1230
    maxillary/nasal base, 1230
    orbito-naso-zygomatic, 1230
    posterior cranial vault, 1230
Affricative airstream obstructions, 236
Age and aging considerations
  age-related macular degeneration, 12
  facial aging, 983-986
  periorbital aging, 984-985
  recovery issues, 988
AHI. *See* Apnea-hypopnea index (AHI).
Airstream obstructions, affricative, 236
Airways
  benchmark parameters checklist, 323
  continuous positive airway pressure (CPAP), 1009
  difficult, 319
  management techniques, 308-336
    back-up plans, 318-319
    decision factors for, 317, 318f
    dentofacial deformity considerations for, 318f, 319-320
    difficult airway guidelines, 319
    extubation, benchmark airway parameters checklist for, 323
    extubation, criteria for, 321-322
    extubation, planning for, 322
    extubation, strategies for, 321
    face-mask ventilation, adequate, 319
    head examinations, 317-318
    intubation strategies, 319
Airways *(Continued)*
    lower jaw forward protrusion, inability to, 317
    Mallampati test, 318
    mouth-opening ability, 317
    neck examinations and, 317-318
    neck extension, limited, 317-318
    oxygenation maintenance, 318
    perioperative, 317-323
    preintubation preparation, 320-321
    presurgical airway evaluations, 319
    thyromental distance, 318
    tracheal intubation, complication prevention and, 318-319
  nasal, 287-307. *See also* Nasal airway considerations.
    case study of, 298f-300f, 302-304
    chronic obstructive nasal breathing, 287-295
    COG-ENT chronic rhinosinusitis study, 301
    historical perspectives of, 287
    Le Fort I down-fracture osteotomies, 287-288, 293f-295f, 296-297
    maxillary arch expansion, 290f, 297-301
    nasal floor recontouring, 290f, 295f, 297-301, 298f-300f
    Nasal Obstruction Symptom Evaluation (NOSE) survey, 296
    quality-of-life issues, 301
    residual postoperative nasal obstruction, 297
Alberti, Leon Battista, 345
Alexander the Great, 341-342, 341f
Alginate impressions, 380f-385f, 415
Allogenic grafts, 622-623. *See also* Grafts and grafting.
Allografts, 608. *See also* Grafts and grafting.
Alloplastic grafts, 620-622, 636. *See also* Grafts and grafting.
Alloy
  arch wires, 162
  brackets, 142
Alterations, aesthetic. *See also* Aesthetics.
  chin, 1564-1612
  ears, prominent, 1703-1745
  fundamentals of, 337-351
  soft tissues, face *vs.* neck, 1746-1783
Alveolar bone, 177-178
American Journal of Dental Science (1849), 26
Analytic model-planning. *See also* Planning techniques.
  mandible-first, 424-440. *See also* Mandible-first analytic model-planning.
    advantages of, 424, 425f-426f
    case studies, hemifacial microsomia (HFM), 339f, 365b, 430b, 430f-436f, 433b. *See also* Hemifacial microsomia (HFM).
    centric relation (CR), 424-427
    clinical settings for, 439
    execution of, 428-429
    technical aspects of, 428-429
    techniques for, 429
  standard, 374-423. *See also* Standard analytic model-planning.
    accuracy of, 389f-393f, 406-407
    alginate impressions, 380f-385f, 415
    cast impressions, 380f-385f, 415
    components of, 374, 375f-404f
    controversies of, 405-408
    direct visual examinations, 408
    Erickson Model Table for, 389f-393f, 406-407. *See also* Erickson Model Table.
    face-bow registration and transfer, 375f-385f, 408-416, 409f-415f
    goals of, 374-405, 420
    intermediate splint contraction, maxilla positioning, 417-418
    key measurements, 408
    key steps for, 375f-393f, 407
    reorientation. mandibular cast, 418
    reorientation, maxilla, 417
    requirements of, 406-407
    segmentation, maxilla, 417
    semi-adjustable articulators, 408-416
    spatial relationship captures, 408-416
    standard facial records, 408
    studies of, 419-420
Anatomic crowns, 172, 173f
Anatomic roots, 172, 173f
Anchorage devices, temporary (TADs), 142-143
Anchorage roles, 143
Ancient Greek scholars, 18-20
Andrews, Lawrence F., 24-25, 24f

Note: Page numbers followed by "b" indicate boxes; "f" figures; "t" tables.

Andrews' standard analyses, 363-367
Anesthesia and related considerations, 308-336
  blood replacement needs and, 313-317
    approaches to, 313
    complex surgeries, 315t-316t
    controversies in, 314-317
    historical perspectives of, 314
    studies of, 314
  convalescence, at home/outpatient and, 325
    outpatient regimens, 325
    patient education materials for, 326
    physical therapy, 328b-330b
  convalescence, in-hospital and, 323-325
    airway needs, 323
    antibiotics, 325
    diet and nutrition, 324
    discharge criteria, 325
    intravenous (IV) fluid therapy, 324
    monitoring equipment, 323-324
    nasal medications, 325
    nausea management, 324
    pain management, 325
    settings for, 323
    vomiting management, 324
    wound care, 324
  deliberate hypotensive anesthesia (DHA), 308-313
    complications of, 310
    historical perspectives of, 308-309, 311-313
    inducement techniques for, 309
    mean arterial pressure (MAP) and, 309, 318
    monitoring during, 310
    organ function effects of, 309-310
    orthognathic surgery-specific effects of, 310-313
    patient positioning and, 309
  hemorrhage depressors and, 311
    blood loss limitation effects of, 311
    desmopressin, 311
    tranexamic acids, 311
  perioperative airway management and, 317-323. *See also* Perioperative airway management.
    back-up plans, 318-319
    decision factors for, 317, 318f
    dentofacial deformity considerations for, 318f, 319-320
    difficult airway guidelines, 319
    extubation, benchmark airway parameters checklist for, 323
    extubation, criteria for, 321-322
    extubation, planning for, 322
    extubation, strategies for, 321
    face-mask ventilation, adequate, 319
    head examinations, 317-318
Anesthesia and related considerations *(Continued)*
    immediate preintubation preparation, 320-321
    intubation strategies, 319
    lower jaw forward protrusion, inability to, 317
    Mallampati test, 318
    mouth-opening ability, 317
    neck examinations and, 317-318
    neck extension, limited, 317-318
    oxygenation maintenance, 318
    presurgical airway evaluations, 319
    thyromental distance, 318
    tracheal intubation, complication prevention and, 318-319
    upper airway trauma prevention, 318
Angle, Edward H., 20-23, 20f-22f
Anterior limit lines, 363-367, 364f-365f
Anthropometric considerations, 1159
Anthropometric surface analyses, 363
Antibiotics, 325
Antihelical folds
  creation *vs*. deepening, 1709f-1710f, 1712-1713
  marking of, 1712
  too-sharp, 1714
Apert syndrome, 1184-1200, 1190f-1193f, 1707f-1709f
  obstructive sleep apnea (OSA) and, 996. *See also* Obstructive sleep apnea (OSA).
  reconstruction staging for, 1251-1254
  skeletal growth, environmental and neuromotor effects on, 115
Apex, root, 172, 173f
Apnea-hypopnea index (AHI), 992-1000
Apnea, sleep, 992-1058. *See also* Obstructive sleep apnea (OSA).
  airway management and, 1002
  central sleep apnea, 1000
  in childhood, 1000-1002
  clinical characteristics of, 993-1000, 995f, 997f-998f
  clinical evaluations for, 997-999
  diagnoses of, 993-1000
  etiology of, 992
  functional obstruction sites, 995f, 997f, 1000
  prevalence of, 992
  public health burden of, 1049
  risk factors for, 996-997
  severity of, 993-1000
  studies of, 992
  treatment options for, 1011f-1045f
  treatment rationales for, 993
A-point to B-point relationships, 367
Appliances. *See also* Orthodontic considerations.
  activated, 142
Appliances *(Continued)*
  fixed, 156-163
    arch wires, 162
    auxiliary attachments, 161
    banding *vs*. bonding, 161-162
    bracket materials of, 161-162
    cements and adhesives, 162
    clear brackets, 161
    design of, 161-162
    edgewise appliances, 156-161
    historical perspectives of, 156-161
    intra-oral bracket auxiliaries, 162-163
    prescriptions for, 161
    rotational control of, 161
  removable, 163-164
    clinical judgment and, 164
    functional appliances, 163-164
    historical perspectives of, 163
    Invisalign, 164
    retention appliances, 164
    vs. temporary skeletal anchorage (TADs), 166
Applied orthodontic force, 142
Arch
  maxillary
    constriction, 640-685
    expansion, 290f, 297-301
  syndromes, brachial, 73-77, 76f-77f, 1095
  wires, 162
Architectural orders, Greek, 342, 343f-344f
Aristotle, 18-20, 340-341
Articulation tests, 237, 237b
Articulators, semi-adjustable, 408-416
Ascending ramus defects, 623-626
Aseptic necrosis, 506-508, 506f-507f
Assessment-related considerations
  clinical evaluations, 351-369. *See also* Planning techniques.
    approaches to, 351
    global head and neck assessments, 351-360. *See also* Global head and neck assessments.
      cervical spine range-of-motion (ROM), current, 360
  mastication factors, 227-263
  nasal airway factors, 287-307. *See also* Airways.
  orthodontic factors, 135-170
  psychosocial factors, 209-226
  seven critical aspects, 351-360
  speech-related factors, 227-263
  swallowing factors, 227-263
Associated complications, 475-542. *See also* Complication-related considerations.
  for chin, oblique osteotomies, 525-527
  fixation factors, 479-481, 481f
  general factors, 475-486
  patient education materials for, 527
  risk factors, 475-486
Associated complications *(Continued)*
  for sagittal split ramus osteotomies (SSROs), mandibular, 486-504
Astragalizontes (Polykleitos), 338-339, 338f-339f
Asymmetric Class III dentofacial deformity, 807. *See also* Asymmetric mandibular excess.
Asymmetric mandibular excess, 807-865. *See also* Excess patterns.
  clinical characteristics and types of, 807, 809f-853f
  condylar hyperactivity
    active unilateral, 860-861
    type I, 854-855
    type II, 855-860
  etiology of, 807, 809f-853f, 863
  hemimandibular elongation, 855-860
  hemimandibular hyperplasia (HMH), 854-855
  high partial condylectomy, 860-861
  jaw growth maturity assessments, 860
  misdiagnosis of, 807, 808f
  presurgical orthodontic treatments, 861
  studies of, 853
  surgical approaches, 861-862
    chin asymmetry influences, 861
    complications of, 862
    informed consent considerations for, 862
    malocclusion and, 862
    patient education for, 862
    temporomandibular disorders (TMDs) and, 862-863
At home/outpatient convalescence, 325. *See also* Convalescence.
  anesthesia considerations and, 308-336. *See also* Anesthesia and related considerations.
  outpatient regimens, 325
  physical therapy, 328b-330b
Attached gingiva, 174-176, 174f-175f
Attachment apparatus lesions, 180
Attended polysomnography, 999
Auditory distortion speech errors, 237-240
Augmentation implants, 636
Autogenous blood banking, 313
Autogenous iliac donor sites, 609-614. *See also* Donor sites.
Autogenous rib donor sites, 623-626. *See also* Donor sites.
Autografts, 608. *See also* Grafts and grafting.
Auxiliary attachments, 161
Avascular age-related macular degeneration, 12
Avascular necrosis theory, 1531
Axis, ear, 1706

B

Background perspectives. *See* Historical perspectives.
Back-up plans, 318-319
Bacterial plaque, 181

Bad split, 477
BAHA options. *See* Bone-anchored hearing aid (BAHA) options.
Bandeau, 18
Banding *vs*. bonding, 161-162
Bandolet, 18
Banking blood, 313
Bariatric surgery, 1002-1008
Barrow, G.V., 28
Baseline eating disorders, 218
Baseline maxillomandibular skeletal disharmony, 1748f, 1749, 1750f-1754f
Baseline mental health, 218
Basic concepts
  blood loss and fluid replacement, 308-336
  convalescence, 308-336
  definition and prevalence, 61-68
  facial perspectives, 1-17
  formation influences, 69-134
  historical perspectives, 18-60
  mastication considerations, 227-263
  nasal airway considerations, 287-307
  orthodontic considerations, 135-170
  periodontal considerations, 171-208
  psychosocial considerations, 209-226
  speech-related considerations, 227-263
  swallowing considerations, 227-263
  techniques
    airway management, 308-336
    anesthesia, 308-336
  temporomandibular disorders (TMDs), 264-286
BCLP. *See* Bilateral cleft lip/palate (BCLP).
BDD. *See* Body dysmorphic disorder (BDD).
Beauty, 337
Beauty, definitions of, 337
Begg, Raymond, 24, 24f
Behaviors, high-risk, 218
Bell, William H., 32, 32f, 41-47
Benchmark airway parameters checklist, 323. *See also* Airways.
Bending response, 137
Bilateral cleft lip/palate (BCLP), 1371-1440. *See also* Cleft-orthognathic surgeries.
  in adolescents, 1373-1428
  coordinated team approach, 1372
  facial growth implications of, 1371-1372
  in infants, 1371-1372
  jaw deformities and, 1428
  orthodontic approaches, 898, 1428
  orthognathic approaches, 1374f-1427f, 1429-1431
    alveolar cleft management, 1431
    chin deformity management, 1432
    controversies of, 1433-1436
Bilateral cleft lip/palate (BCLP) *(Continued)*
    distraction osteogenesis (DO), 1373, 1436
    down-fracture, cleft maxilla, 1431
    evolution of, 1429
    horizontal advancement, cleft maxilla, 1431
    initial surgical healing, 1432
    large residual palatal fistula, closure of, 1433-1434
    Le Fort I osteotomies, modified, 1430-1431
    Le Fort I osteotomies, standard, 1429-1430
    Le Fort I osteotomies, three-segment, 1430-1431
    mandibular deformity, management of, 1432
    maxillary reconstruction, staging of, 1433
    mobilization, cleft maxilla, 1431
    nasal cavity, management of, 1431-1432
    nose, management of, 1431-1432
    pitfalls of, 1431-1432
    residual oronasal fistula, closure of, 1431
    studies of, 1432-1433
    timing of, 1373
    unresolved issues of, 1433-1436
    vertical lengthening, cleft maxilla, 1431
  presurgical assessments, 1428-1429
  residual deformities, 1373-1428
  treatment protocols, 1372-1373, 1374f-1427f
Bimaxillary dental protrusive growth patterns, 897-944
  clinical characteristics of, 898, 942
  dental findings for, 898
  functional aspects of, 898
  orthodontic approach
    historical perspectives of, 899
    studies of, 899-940
    techniques, 900f-939f
  prevalence of, 897
  radiologic findings for, 898-899
  surgical approaches, 940-941
    complications of, 941
    informed consent and, 941
    malocclusion after, 941-942
    patient education for, 941
    postsurgical considerations, 941
    temporomandibular disorders (TMDs) and, 942. *See also* Temporomandibular disorders (TMDs).
  treatment pitfalls of, 899
Binder syndrome, 1159-1181
  anthropometric explanations of, 1159
  approaches to
    comparisons, 1179-1180
    current, 1160-1177, 1160f-1176f
Binder syndrome *(Continued)*
  characteristics of, 1159-1160
  controversies of, 1177-1179
  historical perspectives of, 1159
  skeletal stability, post-reconstruction, 1177
    method comparisons, 1177
    patient comparisons, 1177
    result comparisons, 1177
  unresolved issues of, 1177-1179
Binding, prominent ears, 1739-1740. *See also* Prominent ears.
Biocoral alloplastic materials, 614
Bioelectric theory, 137-141
Biofax alloplastic materials, 614
Biologic envelopes, 136
Biologic mechanisms, grafts, 607-609. *See also* Grafts and grafting.
  bone morphogenic proteins (BMPs), 607
  comparisons of, 607
  osteoconduction, 607, 608f
  osteogenesis and, 607
  osteoinduction and, 607
Biologic width, 175-176
Biosynthetic corneas, 12
Biotype considerations, 156
Bisphosphonates, 142-143
Blair, Vilray P., 22-23, 26-32, 27f, 29f-30f
Blood banking, 313
Blood loss and fluid replacement, 308-336
Blood replacement and related considerations, 308-336
  convalescence and. *See* Convalescence.
  deliberate hypotensive anesthesia (DHA), 308-313. *See also* Anesthesia and related considerations.
    complications of, 310
    historical perspectives of, 308-309, 311-313
    inducement techniques for, 309
    mean arterial pressure (MAP) and, 309, 318
    monitoring during, 310
    organ function effects of, 309-310
    orthognathic surgery-specific effects of, 310-313
    patient positioning and, 309
  hemorrhage depressors and, 311
    blood loss limitation effects of, 311
    desmopressin, 311
    tranexamic acids, 311
  perioperative airway management and, 317-323
  replacement needs and, 313-317
    approaches to, 313
    complex surgeries, 315t-316t
    controversies in, 314-317
    historical perspectives of, 314
    studies of, 314
BMI. *See* Body mass index (BMI).
BMPs. *See* Bone morphogenic proteins (BMPs).
Body dysmorphic disorder (BDD), 219, 987
Body mass index (BMI), 995-996, 995f
Bonding *vs*. banding, 161-162
Bone-anchored hearing aid (BAHA) options, 1151-1152
Bone grafts and grafting
  alloplastic grafts, 636
  for ascending ramus defects, 623-626
    autogenous rib donor sites, 623-626
    costochondral donor sites, 623-626
    procedures for, 623-626, 624f-625f
  biologic mechanisms of, 607-609
    bone morphogenic proteins (BMPs), 607
    comparisons of, 607
    osteoconduction, 607, 608f
    osteogenesis and, 607
    osteoinduction and, 607
  cartilage grafting, nasal reconstruction, 626-636
    autogenous ear (conchal) cartilage donor sites, 629-630
    autogenous nose (septal) cartilage donor sites, 610f, 626-629
    procedures for, 626-629, 627f-629f
  for condyle defects, 623-626
    autogenous rib donor sites, 623-626
    costochondral donor sites, 623-626
    procedures for, 623-626, 624f-625f
  facial skeleton augmentation, 636
  goals of, 636
  for interpositional defects and gaps, mandible *vs*. maxilla, 609-623
    allogenic grafts, 622-623
    alloplastic grafts, 620-622
    autogenous iliac donor sites, 609-614
    corticocancellous bloc donor sites, 614-620, 615f
    human sterile freeze-dried iliac grafts, 622-623
    particulate cancellous donor sites, 609-614
    porous hydroxyapatite bloc implants, 620-622, 621f-622f
    procedures for, 609-613, 610f-613f, 616-620, 616f-620f
  options for, 607-609
  porous polyethylene augmentation implants, 636
  prevalence of, 607
  types of, 608-609
    allografts, 608
    autografts, 608
    graft substitutes, 608-609

Bone grafts and grafting *(Continued)*
U.S. Food and Drug Administration (USDA) approval of, 608-609, 622
Bone morphogenic proteins (BMPs), 607
Bone resorption, osteoclast-mediated, 142-143
Bony defects, residual, 1373-1428, 1443, 1444f-1445f
Brachial arch syndromes, 73-77, 76f-77f, 1095. *See also* Hemifacial microsomia (HFM).
Brackets
clear, 161
composite, 161
intra-oral auxiliaries, 162-163
materials for, 161-162
self-ligating, 142, 161-162
self-ligating, active, 161
Breakage, instrument, 486, 492f
Broadbent, Holly, 23-24, 23f-24f
Buccal tooth surfaces, 138f, 172
Bzoch Error Pattern Diagnostic Articulation Test, 237, 237b

C

Caldwell, J.B., 21-22
Camouflage approach
orthodontic, 202-203, 642-643, 688-689, 760
surgical, 689
Camouflage procedures, 614
cAMP levels. *See* Cyclic adenosine monophosphate (cAMP) levels.
Captures
centric relation (CR), 427. *See also* Centric relation (CR).
spatial relationships, 408-416
Carpenter syndrome, 1226
Cartilage grafting, 626-636. *See also* Grafts and grafting.
Casts
impressions, 380f-385f, 415
mandibular, 418
Cause-effect relationships, 252-253
CDP. *See* Chondrodysplasia punctata (CDP).
CEJ. *See* Cementoenamel junction (CEJ).
Cementoenamel junction (CEJ), 172, 173f
Cements and adhesives, 162
Cementum, 136-137, 136f, 138f, 173f, 174, 176-177
Cementum resorption, 180
Central pattern generators (CPGs), 227
Central sleep apnea, 1000. *See also* Obstructive sleep apnea (OSA).
Centric relation (CR), 424-427
capture of, 427
controversies of, 424-427
definition of, 424-427
unstable *vs*. unreliable, 427
Cephalometers, 23-24
Cephalometric analyses, 363
Cephalometric upper airway length (UAL), 999
Cephalostat devices, 23-24, 24f
Cervical fascia management, 1762-1763
Cervical spine range-of-motion (ROM), 360
Champy, Maxime, 50
Cheese wiring, 37
Cheever, David Williams, 30-32, 31f
Chin, 1564-1612
aesthetics of, 1564. *See also* Aesthetics.
characteristics, 1564-1571, 1565f, 1567f-1571f
errors in, 1601
favorable, achievement of, 1600-1601
alloplastic augmentation, 1604
anatomy of, 1564
complications and unfavorable results, 1602-1604
altered sensibility, 1602
infection, 1602
patient dissatisfaction, 1603-1604
prevalence of, 1602
teeth, injuries to, 1603, 1603f
wound-healing complications, 1602-1603
deformities, patterns of, 1571-1597
asymmetries, 1596-1597, 1596f
horizontal deficiency, with/without vertical discrepancy, 1565f-1571f, 1571-1572, 1573f-1593f
horizontal prominence, with vertical discrepancy, 1572-1595, 1573f-1576f, 1594f-1595f
dysplasia, 1373-1428, 1443, 1444f-1445f
historical perspectives of, 1564
maxillary-mandibular chin advancement (MMCA), 992-1000
osseous genioplasty
biologic foundations of, 1571
pitfall avoidance strategies for, 1594f-1599f, 1601-1602
techniques, 1597-1600, 1597f-1599f
patient education for, 1604-1605
Chondritis, 1739
Chondrodysplasia punctata (CDP), 1159-1181. *See also* Binder syndrome.
anthropometric explanations of, 1159
approaches to, 1160-1177, 1160f-1176f
characteristics of, 1159-1160
controversies of, 1177-1179
historical perspectives of, 1159
skeletal stability, post-reconstruction, 1177
unresolved issues of, 1177-1179
Chronic obstructive nasal breathing, 287-295
Cingulum, 173f, 174
Classic beauty, 337
Clear brackets, 161. *See also* Brackets.
Cleft-dental gaps, 1365, 1373-1428. *See also* Cleft-orthognathic surgeries.
Cleft-orthognathic surgeries
bilateral lip/palate (BCLP), 1371-1440
in adolescents, 1373-1428
coordinated team approach, 1372
facial growth implications of, 1371-1372
in infants, 1371-1372
jaw deformities and, 1428
orthodontic approaches, 898
orthognathic approach, 1374f-1427f
orthognathic approaches, 1429-1431
presurgical assessments, 1428-1429
residual deformities, 1373-1428
treatment protocols, 1372-1373, 1374f-1427f
formation influences and, 77-91, 79f-90f. *See also* Formation influences.
isolated cleft palate (ICP), 1441-1470
in adolescents, 1443
vs. cleft lip/palate, 1441
controversies of, 1467-1468
coordinated team approaches, 1442
facial growth implications of, 1441-1442
jaw deformities and, 1443
presurgical assessments, 1443-1465
residual deformities, 1443, 1444f-1445f
surgical approaches, 1444f-1465f, 1465-1466
treatment protocols, 1442
unresolved issues of, 1467-1468
unilateral cleft lip/palate (UCLP), 1297-1370. *See also* Unilateral cleft lip/palate (UCLP).
coordinated team approach, 1302-1304
facial growth implications, 1297-1302
historical perspectives of, 1297
in infants, 1297-1302
jaw deformities and, 1356-1357
orthodontic approaches, 1356-1357
presurgical assessments, 1357
treatment protocols, 1299f-1305f, 1304-1306, 1307f-1354f
Clinical crowns, 172, 173f
Clinical evaluations, 351-369. *See also* Planning techniques.
approaches to, 351
global head and neck assessments, 351-360
current range-of-motion (ROM), cervical spine, 360
Clinical evaluations *(Continued)*
current range-of-motion (ROM), mandibular, 360
current range-of-motion (ROM), neck, 360
dental rehabilitation needs, 352, 353f-359f
facial aesthetic units, unique/discrete, 352, 353f-359f
facial skeleton, lower, 360
facial skeleton, upper, 352, 353f-359f
overlying soft-tissue envelope, 351-352
seven critical aspects of, 351-360
temporomandibular disorders (TMDs), 360
maxillomandibular surgical planning, detailed, 360-368, 364f-365f
anterior nasal spine morphology, 367-368
A-point to B-point relationships, 367
chin morphology, baseline, 368
eight critical decisions, 353f-359f, 360-368
facial views, natural head position, 367
jaw reorientations, 368
maxillary cant (upper face reference), 361-362
maxillary dental midline (upper face reference), 361
maxillary incisor crown horizontal position (upper face reference), 363-367
maxillary incisor crown vertical position (upper face reference), 362-363
maxillary reorientation, 367-368
maxilla segmentation, 368
nose floor, baseline pyriform, 367-368
osseous genioplasty considerations, 368
pitch orientation, 367
pogonion locations, 368
rims, baseline pyriform, 367-368
roll orientation, 361-362
yaw orientation, 361
Clinical judgment considerations, 164
Clinical roots, 172, 173f
Co-destructive factors, 180
COG-ENT chronic rhinosinusitis study, 301
Combined visual/auditory distortion speech errors, 237-240
Compensation, dental
orthodontic-introduced, 61
skeletal discrepancy, 61, 62f
Complex craniosynostosis, 1183-1226, 1185f-1189f
Complex malformations, 1261-1296. *See also* Malformations.
comparisons of, 1261, 1288

Complex malformations *(Continued)*
Down (Trisomy 21) syndrome, 1265-1271
characteristics of, 1198f-1200f, 1265-1271, 1266f-1269f
historical perspectives of, 1265-1271
macroglossia in, 1270-1271
reconstruction in, 1271
hemihyperplasia, facial, 1276-1277
characteristics of, 1276-1277, 1278f-1279f
vs. hemimandibular hyperplasia (HMH), 1277
historical perspectives of, 1276
neoplasms, associated, 1276-1277
reconstruction in, 1277
Klippel-Feil anomaly (KFA), 1261-1265
characteristics of, 1261-1262
historical perspectives of, 1261
reconstruction in, 1262-1265, 1263f-1265f
neurofibromatosis, 1271-1274
acoustic type (type II), 1271
characteristics of, 776f-779f, 1271-1274
historical perspectives of, 1271
orbitotemporal, 1274
reconstruction in, 1274
von Recklinghausen type (type I), 1273-1274
prevalence of, 1261
vascular, 1277-1288
arteriovenous, 1280-1282, 1286f-1287f
capillary, 1288
characteristics of, 1277, 1280f-1287f
complications of, 1277
historical perspectives of, 1277
lymphatic, 1277-1279, 1282f-1283f
venous, 1279-1280
velocardiofacial syndrome (VCFS), 1288
characteristics of, 1288
pathophysiology of, 1288
prevalence of, 1288
reconstruction in, 1288
Complex surgeries, 315t-316t
Complication-related considerations, 475-542
for chin, oblique osteotomies, 525-527
infection, 525
injuries, teeth, 526
nerve injuries, mental, 525
suboptimal facial aesthetics, 526
wound healing, 526
fixation factors, 479-481, 481f
plate removal needs, 479
screw removal needs, 479
Complication-related considerations *(Continued)*
titanium *vs*. resorbable, 479-481
general factors, 475-486
associated infections, 481-486, 482f-492f
comprehensive dental rehabilitation need, 477
foreign-body reactions, 486, 492f
functional assessments, head and neck structures, 478
instrument breakage, 486, 492f
operating room team preparation, 478
temporomandibular disorders (TMDs), 478
third molar management, 478-479, 480f
patient education materials for, 527
risk factors, 475-486
cervical spine, 477
comprehensive dental rehabilitation need, 477
historical perspectives of, 475
medical, 475-476
perioperative airway, 476-477, 476f
prevalence of, 475
psychological, 477
would-healing, 476
for sagittal split ramus osteotomies (SSROs), mandibular, 486-504
bleeding, significant, 503-504
nerve injuries, facial, 503
nerve injuries, inferior alveolar, 500-502, 500f-501f
nerve injuries, lingual, 502-503, 503f
nerve injuries, mental, 500-502, 500f-501f
unfavorable, 486-500, 493f-500f
Composite brackets, 161. *See also* Brackets.
Comprehensive dental rehabilitation, 477, 982
Conchal hypertrophy, 1714
Conchal-mastoid suture placement, 1712f, 1713
Conchal wall resection, 1241f, 1706, 1711f, 1712
Condylar hyperactivity
active unilateral, 860-861
type I, 854-855
type II, 855-860
Condylar hyperplasia, 807. *See also* Asymmetric mandibular excess.
Condylar resorption, idiopathic (ICR), 1530-1563
case studies of, 1534
idiopathic condylar resorption (ICR), 1538b, 1539f-1547f, 1543b-1548b, 1554b, 1555f-1560f
Condylar resorption, idiopathic (ICR) *(Continued)*
progressive condylar resorption (PCR), 1534b-1538b, 1534f-1537f
progressive condylar resorption/idiopathic condylar resorption (PCR/ICR), 1548b, 1549f-1553f
clinical perspectives of, 1531-1532
etiology theories of, 1530-1531, 1560
avascular necrosis theory, 1531
dominant theory, 1530-1531
favorable results of, 1560
jaw deformity correction, clinical approaches to, 1533
morphologic findings of, 1530
prevalence of, 1530
Condylectomy, partial, 860-861
Congenital myopathy effects, 115-118, 116f-117f, 119f-120f
Consent, informed. *See* Informed consent.
Conservative surgery, 22
Consonant sounds, 236
Constriction, maxillary arch, 640-685
Contact primary bone healing, 48-50
Continuous positive airway pressure (CPAP), 1009
Continuous tooth movement, 142. *See also* Tooth.
Contour irregularities, 1766
Convalescence, 308-336
anesthesia considerations and, 308-336. *See also* Anesthesia and related considerations.
at home/outpatient, 325
outpatient regimens, 325
physical therapy, 328b-330b
in-hospital, 323-325
airway needs, 323
antibiotics, 325
diet and nutrition, 324
discharge criteria, 325
intravenous (IV) fluid therapy, 324
monitoring equipment, 323-324
nasal medications, 325
nausea management, 324
pain management, 325
settings for, 323
vomiting management, 324
wound care, 324
Coordinated team approach, 1372
Coordinated team approaches, 1442
Corneal protection, 1709
Corneas, biosynthetic, 12
Cortical cuts, 442-446, 444f-445f
Cortical wall roles, 143-144
Corticocancellous bloc donor sites, 614-620, 615f. *See also* Donor sites.
Corticosteroid hormones, 142-143
Corticotomies
corticotomy-facilitated orthodontics, 164-165
maxillary, 44
Costochondral donor sites, 623-626. *See also* Donor sites.
Costochondral graft reconstruction, 1122-1150, 1123f-1149f. *See also* Grafts and grafting.
CPAP. *See* Continuous positive airway pressure (CPAP).
CPGs. *See* Central pattern generators (CPGs).
CR. *See* Centric relation (CR).
Cranial nerve dysfunction, 505
Cranial vault aesthetic unit, 1230
Craniofacial evolution, 4-5
Craniofacial microsomia, 1095. *See also* Hemifacial microsomia (HFM).
Craniofacial morphologic changes, 985-986
Craniosynostosis syndromes, 1182-1260
Apert syndrome, 1184-1200, 1190f-1193f, 1707f-1709f
approaches to, 1230-1246
in children, 1232-1237
comparisons of, 1254
historical perspectives of, 1230-1231
preferred, 1254
surgical timing philosophy, 1231-1232
upper midface deformities, 1232
in young adults, 1232-1237
associated syndromes, 1183
Carpenter syndrome, 1226
comparisons of, 1182-1183, 1185f-1189f
complex craniosynostosis, 1183-1226, 1185f-1189f
Crouzon syndrome, 1201, 1202f-1207f, 1213f-1220f, 1711f
definitions of, 1182
etiology of, 1182-1183
morphologic considerations of, 1229-1230
fronto-forehead aesthetic unit, 1230
maxillary/nasal base aesthetic unit, 1230
orbito-naso-zygomatic aesthetic unit, 1230
posterior cranial vault aesthetic unit, 1230
mutations in, 1183
neurologic aspects of, 1226-1228
Pfeiffer syndrome, 1201, 1221f-1227f
reconstruction staging
for Apert syndrome, 1251-1254
for Crouzon syndrome, 1247f-1249f, 1249-1251
Saethre-Chotzen syndrome, 1201
upper airway effects of, 1228-1229

Creutzfeldt-Jakob disease, 313
Crevicular epithelium, 174-175, 174f-175f
Crevicular gingiva, 175-176
Crossbite, unilateral maxillary, 271-272
Crouzon syndrome, 1201, 1202f-1207f, 1213f-1220f, 1247f-1249f, 1249-1251, 1711f
  obstructive sleep apnea (OSA) and, 996. *See also* Obstructive sleep apnea (OSA).
  reconstruction staging for, 1247f-1249f, 1249-1251
Crowns, anatomic, 172, 173f
Curing lights, halogen, 162
Cyclic adenosine monophosphate (cAMP) levels, 141-142

D
D (vitamin), 75
D_3 (vitamin), 142-143
Dali, Salvador, 349-350, 350f
DALL. *See* Dental anterior limit line (DALL).
da Vinci, Leonardo, 18-20, 342, 344f, 345-346, 347f-348f
De Architectura (Marcus Vitruvius Polio), 342, 342f
Decision-making processes, patient *vs*. family, 219
Deepening, antihelical folds, 1709f-1710f, 1712-1713
Defatting, neck, 1762-1763
Deficiency patterns. *See also* Patterns and presentations.
  mandibular, primary, 640-685
  maxillary, 686-757
  maxillomandibular, 866-896
Definition and prevalence, 61-68
  dental compensation
    orthodontic-introduced, 61
    skeletal discrepancy, 61, 62f
  dentofacial deformity definitions, 61, 66
  diagnoses, accurate, 63-64
  interdisciplinary approaches, 61
  jaw deformity and malocclusion prevalence, 64-65
    horizontal dimension, 64
    U.K. Index of Treatment Need, 65
    U.S. National Health and Nutrition Examination Survey III (NHANES III), 64
    vertical dimension, 64
  orthognathic procedures, 66
Deflections, septal, 287-288
Deformity-related surgical principles and practice
  cleft-orthognathic surgeries
    bilateral lip/palate (BCLP), 1371-1440
    isolated cleft palate (ICP), 1441-1470
    unilateral lip/palate (UCLP), 1297-1370
  fundamentals
    blood loss and fluid replacement, 308-336
    convalescence, 308-336
    definition and prevalence, 61-68
Deformity-related surgical principles and practice *(Continued)*
    facial perspectives, 1-17
    formation influences, 69-134
    historical perspectives, 18-60
    mastication considerations, 227-263
    nasal airway considerations, 287-307
    orthodontic considerations, 135-170
    periodontal considerations, 171-208
    psychosocial considerations, 209-226
    speech-related considerations, 227-263
    swallowing considerations, 227-263
    techniques, airway management, 308-336
    techniques, anesthesia, 308-336
    temporomandibular disorders (TMDs), 264-286
  growth patterns and presentations
    constriction, maxillary arch, 640-685
    deficiency, mandibular (primary), 640-685
    deficiency, maxillary, 686-757
    deficiency, maxillomandibular, 866-896
    deformities, mandibular, 758-806
    dentofacial disharmony, adult, 945-991
    dentofacial disharmony, teenage/young adult, 897-944
    excess, mandibular (asymmetric), 807-865
    excess, mandibular (relative), 686-757
    excess, maxillary (vertical), 758-806
    face, long, 758-806
    face, short, 866-896
    obstructive sleep apnea (OSA), 992-1058
    protrusive, bimaxillary dental, 897-944
  malformations
    Binder syndrome, 1159-1181
    complex, 1261-1296
    craniosynostosis syndromes, 1182-1260
    hemifacial microsomia (HFM), 1095-1158
    Treacher Collins syndrome (TCS), 1059-1094
  posttraumatic deformities
    aesthetic alterations, chin, 1564-1612
    aesthetic alterations, prominent ears, 1703-1745
    aesthetic alterations, soft tissues (lower face), 1746-1783
    aesthetic alterations, soft tissues (neck), 1746-1783
Deformity-related surgical principles and practice *(Continued)*
    idiopathic condylar resorption (ICR), 1530-1563
    jaw, secondary, 1471-1529
  treatment planning techniques
    analytic model-planning, mandible-first, 424-440
    analytic model-planning, standard, 374-423
    complication-related considerations, 475-542
    fundamentals, 337-373
    grafts and grafting, 607-639
    malocclusion prevention, 543-606
    procedure sequencing, 441-474
Degeneration factors, 171
Degenerative skin changes, 1749
Deliberate hypotensive anesthesia (DHA). *See also* Anesthesia and related considerations.
  complications of, 310
  historical perspectives of, 308-309, 311-313
  inducement techniques for, 309
  mean arterial pressure (MAP) and, 309, 318
  monitoring during, 310
  organ function effects of, 309-310
  orthognathic surgery-specific effects of, 310-313
  patient positioning and, 309
della Francesca, Piero, 345-346, 346f
Demineralization prevention, 162
Dental anchorage, 143
Dental anterior limit line (DALL), 363-367, 364f-365f
Dental-cleft gaps, 1365, 1373-1428. *See also* Cleft-orthognathic surgeries.
Dental compensation
  orthodontic-introduced, 61
  skeletal discrepancy, 61, 62f
Dental Cosmos (Whipple), 28
Dental-only approach, 1767-1768
Dental protrusive patterns, bimaxillary, 897-944
  characteristics of, 942
  clinical characteristics of, 898, 942
  dental findings for, 898
  functional aspects of, 898
  orthodontic approach
    historical perspectives of, 899
    studies of, 899-940
    techniques, 900f-939f
  prevalence of, 897
  radiologic findings for, 898-899
  surgical approaches, 940-941
    complications of, 941
    informed consent and, 941
    malocclusion after, 941-942
    patient education for, 941
    postsurgical considerations, 941
    temporomandibular disorders (TMDs) and, 942. *See also* Temporomandibular disorders (TMDs).
Dentin, 173f, 174
Dentofacial deformity-related surgical principles and practice
  cleft-orthognathic surgeries
    bilateral lip/palate (BCLP), 1371-1440
    isolated cleft palate (ICP), 1441-1470
    unilateral lip/palate (UCLP), 1297-1370
  fundamentals
    blood loss and fluid replacement, 308-336
    convalescence, 308-336
    definition and prevalence, 61-68
    facial perspectives, 1-17
    formation influences, 69-134
    historical perspectives, 18-60
    mastication considerations, 227-263
    nasal airway considerations, 287-307
    orthodontic considerations, 135-170
    periodontal considerations, 171-208
    psychosocial considerations, 209-226
    speech-related considerations, 227-263
    swallowing considerations, 227-263
    techniques, airway management, 308-336
    techniques, anesthesia, 308-336
    temporomandibular disorders (TMDs), 264-286
  growth patterns and presentations
    constriction, maxillary arch, 640-685
    deficiency, mandibular (primary), 640-685
    deficiency, maxillary, 686-757
    deficiency, maxillomandibular, 866-896
    deformities, mandibular, 758-806
    dentofacial disharmony, adult, 945-991
    dentofacial disharmony, teenage/young adult, 897-944
    excess, mandibular (asymmetric), 807-865
    excess, mandibular (relative), 686-757
    excess, maxillary (vertical), 758-806
    face, long, 758-806
    face, short, 866-896
    obstructive sleep apnea (OSA), 992-1058
    protrusive patterns, bimaxillary dental, 897-944
  malformations
    Binder syndrome, 1159-1181
    complex, 1261-1296
    craniosynostosis syndromes, 1182-1260
    hemifacial microsomia (HFM), 1095-1158

Dentofacial deformity-related surgical principles and practice *(Continued)*
Treacher Collins syndrome (TCS), 1059-1094
posttraumatic deformities
aesthetic alterations, chin, 1564-1612
aesthetic alterations, prominent ears, 1703-1745
aesthetic alterations, soft tissues (lower face), 1746-1783
aesthetic alterations, soft tissues (neck), 1746-1783
idiopathic condylar resorption (ICR), 1530-1563
jaw, secondary, 1471-1529
treatment planning techniques
analytic model-planning, mandible-first, 424-440
analytic model-planning, standard, 374-423
complication-related considerations, 475-542
fundamentals, 337-373
grafts and grafting, 607-639
malocclusion prevention, 543-606
procedure sequencing, 441-474
Dentofacial disharmonies
adult, 945-991. *See also* Adult dentofacial disharmony.
comprehensive dental rehabilitation and, 982
facial aging and rejuvenation, 983-986
functional assessments for, 945-981
previous suboptimal facial surgery and, 983
recovery considerations, 987-988
special medical considerations for, 945
techniques for, 945, 946f-981f
temporomandibular disorders (TMDs) and, 981-982
treatment considerations for, 982-983
wound healing risk factors for, 982
teenage/young adult, 897-944
bimaxillary dental protrusive growth patterns, 897-944
characteristics of, 942
clinical characteristics of, 898
dental findings for, 898
functional aspects of, 898
orthodontic approach, 898
prevalence of, 897
radiologic findings for, 898-899
surgical approaches, 940-941
treatment pitfalls of, 899
Depressors, hemorrhage, 311. *See also* Blood replacement and related considerations.
blood loss limitation effects of, 311
desmopressin, 311
tranexamic acids, 311
De Re Aedificatoria (Bracciolini), 342
Desmopressin, 311
Detailed maxillomandibular surgical planning, 360-368, 364f-365f. *See also* Planning techniques.
anterior nasal spine morphology, 367-368
A-point to B-point relationships, 367
chin morphology, baseline, 368
eight critical decisions, 353f-359f, 360-368
facial views, natural head position, 367
jaw reorientations, 368
maxillary cant (upper face reference), 361-362
maxillary dental midline (upper face reference), 361
maxillary incisor crown horizontal position (upper face reference), 363-367
maxillary reorientation, 367-368
maxilla segmentation, 368
nose floor, baseline pyriform, 367-368
osseous genioplasty considerations, 368
pitch orientation, 367
pogonion locations, 368
rims, baseline pyriform, 367-368
roll orientation, 361-362
yaw orientation, 361
Developmental-hereditary-environmental influences, 69-134
comparisons of, 69
head and neck development, 69-72
face, 72, 72f-73f
palate, 72, 74f-75f
skull, 69-72, 70f-71f
hereditary developmental tendencies, 102-105
comparisons of, 102-105, 105f-108f, 157f
prevalence of, 102
skeletal growth, environmental and neuromotor effects on, 105-118, 109f-112f
Apert syndrome, 115. *See also* Apert syndrome.
comparisons of, 105, 109f-112f
congenital myopathy effects, 115-118, 116f-117f, 119f-120f
Crouzon syndrome, 115
masseteric muscle hypertrophy, 113
muscle effects, 105-115, 108f-111f
respiration pattern effects, 115, 116f-117f
Developmental-hereditary-environmental influences *(Continued)*
soft tissue effects, 105-115, 108f
syndromes and anomalies, 73-102. *See also* Syndromes.
achondroplasia, 91
acromegaly, 102
brachial arch syndromes, 73-77, 76f-77f
cleft lip/palate, 77-91, 79f-90f. *See also* Cleft-orthognathic surgeries.
comparisons of, 73
Goldenhar syndrome, 73-77, 76f-77f
hemifacial hypertrophy, 102
midline tissue structure deficiencies, 73
neural crest origin deficiencies and anomalies, 73-77, 76f-77f
overgrowth syndromes and anomalies, 102, 103f
premature suture closure, cranial vault and skull base, 70f-71f, 91-92, 92f-102f
Robin sequence, 90f, 91
Stickler syndrome, 85f-89f, 91
synostosis, 70f-71f, 91-92
teratogens and, 75
Treacher Collins syndrome (TCS), 73-77, 76f-77f
van der Wound syndrome, 77-91, 84f
trauma effects, 118-124, 121f-123f
tumor and growth effects, 124, 125f-131f
Developmental tendencies, 102-105
Deviated mandibular prognathism, 807. *See also* Asymmetric mandibular excess.
DHA. *See* Deliberate hypotensive anesthesia (DHA).
Diadumenos (Polykleitos), 338-339, 338f-339f
Diet and nutrition considerations, 324
Differentiation, morphologic, 985-986
Difficult airway guidelines, 319. *See also* Airways.
Digastric muscle ridging, 1766
Dilantin, 75
Dingman, Reed O., 28
Direct visual examinations, 408
Discharge criteria, 325
Disharmonies, dentofacial
adult, 945-991
teenage/young adult, 897-944
Displacement resistance, 143
Distal tooth surfaces, 138f, 172
Distortion speech errors, 237-240
Distraction osteogenesis (DO), 1373, 1436
Dog ear, 1708
Dominant theory, 1530-1531
Donor-directed blood banking, 313
Donor sites. *See also* Grafts and grafting.
autogenous iliac, 609-614
autogenous rib, 623-626
corticocancellous bloc, 614-620, 615f
costochondral, 623-626
Doryphoros (Polykleitos), 338-339, 338f-339f
Down-fracture osteotomies, 287-288, 293f-295f, 296-297
Down (Trisomy 21) syndrome, 1265-1271. *See also* Complex malformations.
characteristics of, 1198f-1200f, 1265-1271, 1266f-1269f
historical perspectives of, 1265-1271
macroglossia in, 1270-1271
obstructive sleep apnea (OSA) and, 996. *See also* Obstructive sleep apnea (OSA).
reconstruction in, 1271
Dysesthesia, 987-988
Dysmorphic faces, 12, 13f-14f
Dysmorphology, 1097-1099
Dysostosis, unilateral oto-mandibular, 1095. *See also* Hemifacial microsomia (HFM).
Dysplasia
chin, 1373-1428, 1443, 1444f-1445f. *See also* Chin.
lateral facial, 1095
oculo-auriculo-vertebral, 1095. *See also* Hemifacial microsomia (HFM).
Dystonia, mandibulofacial, 1059-1094. *See also* Treacher Collins syndrome (TCS).
approach comparisons, 1091
characteristics of, 1059-1061, 1060f
dysmorphology with, 1062-1063
facial growth potential with, 1063-1069, 1064f-1069f
in infancy and early childhood, 1061-1062
inheritance and genetic markers, 1061, 1061f
skeletal reconstruction staging, 1072-1087, 1073f-1079f
temporomandibular joint (TMJ)-mandibular malformation classification, 1069-1072, 1070f-1071f
testing for, 1061

E

Earlobes
excessive, 1730. *See also* Ears, prominent.
set-back, 1713
Early loosening, 166
Early malocclusion, 543-555
Ears, prominent, 1703-1745
aesthetic objectives for, 1706
ear axis, 1706

Ears, prominent *(Continued)*
ear position, 1706
ear protrusion, 1706
ear size, 1706
anatomy of, 1704f, 1705
binding of, 1739-1740
complications and suboptimal results, 1714-1739
abscesses, stitch, 1739
antihelical folds, too-sharp, 1714
chondritis, 1739
conchal hypertrophy, inadequate management of, 1714
ear asymmetry, residual, 1731-1739
external auditory canal, pinching of, 1714
flat ear appearance, postsurgical, 1739
infection, cartilage, 1739
necrosis with skin loss, 1739
prevalence of, 1714
prominent earlobe, excessive, 1730
scar formation, abnormal, 1739
suture granulomas, 1739
telephone ear appearance, postsurgical, 1739
upper helix, failure *vs*. relapse, 1730-1731
embryology of, 1705
historical perspectives of, 1704-1705
non-surgical correction, newborns, 1739-1740
vs. normal ears, 1703, 1704f, 1740
otoplasty procedures for, 1708-1713
antihelical fold, creation *vs*. deepening, 1709f-1710f, 1712-1713
antihelical fold, marking of, 1712
conchal-mastoid suture placement, 1712f, 1713
conchal wall resection, 1241f, 1706, 1711f, 1712
corneal protection, 1709
draping, 1709
dressing placement, 1713, 1714f
general anesthesia intubation, 1709
local anesthesia, 1709
medications, 1709
postauricular flaps, elevation of, 1708f, 1710-1712
postauricular skin excision, 1707f, 1710
postoperative care, 1713
preparation, 1709
results, assessments, 1713-1714, 1715f-1716f, 1718f-1726f, 1728f-1738f
set-back, earlobe, 1713
set-back, lower third of ear, 1713
set-back, middle third of ear, 1712f, 1713
set-back, resection, 1241f, 1711f, 1712

Ears, prominent *(Continued)*
set-back, upper third of ear, 1713
skin closure, 1713
superior helix-mastoid suture placement, 1713
surgical markings, 1709-1710
patient education materials for, 1740
prevalence of, 1183, 1703-1704
surgical goals for, 1704
surgical principles for, 1706-1707
goals, 1704
techniques, 1707-1708, 1707f-1712f
timing, 1705-1706
Eating disorders, baseline, 218
Edgewire, 21-22
Edgewise appliances, 156-161. *See also* Fixed appliances.
Education, patient. *See* Patient education.
Eicosanoids, 142-143
Elements (Euclid), 341-342, 341f-342f
Elongation, hemimandibular, 855-860
Enamel, 172-174, 173f
Envelopes
biologic, 136
overlying soft-tissue, 351-352
Environmental-developmental-hereditary influences, 69-134
comparisons of, 69
head and neck development, 69-72
face, 72, 72f-73f
palate, 72, 74f-75f
skull, 69-72, 70f-71f
hereditary developmental tendencies, 102-105
comparisons of, 102-105, 105f-108f, 157f
prevalence of, 102
skeletal growth, environmental and neuromotor effects on, 105-118, 109f-112f
Apert syndrome, 115
comparisons of, 105, 109f-112f
congenital myopathy effects, 115-118, 116f-117f, 119f-120f
Crouzon syndrome, 115
masseteric muscle hypertrophy, 113
muscle effects, 105-115, 108f-111f
respiration pattern effects, 115, 116f-117f
soft tissue effects, 105-115, 108f
syndromes and anomalies, 73-102
achondroplasia, 91
acromegaly, 102
brachial arch syndromes, 73-77, 76f-77f
cleft lip/palate, 77-91, 79f-90f. *See also* Cleft-orthognathic surgeries.
comparisons of, 73
Goldenhar syndrome, 73-77, 76f-77f

Environmental-developmental-hereditary influences *(Continued)*
hemifacial hypotrophy, 102
midline tissue structure deficiencies, 73
neural crest origin deficiencies and anomalies, 73-77, 76f-77f
overgrowth syndromes and anomalies, 102, 103f
premature suture closure, cranial vault and skull base, 70f-71f, 91-92, 92f-102f
Robin sequence, 90f, 91
Stickler syndrome, 85f-89f, 91
synostosis, 70f-71f, 91-92
teratogens and, 75
Treacher Collins syndrome (TCS), 73-77, 76f-77f
van der Wound syndrome, 77-91, 84f
trauma effects, 118-124, 121f-123f
tumor and growth effects, 124, 125f-131f
Epidemiological considerations. *See* Prevalence and definitions.
Epiglottis, 995, 995f
Epithelium, oral, 174-175, 174f-175f
Epworth Sleepiness Scale (ESS), 998
Equilibrium principle, 338-339
Erickson Model Table, 389f-393f, 406-407. *See also* Analytic model-planning.
reorientation, maxillary casts, 417
site measurements, maxilla reorientation, 406
spatial orientation
articulator-mounted mandibular unit, 416
articulator-mounted maxillary unit, 416
Erupted wisdom teeth, removal of, 455, 455f
Erythropoietin, recombinant human, 313
ESS. *See* Epworth Sleepiness Scale (ESS).
Estrogens, 142-143
Etiology. *See* Prevalence and definitions.
Etruscans, 18-20, 19f
Euclid, 341-342, 341f
Euclid of Alexandria, 337
Evaluation-related considerations
clinical evaluations, 351-369. *See also* Planning techniques.
approaches to, 351
global head and neck assessments, 351-360. *See also* Global head and neck assessments.
mastication factors, 227-263
nasal airway factors, 287-307. *See also* Airways.
orthodontic factors, 135-170

Evaluation-related considerations *(Continued)*
psychosocial factors, 209-226
seven critical aspects, 351-360
speech-related factors, 227-263
swallowing factors, 227-263
Evolutionary thinking, facial aesthetics, 338-350. *See also* Ideal facial aesthetics.
Evolutionary transformations, 1, 4-6
craniofacial evolution, 4-5
definition of, 1
hominid species, 6, 8f-9f
mammalian brain, 4, 5f
molecular components, 4-5
primate brain, 4-5
skull comparisons, 5-6, 6f-8f
Excess patterns
mandibular, asymmetric, 807-865
mandibular, relative, 686-757
maxillary, vertical, 758-806
Expansion, maxillary arch, 290f, 297-301
External auditory canal reconstruction, 1151-1152
External ear reconstruction, 1151
Extraocular cranial nerve dysfunction, 505
Extreme and mean ratio (Euclid), 337
Extubation
benchmark airway parameters checklist for, 323
criteria for, 321-322
vs. intubation. *See* Intubation.
planning for, 322
strategies for, 321

F

Face
aesthetics, ideal, 337-351. *See also* Ideal facial aesthetics.
aging and rejuvenation, 983-986
craniofacial morphologic changes, 985-986
mandibular skeletal changes, 986
midface aging, 984-985
morphologic differentiation, 985-986
periorbital aging, 984-985
special considerations for, 983-984
dysmorphic faces, 12, 13f-14f
facial hemihyperplasia, 1276-1277. *See also* Facial hemihyperplasia.
facial perspectives, 1-17. *See also* Facial perspectives.
facial tooth surfaces, 138f, 172
harmony, 1747
lower, soft tissues, 1746-1783. *See also* Lower face and neck soft tissues.
complications and unfavorable results, 1766
envelope, aging of, 70f-71f, 1746-1749
midface rejuvenation, approaches to, 1749-1767

Face *(Continued)*
midface rejuvenation, principles of, 1747-1749
patient education materials for, 1773
treatment pitfalls, 1766-1767
patterns
long, 758-806
short, 866-896
skeleton augmentation, 636
standard facial records, 408
Face-bow registration and transfer, 375f-385f, 408-416, 409f-415f
Face-lift approach, traditional, 1749-1758, 1755f-1761f
Face-mask ventilation, adequate, 319
Facial hemihyperplasia, 1276-1277. *See also* Complex malformations.
characteristics of, 1276-1277, 1278f-1279f
vs. hemimandibular hyperplasia (HMH), 1277
historical perspectives of, 1276
neoplasms, associated, 1276-1277
reconstruction in, 1277
Facial perspectives, 1-17. *See also* Face.
aesthetics, ideal, 337-351
artistic perspectives, 16, 16f
craniofacial articulations, 15t, 16
craniofacial growth and development, 6-11
bone modification, 6, 8t
facial alterations, 6-10, 10f-11f
facial asymmetry, 10, 11f
skull as community of bones *vs*. joints, 10-11, 11f, 12t
skull size comparisons, 6, 9f
definitions, 1, 3b
behavioral, 1, 2f-3f
evolutionary transformations, 1
structural, 1, 1f-2f
symbolic, 1, 3f
dysmorphic faces, 12, 13f-14f
evolutionary transformations, 1, 4-6
craniofacial evolution, 4-5
hominid species, 6, 8f-9f
mammalian brain, 4, 5f
molecular components, 4-5
primate brain, 4-5
skull comparisons, 5-6, 6f-8f
facial surgery limitations, 12, 14f
life cycle stages, 1-3, 3b-4b, 4f-5f
prosopagnosia, 12-15, 15f
psychiatric genetics, 15-16, 15t
vision restoration, 12, 15f
Facial Proportions of Helen Wills (Ghyka), 349-350, 349f
Facial tooth surfaces, 138f, 172
Facio-auriculo-vertebral sequence, 1095. *See also* Hemifacial microsomia (HFM).
FALL. *See* Forehead anterior limit line (FALL).
FA point. *See* Forehead anterior (FA) point.
Farrar, J.N., 18-20, 20f
Fat atrophy *vs*. hypertrophy, 1749
Fauchard, Pierre, 18-20, 19f
Fechner, Gustav, 348-349, 349f
Fibonacci, Leonardo, 343
Fibonacci numbers, 343, 344f-345f
Fibrocytes, 136-137, 136f, 138f
First and second brachial arch syndrome, 1095. *See also* Hemifacial microsomia (HFM).
Fisher-Logermann Test of Articulation, 237, 237b
Five Platonic solids, 341-342, 341f
Fixation factors, 479-481, 481f
plate removal needs, 479
screw removal needs, 479
titanium *vs*. resorbable, 479-481
Fixed appliances, 156-163. *See also* Appliances; Orthodontic considerations.
arch wires, 162
auxiliary attachments, 161
banding *vs*. bonding, 161-162
bracket materials of, 161-162
cements and adhesives, 162
clear brackets, 161
design of, 161-162
edgewise appliances, 156-161
historical perspectives of, 156-161
intra-oral bracket auxiliaries, 162-163
prescriptions for, 161
rotational control of, 161
Flat ear appearance, postsurgical, 1739
Fluid replacement and blood loss, 308-336
blood replacement needs and, 313-317
approaches to, 313
complex surgeries, 315t-316t
controversies in, 314-317
historical perspectives of, 314
studies of, 314
convalescence and. *See* Convalescence.
deliberate hypotensive anesthesia (DHA) and, 308-313. *See also* Deliberate hypotensive anesthesia (DHA).
hemorrhage depressors and, 311
blood loss limitation effects of, 311
desmopressin, 311
tranexamic acids, 311
perioperative airway management and, 317-323. *See also* Perioperative airway management.
Fluoride-releasing bonding cements, 162
Force duration, 142
Forehead anterior limit line (FALL), 363-367, 364f-365f
Forehead anterior (FA) point, 363-367, 364f-365f
Foreign-body reactions, 486, 492f
Formation influences, 69-134
comparisons of, 69
head and neck development, 69-72
face, 72, 72f-73f
palate, 72, 74f-75f
skull, 69-72, 70f-71f
hereditary developmental tendencies, 102-105
comparisons of, 102-105, 105f-108f, 157f
prevalence of, 102
skeletal growth, environmental and neuromotor effects on, 105-118, 109f-112f
Apert syndrome, 115
comparisons of, 105, 109f-112f
congenital myopathy effects, 115-118, 116f-117f, 119f-120f
Crouzon syndrome, 115
masseteric muscle hypertrophy, 113
muscle effects, 105-115, 108f-111f
respiration pattern effects, 115, 116f-117f
soft tissue effects, 105-115, 108f
syndromes and anomalies, 73-102
achondroplasia, 91
acromegaly, 102
brachial arch syndromes, 73-77, 76f-77f
cleft lip/palate, 77-91, 79f-90f. *See also* Cleft-orthognathic surgeries.
comparisons of, 73
Goldenhar syndrome, 73-77, 76f-77f
hemifacial hypotrophy, 102
midline tissue structure deficiencies, 73
neural crest origin deficiencies and anomalies, 73-77, 76f-77f
overgrowth syndromes and anomalies, 102, 103f
premature suture closure, cranial vault and skull base, 70f-71f, 91-92, 92f-102f
Robin sequence, 90f, 91
Stickler syndrome, 85f-89f, 91
synostosis, 70f-71f, 91-92
teratogens and, 75
Treacher Collins syndrome (TCS), 73-77, 76f-77f
van der Wound syndrome, 77-91, 84f
trauma effects, 118-124, 121f-123f
tumor and growth effects, 124, 125f-131f
Form follows function concept, 337
Free gingiva, 174-175, 174f-175f
Freeze-dried iliac grafts, 622-623. *See also* Grafts and grafting.
Fricative airstream obstruction, 236
Friction forces, 161
Frontal undermining resorption, 180
Fronto-forehead aesthetic unit, 1230
Fuller, Richard Buckminster, 337
Functional appliances, 163-164. *See also* Appliances.
Functional assessments
for adult dentofacial disharmony, 945-981
Fundamentals
blood loss and fluid replacement, 308-336
convalescence, 308-336
definition and prevalence, 61-68
facial perspectives, 1-17
formation influences, 69-134
historical perspectives, 18-60
mastication considerations, 227-263
nasal airway considerations, 287-307
orthodontic considerations, 135-170
periodontal considerations, 171-208
planning techniques, 337-373
psychosocial considerations, 209-226
speech-related considerations, 227-263
swallowing considerations, 227-263
techniques
airway management, 308-336
anesthesia, 308-336
temporomandibular disorders (TMDs), 264-286
Future innovations, 56

G

Galen, 339
GALL. *See* Goal anterior limit line (GALL).
Gaps, cleft-dental, 1373-1428
Genioplasty, osseous
biologic foundations of, 1571
pitfall avoidance strategies for, 1594f-1599f, 1601-1602
techniques, 1597-1600, 1597f-1599f
Geometry of Art and Life (Ghyka), 349-350, 349f
Ghyka, Matila, 349-350, 349f
Gilles, Harold, 33-34, 33f, 39, 55, 55f
Ginestet, Gustave, 53-55
Gingiva, 174-178, 175f
Gingival. *See also* Gingiva.
biotype, 200
fibers, 175-176, 175f
interface, 181
sulcus, 176
Gingivitis, 178-179, 178f-179f
Global head and neck assessments, 351-360
current range-of-motion (ROM)
cervical spine, 360
mandibular, 360
neck, 360

Global head and neck assessments *(Continued)*
dental rehabilitation needs, 352, 353f-359f
facial aesthetic units, unique/discrete, 352, 353f-359f
facial skeleton
lower, 360
upper, 352, 353f-359f
overlying soft-tissue envelope, 351-352
seven critical aspects of, 351-360
temporomandibular disorders, 360
Goal anterior limit line (GALL), 363-367, 364f-365f
Gold arch wires, 162
Goldenblat syndrome, 1095. *See also* Hemifacial microsomia (HFM).
Goldenhar syndrome, 73-77, 76f-77f
Golden ratio, 337, 342
Goldman-Fristoe Test of Articulation, 237, 237b
Gor-Tex alloplastic materials, 614
Grafts and grafting, 607-639
alloplastic grafts, 636
for ascending ramus defects, 623-626
autogenous rib donor sites, 623-626
costochondral donor sites, 623-626
procedures for, 623-626, 624f-625f
biologic mechanisms of, 607-609
bone morphogenic proteins (BMPs), 607
comparisons of, 607
osteoconduction, 607, 608f
osteogenesis and, 607
osteoinduction and, 607
cartilage grafting, nasal reconstruction, 626-636
autogenous ear (conchal) cartilage donor sites, 629-630
autogenous nose (septal) cartilage donor sites, 610f, 626-629
autogenous rib (cartilage only) donor sites, 630-636, 631f, 633f-635f
procedures for, 626-630, 627f-629f, 631f, 632-636, 633f-635f
for condyle defects, 623-626
autogenous rib donor sites, 623-626
costochondral donor sites, 623-626
procedures for, 623-626, 624f-625f
costochondral graft reconstruction, 1122-1150, 1123f-1149f
facial skeleton augmentation, 636
goals of, 636
Grafts and grafting *(Continued)*
for interpositional defects and gaps, mandible *vs.* maxilla, 609-623
allogenic grafts, 622-623
alloplastic grafts, 620-622
autogenous iliac donor sites, 609-620
corticocancellous bloc donor sites, 614-620, 615f, 622-623
human sterile freeze-dried iliac grafts, 622-623
particulate cancellous donor sites, 609-614
porous hydroxyapatite bloc implants, 620-622, 621f-622f
procedures for, 609-613, 610f-613f, 616-620, 616f-620f
options for, 607-609
porous polyethylene augmentation implants, 636
prevalence of, 607
types of, 608-609
allografts, 608
autografts, 608
graft substitutes, 608-609
U.S. Food and Drug Administration (USDA) approval of, 608-609, 622
Granulomas, suture, 1739
Greek architectural orders, 342, 343f-344f
Gregory, E.H., 26
Grow out concept, 91
Growth modification roles, 642, 760, 867-892
Growth patterns and presentations
constriction, maxillary arch, 640-685
deficiency
mandibular, primary, 640-685
maxillary, 686-757
maxillomandibular, 866-896
deformities, mandibular, 758-806
dentofacial disharmonies
adult, 945-991
teenage/young adult, 897-944
excess
mandibular, asymmetric, 807-865
mandibular, relative, 686-757
maxillary, vertical, 758-806
face
long, 758-806
short, 866-896
obstructive sleep apnea (OSA), 992-1058
protrusive patterns, bimaxillary dental, 897-944
Guiot, Gerard, 55
Gummy smile treatments, 802

H

Halogen curing lights, 162
Harmony, facial, 1747
Head assessments, global, 351-360. *See also* Global head and neck assessments.
Head development, 69-72
Head of an Old Man (da Vinci), 348, 348f
Health therapeutics establishment, 203
Hemifacial microsomia (HFM), 1095-1158
approach comparisons, 1152
case studies of, 339f, 365b, 430b, 430f-436f, 433b
causes of, 73-77
characteristics of, 1095
classification patterns of, 1096
dysmorphology with, 1097-1099
audiologic findings, 1098-1099
auditory canal, external, 1098-1099
cranio-orbito-zygomatic region, 1099
ear structures, inner, 1098-1099
ear structures, middle, 1098-1099
facial soft tissues, 1097-1098, 1098f-1099f
maxillomandibular region, 1098f, 1099
facial growth potential with, 1099-1110
importance of, 1099-1100, 1101f-1102f
longitudinal radiographic maxillofacial studies, 1100
long-term facial growth studies, 1100
mandibular growth studies, 1100
metallic jaw implant studies, 1100
progressive mandibular asymmetry studies, 1100-1110, 1103f-1110f, 1112f
historical perspectives of, 1177
in infancy and early childhood, 1096-1097, 1097f
inheritance patterns of, 1095-1096
temporomandibular joint (TMJ)-mandibular malformation classifications, 1101f-1102f, 1110-1111
bone-anchored hearing aid (BAHA) options, 1151-1152
comparisons of, 1111
costochondral graft reconstruction, mandibular, 1122-1150, 1123f-1149f
external auditory canal reconstruction, 1151-1152
external ear reconstruction, 1151
facial soft tissue reconstruction, 1150-1151
first-stage mandibular reconstruction, 1114-1150, 1115f-1120f
Hemifacial microsomia (HFM) *(Continued)*
maxillofacial reconstruction, 1113-1114
middle ear reconstruction, 1151-1152
mixed dentition, 1114-1150, 1115f-1120f
orbital reconstruction, 1111-1113, 1112f
osteodistraction, mandibular reconstruction, 1121-1122
osteodistraction, residual mandibular deformity, 1150
zygomatic reconstruction, 1111-1113, 1112f
Hemihyperplasia, facial, 1276-1277. *See also* Complex malformations.
characteristics of, 1276-1277, 1278f-1279f
vs. hemimandibular hyperplasia (HMH), 1277
historical perspectives of, 1276
neoplasms, associated, 1276-1277
reconstruction in, 1277
Hemimandibular elongation, 855-860
Hemimandibular hyperplasia (HMH), 854-855, 1277
Hemorrhage depressors, 311. *See also* Blood replacement and related considerations.
blood loss limitation effects of, 311
desmopressin, 311
tranexamic acids, 311
Hereditary-developmental-environmental influences, 69-134
comparisons of, 69, 124
head and neck development, 69-72
face, 72, 72f-73f
palate, 72, 74f-75f
skull, 69-72, 70f-71f
hereditary developmental tendencies, 102-105
comparisons of, 102-105, 105f-108f, 157f
prevalence of, 102
skeletal growth, environmental and neuromotor effects on, 105-118, 109f-112f
Apert syndrome, 115
comparisons of, 105, 109f-112f
congenital myopathy effects, 115-118, 116f-117f, 119f-120f
Crouzon syndrome, 115
masseteric muscle hypertrophy, 113
muscle effects, 105-115, 108f-112f, 114f
respiration pattern effects, 115, 116f-117f
soft tissue effects, 105-115, 108f
syndromes and anomalies, 73-102. *See also* Syndromes.
achondroplasia, 91

Hereditary-developmental-environmental influences *(Continued)*
acromegaly, 102
brachial arch syndromes, 73-77, 76f-77f
cleft lip/palate, 77-91, 79f-90f. *See also* Cleft-orthognathic surgeries.
comparisons of, 73
Goldenhar syndrome, 73-77, 76f-77f
hemifacial hypotrophy, 102
midline tissue structure deficiencies, 73
neural crest origin deficiencies and anomalies, 73-77, 76f-77f
overgrowth syndromes and anomalies, 102, 103f
premature suture closure, cranial vault and skull base, 70f-71f, 91-92, 92f-102f
Robin sequence, 90f, 91
Stickler syndrome, 85f-89f, 91
synostosis, 70f-71f, 91-92
teratogens and, 75
Treacher Collins syndrome (TCS), 73-77, 76f-77f
van der Wound syndrome, 77-91, 84f
trauma effects, 118-124, 121f-123f
tumor and growth effects, 124, 125f-131f
Hereditary developmental tendencies, 102-105
HFM. *See* Hemifacial microsomia (HFM).
High partial condylectomy, 860-861
High-performance halogen curing lights, 162
High-risk social behaviors, 218
Hinds, Edward C., 41
Hippocrates, 18-20
Hippocratic principles, 338-339
Histomorphometric analyses, 165
Historical perspectives, 18-60
of ideal facial aesthetics, 337-351. *See also* Ideal facial aesthetics.
milestones
corticotomies, maxillary, 44
craniomaxillary surgery, 47-53, 48f-49f
craniomaxillary surgery, plate and screw fixation for, 47-53, 48f-49f
future innovations, impacts on, 56
mandibular ramus, transoral sagittal splitting of, 34-37, 35f
maxillary corticotomies, revascularization and bone healing after, 44
orthodontia, first use of, 18
osteotomies, dento-osseous segmental (one tooth), 45
Historical perspectives *(Continued)*
osteotomies, Le Fort I (Down-fracture), 44
osteotomies, Le Fort I (type), 38-41, 40f
osteotomies, Le Fort I (Down-fracture) with segmentation, 47
osteotomies, mandibular anterior segmental, 42-43, 43f
osteotomies, mandibular sagittal split, 45
osteotomies, mandibular vertical oblique, 44
osteotomies, maxillary, 30-32
osteotomies, maxillary posterior segmental, 43-44, 43f
osteotomies, maxillary segmental, 42
osteotomies, oblique (chin), 45-47
plate and screw fixation, 47-53, 48f-49f
retrognathism, mandibular advancement for, 29-30
revascularization and bone healing, 44
standard orthognathic procedures, biologic basis studies, 41-47
transoral sliding osseous genioplasty, 37, 37f-38f
pioneers, craniofacial surgery, 53-56
Ginestet, Gustave, 53-55
Guiot, Gerard, 55
Tessier, Paul, 53-56, 55f-56f
Virenque, Maurice, 53-55
pioneers, craniomaxillofacial surgery, 47-53
Champy, Maxime, 50
Luhr, Hans G., 47-53
Michelet, Francois, 50
Rosen, Harvey, 50-53
Schuchardt, Hans, 47-48
Spiessel, Bernd, 50
pioneers, orthodontic, 18-26
Ancient Greek scholars, 18-20
Andrews, Lawrence F., 24-25, 24f
Angle, Edward H., 20-23, 20f-22f
Aristotle, 18-20
Begg, Raymond, 24, 24f
Broadbent, Holly, 23-24, 23f-24f
da Vinci, Leonardo, 18-20
Etruscans, 18-20, 19f
Farrar, J.N., 18-20, 20f
Fauchard, Pierre, 18-20, 19f
Hippocrates, 18-20
Kingsley, Norman W., 18-20, 19f-20f
Profitt, William R., 25-26, 26f
Schange, J.M. Alexis, 18-20
Tweed, Charles, 24, 24f
pioneers, orthognathic surgery, 26-32
Barrow, G.V., 28
Historical perspectives *(Continued)*
Bell, William H., 32, 32f, 41-47
Blair, Vilray P., 22-23, 26-32, 27f, 29f-30f
Caldwell, J.B., 21-22
Cheever, David Williams, 30-32, 31f
Dingman, Reed O., 28
Gilles, Harold, 33-34, 33f, 39, 55, 55f
Gregory, E.H., 26
Hinds, Edward C., 41
Hullihen, Simon, 26-29, 27f
Kazanjian, Varaztad, 30, 31f
Kennedy, J.W., 44
Letterman, G.S., 21-22
Levy, B.M., 42-44
Luhr, Hans, 28-29, 32, 32f
Obwegeser, Hugo L., 32-41, 32f
Pont, Dal, 35-37
Schmid, Edward, 33-34
Schuchardt, Karl, 30-31
Tessier, Paul Louis, 33-34, 34f
Trauner, R., 32-34
Tupper, P., 26
Walker, Robert V., 41-42, 41f
Wassmund, Martin, 30-32, 39
Whipple, J.W., 26-28
HMH. *See* Hemimandibular hyperplasia (HMH).
Hollowing, submental hollowing, 1766
Home/outpatient convalescence, 325. *See also* Convalescence.
anesthesia considerations and, 308-336. *See also* Anesthesia and related considerations.
outpatient regimens, 325
physical therapy, 328b-330b
Hominid species, 6, 8f-9f
Homo floresiensis, 6, 8f-9f
Homo neanderthalensis, 6, 8f-9f
Homo sapiens, 6, 8f-9f
Horizontal deficiency, chin, 1565f-1571f, 1571-1572, 1573f-1593f. *See also* Chin.
Horizontal dimensions, 64
Horizontal prominence, chin, 1572-1595, 1573f-1576f, 1594f-1595f. *See also* Chin.
Hullihen, Simon, 26-29, 27f
Human Ratio (Zeising), 349, 349f
Human sterile freeze-dried iliac grafts, 622-623. *See also* Grafts and grafting.
Hyaline degeneration, 180
Hyaline formation, 165
Hyalinized histologic appearances, 141
Hydroxyapatite alloplastic materials, 614
Hyperactivity, condylar, 860-861
active unilateral, 860-861
type I, 854-855
type II, 855-860
Hyperplasia, hemimandibular (HMH), 854-855, 1277
Hypertrophic turbinates, 287-288, 290f. *See also* Turbinates.
Hypertrophy, conchal, 1714
Hypoplasia, nasomaxillary, 1159. *See also* Binder syndrome.
Hypotensive anesthesia, deliberate (DHA), 308-313. *See also* Anesthesia and related considerations; Deliberate hypotensive anesthesia (DHA).
complications of, 310
historical perspectives of, 308-309, 311-313
inducement techniques for, 309
mean arterial pressure (MAP) and, 309, 318
monitoring during, 310
organ function effects of, 309-310
orthognathic surgery-specific effects of, 310-313
patient positioning and, 309

I

ICR. *See* Idiopathic condylar resorption (ICR).
Ideal facial aesthetics, 337-351
current philosophy, 350-351
evolutionary thinking, 338-350
Alexander the Great, 341-342, 341f
Aristotle, 340-341
Bracciolini, Poggio, 342
da Vinci, Leonardo, 342, 344f, 345-346, 347f-348f
Euclid, 341-342, 341f
Fibonacci, Leonardo, 343
Galen, 339
Hippocratic principles, 338-339
Michelangelo, 342
Phidias, sculptures of, 339, 340f
planetary symphony concept, 338
Plato, 339-341, 340f-341f
Polio, Marcus Vitruvius, 342, 342f
Polykleitos, sculptures of, 338-350, 338f-339f
Pythagoras, 338-350, 338f
Zeising, Adolf, 349, 349f
fundamentals of, 337
beauty, 337
proportion, 337
symmetry, 337
planning techniques for. *See* Planning techniques.
Ideal Rectangle (Fechner), 348-349, 349f
Idiopathic condylar resorption (ICR), 1530-1563
case studies of, 1534
idiopathic condylar resorption (ICR), 1538b, 1539f-1547f, 1543b-1548b, 1554b, 1555f-1560f
progressive condylar resorption (PCR), 1530, 1534b-1538b, 1534f-1537f

Idiopathic condylar resorption (ICR) *(Continued)*
progressive condylar resorption/idiopathic condylar resorption (PCR/ICR), 1548b, 1549f-1553f
clinical perspectives of, 1531-1532
etiology theories of, 1530-1531, 1560
avascular necrosis theory, 1531
dominant theory, 1530-1531
favorable results of, 1560
jaw deformity correction, clinical approaches to, 1533
morphologic findings of, 1530
prevalence of, 1530
Immediate malocclusion, 543-555
Impacted wisdom teeth, removal of, 455, 455f, 467-468
Implants
porous hydroxyapatite bloc implants, 620-622, 621f-622f
porous polyethylene augmentation, 636
Impressions
alginate, 380f-385f, 415
casts, 380f-385f, 415
Incisal tooth surfaces, 138f, 172
Index of Treatment Need, 65
Inferior alveolar nerve injuries, 500-502, 500f-501f, 987. *See also* Nerve injuries.
Inferior turbinates, 287-288, 289f. *See also* Turbinates.
Informed consent
for asymmetric mandibular excess procedures, 862
long face patterns, 802
for maxillary deficiencies, 754
for primary mandibular deficiencies, 683
Informed consent considerations
maxillary vertical excess, 802
Infraorbital nerve injuries, 987-988. *See also* Nerve injuries.
In-hospital convalescence, 323-325. *See also* Convalescence.
airway needs, 323
anesthesia considerations and, 308-336. *See also* Anesthesia and related considerations.
antibiotics, 325
diet and nutrition, 324
discharge criteria, 325
intravenous (IV) fluid therapy, 324
monitoring equipment, 323-324
nasal medications, 325
nausea management, 324
pain management, 325
settings for, 323
vomiting management, 324
wound care, 324
Insignia system, 161
Instrument breakage, 486, 492f
Interdental papilla, 176
Interdisciplinary approaches, 61
Interdisciplinary evaluations, 259
Interface, gingival, 181
Interpositional defects and gaps, 609-623
Interrupted tooth movement, 142. *See also* Tooth.
Intervention planning techniques
analytic model-planning
mandible-first, 424-440
standard, 374-423
complication-related considerations, 475-542
fundamentals, 337-373
grafts and grafting, 607-639
malocclusion prevention, 543-606
procedure sequencing, 441-474
Intracanalicular osteogenesis, 48-50
Intracranial approach, Tessier, 55
Intranasal region, upper airway, 995, 995f
Intra-oral arch wires, 162-163
Intra-oral bracket axillaries, 162-163
Intravenous (IV) fluid therapy, 324
Intubation
complication prevention and, 318-319
vs. extubation. *See* Extubation.
preintubation preparation, 320-321
strategies, 319
tracheal, 318-319
Invisalign, 164
Iowa Pressure Articulation Test, 237, 237b
Isolated cleft palate (ICP), 1441-1470. *See also* Cleft-orthognathic surgeries.
in adolescents, 1443
vs. cleft lip/palate, 1441
controversies of, 1467-1468
coordinated team approaches, 1442
facial growth implications of, 1441-1442
jaw deformities and, 1443
presurgical assessments, 1443-1465
residual deformities, 1443, 1444f-1445f
surgical approaches, 1444f-1465f, 1465-1466
clinical management, 1466
current techniques, 1466
distraction approach, 1467-1468
evolution of, 1465-1466
initial healing, 1466
large residual palata fistula, closure of, 1468
Le Fort I osteotomies, 1467
skeletal stability, 1467
studies of, 1466-1467
timing considerations, 1442-1443
treatment protocols, 1442
unresolved issues of, 1467-1468
IV fluid therapy. *See* Intravenous (IV) fluid therapy.

J

Jaw deformities, secondary, 1471-1529
posttraumatic orthognathic deformities, 1478-1521
clinical characteristics of, 1478-1480
condylar fractures, 1480-1521, 1484f-1521f
late secondary consequences, 1480-1521
malunion after midface (Le Fort I) fracture, 1480, 1481f-1483f
posttraumatic saddle-nose deformity, 1475-1478
bone and cartilage vault involvement, 1475, 1476f-1478f
cartilage vault only involvement, 1475, 1476f-1478f
clinical characteristics of, 1475, 1476f-1479f
posttraumatic segmental dentoalveolar defects, 1521, 1522f-1526f
posttraumatic temporomandibular joint ankylosis, 1471-1475
controversies of, 1472-1475
observations for, 1475
in pediatric patients, 1471-1475
recommendations for, 1475
studies of, 1471-1472, 1473f-1474f
unresolved issues of, 1472-1475
prevalence of, 1471, 1521-1527
Jaw growth maturity assessments, 860
Jaw reorientations, 368
Junctional epithelium, 174-175, 174f-175f

K

Kanon (Polykleitos), 338-339, 338f-339f
Kanon of beauty (Polyclitus), 368-369
Kazanjian, Varaztad, 30, 31f
Kennedy, J.W., 44
Keratinized tissue, 174-175, 174f-175f
KFA. *See* Klippel-Feil anomaly (KFA).
Kinesiologic positions, 237t, 238f
Kingsley, Norman W., 18-20, 19f-20f
Klippel-Feil anomaly (KFA), 1261-1265. *See also* Complex malformations.
characteristics of, 1261-1262
historical perspectives of, 1261
reconstruction in, 1262-1265, 1263f-1265f

L

Labial tooth surfaces, 138f, 172
Lamina dura, 136-137, 136f, 138f
Lamina propria, 176
Lateral facial dysplasia, 1095. *See also* Hemifacial microsomia (HFM).
Learned rule pattern speech errors, 237-240
Leber congenital amaurosis, 12
Le Fort I osteotomies
complications of, 504-525
aseptic necrosis, maxilla, 506-508, 506f-507f
bleeding, significant, 505-506
blindness, 505
extraocular cranial nerve dysfunction, 505
maxillary sinusitis, acute *vs*. chronic, 517-525, 520f-525f
nasal obstructions, acute *vs*. chronic, 517, 518f-520f
nasolacrimal apparatus dysfunction, 505
nerve injuries, infraorbital, 504-505, 504f
visual disturbances, 505
down-fracture, 287-288, 293f-295f, 296-297
malocclusion and, 563-567. *See also* Malocclusion prevention.
maxillary, 446-448, 447f-448f
modified, 1430-1431
standard, 1429-1430
three-segment, 1430-1431
Letterman, G.S., 21-22
Levy, B.M., 42-44
Life cycle stages, 1-3, 3b-4b, 4f-5f
Light-cured band cements, 162
Light prolonged vector-controlled forces, 137
Limbal cell transplantation, 12
Limited neck extension, 317-318
Limited occlusal slides, 271
Limit lines, anterior, 363-367, 364f-365f
Lingual nerve injuries, 502-503, 503f
Lingual tooth surfaces, 138f, 172
Lip incompetence, 201-202
Lip/palate, cleft. *See also* Cleft-orthognathic surgeries.
bilateral (BCLP), 1371-1440
isolated cleft palate (ICP), 1441-1470
unilateral, 1297-1370
Local anesthesia, 1709
Long face patterns, 758-806. *See also* Face.
clinical characteristics of, 759
complications of, 802
definitive reconstruction for, 761-801
arch mechanics, mandibular, 762
arch mechanics, maxillary, 762
orthodontic preparation, 761-762
preorthodontic periodontal considerations, 761
surgical approach, 762-763, 764f-800f
timing of, 761
dental findings of, 759
diagnostic criteria for, 758
etiology of, 759-760
functional aspects of, 758-759
growth modification, preadolescent patients, 760

Long face patterns *(Continued)*
informed consent considerations, 802
orthodontic camouflage approach to, 760
orthodontic molar intrusion approach to, 760-761
patient education for, 802
postsurgical malocclusion, 802-803
postsurgical orthodontic maintenance, 802
prevalence of, 758, 803
radiographic findings of, 759
surgical planning for, 801-802
gummy smile treatments, 802
incisor inclination management, 801-802
maxillomandibular counterclockwise rotation, 801
pitfalls of, 801-802
presurgical, 763-801
two-jaw surgery needs, 801
temporomandibular disorders (TMDs) and, 803
Loss, blood. *See* Blood loss and fluid replacement.
Lower face and neck soft tissues, 1746-1783. *See also* Aesthetics.
complications and unfavorable results, 1766
contour irregularities, 1766
digastric muscle ridging, 1766
hematoma, 1766
infection, 1766
midline ridging, 1766
nerve injury, 1766
skin irregularities, 1766
skin necrosis, 1766
submandibular gland, 1766
submental fullness, residual, 1766
submental hollowing, residual, 1766
facial envelope, aging of, 70f-71f, 1746-1749
characteristics of, 1747
factors of, 1746-1747
superficial muscular aponeurotic system (SMAS), 1746, 1749-1754
midface and neck rejuvenation, 1749-1767
anterior neck approach, 1758-1766
cervical fascia management, 1762-1763
cervical flap elevation, 1762-1763
face-lift approach, traditional, 1749-1758, 1755f-1761f
neck defatting, 1762-1763
patient selection for, 1762
platysma muscle management, 1762-1763
submental approach, 1758-1766
Lower face and neck soft tissues *(Continued)*
midface and neck rejuvenation, principles of, 1747-1749
baseline maxillomandibular skeletal disharmony, 1748f, 1749, 1750f-1754f
degenerative skin changes, 1749
facial harmony, 1747
fat atrophy *vs*. hypertrophy, 1749
volume redistribution *vs*. skin tension, 1749
patient education materials for, 1773
treatment pitfalls, 1766-1767
dental-only approach, 1767-1768
segmental soft-tissue approach, 1768-1769, 1768f-1769f
soft tissue-only approach, 1767
Lower jaw forward protrusion, inability to, 317
Luhr, Hans G., 28-29, 32, 32f, 47-53

M

Macroglossia, 1270-1271
Malformations
Binder syndrome, 1159-1181
complex, 1261-1296
craniosynostosis syndromes, 1182-1260
hemifacial microsomia (HFM), 1095-1158
Treacher Collins syndrome (TCS), 1059-1094
Mallampati test, 318
Malocclusion prevention, 543-606
in bimaxillary dental protrusive growth patterns, 941-942
in Le Fort I osteotomies, 563-567
early malocclusion, 544f-555f, 563-566
immediate malocclusion, 544f-555f, 563-566
late malocclusion, 564f-566f, 566-567, 568f-570f
technical errors, 567
orthodontic movement limitations and, 543
in orthodontic treatments, 567-595, 568f-592f
dental stability, 567-595
relapse, 567-595
specific dentofacial growth patterns, 593-595
prevalence of, 599
in sagittal split ramus osteotomies (SSROs), mandibular, 543-556, 544f-555f
condylar resorption, 556, 557f-559f
continued growth, mandibular, 556, 560f-566f
early malocclusion, 543-555
Malocclusion prevention *(Continued)*
immediate malocclusion, 543-555
late malocclusion, 555-556
malunion, ramus, 556
remodeling, ramus, 556
in transverse maxillary deficiency, 593-595
Mandible-first analytic model-planning, 424-440. *See also* Planning techniques.
advantages of, 424, 425f-426f
case studies, hemifacial microsomia, 339f, 365b, 430b, 430f-436f, 433b
centric relation (CR), 424-427
capture of, 427
controversies of, 424-427
definition of, 424-427
unstable *vs*. unreliable, 427
clinical settings for, 439
execution of, 428-429
technical aspects of, 428-429
techniques for, 429
Mandibular deficiencies, primary, 640-685. *See also* Deficiency patterns.
camouflage approach, orthodontic, 642-643
clinical characteristics of, 641
definitive reconstruction, 302f-304f, 643-682, 643f-647f, 649f-669f, 674f-682f
basic approach, 682
complications of, 683
informed consent for, 683
patient education of, 683
postsurgical orthodontic maintenance, 682-683
temporomandibular disorders (TMDs) and, 683
timing of, 641-642
dental findings of, 641
functional aspects of, 640
growth modification roles, 642
historical perspectives of, 640
maxillary arch constriction, with/without, 640-685
objectives of, 641-642
in preadolescent patients, 642
prevalence of, 640, 683
radiographic findings of, 641
treatment indications for, 641-642
Mandibular deformities, 758-806
Mandibular dysplasia, 1373-1428, 1443, 1444f-1445f
Mandibular excess. *See also* Excess patterns.
asymmetric, 807-865. *See also* Asymmetric mandibular excess.
active unilateral condylar hyperactivity, 860-861
clinical characteristics and types of, 807, 809f-853f
condylar hyperactivity, type I, 854-855
condylar hyperactivity, type II, 855-860
etiology of, 807, 809f-853f, 863
Mandibular excess *(Continued)*
hemimandibular elongation, 855-860
hemimandibular hyperplasia, 854-855
high partial condylectomy, 860-861
jaw growth maturity assessments, 860
misdiagnosis of, 807, 808f
presurgical orthodontic treatments, 861
studies of, 853
surgical approaches, 861-862
relative, 686-757
Mandibular lateral gnathism, 807. *See also* Asymmetric mandibular excess.
Mandibular range-of-motion (ROM), 360
Mandibular skeletal changes, 986
Mandibulofacial dystonia, 1059-1094. *See also* Treacher Collins syndrome (TCS).
approach comparisons, 1091
characteristics of, 1059-1061, 1060f
dysmorphology with, 1062-1063
facial growth potential with, 1063-1069, 1064f-1069f
historical perspectives of, 1177
in infancy and early childhood, 1061-1062
inheritance and genetic markers, 1061, 1061f
skeletal reconstruction staging, 1072-1087, 1073f-1079f
temporomandibular joint (TMJ)-mandibular malformation classification, 1069-1072, 1070f-1071f
testing for, 1061
MAP. *See* Mean arterial pressure (MAP).
Marginal gingiva, 174-175, 174f-175f
Masseteric muscle hypertrophy, 113
Mastication-swallowing-speech considerations, 227-263
functional mechanisms of, 227
interdisciplinary evaluations, 259
jaw deformities with malocclusion effects, 240-253
cause-effect relationships, 252-253
historical perspectives of, 240-252
studies of, 240-252, 241f-251f
mastication mechanism, 230-232, 231f
mastication mechanisms, 230-232, 231f
neuromuscular masticatory system, 227, 228f-229f, 230t
central pattern generators (CPGs) and, 227
dynamic balance of, 227

Mastication-swallowing-speech considerations *(Continued)*
muscles of, 227, 230t
processes of, 227, 228f-229f
orthodontics effects, 253-259
orthognathic surgery effects, 253-259
patient satisfaction, 259
physiologic mechanisms of, 227
speech, 235-240
air stream obstruction categories, 236
articulation, 235
articulators, individual, 235-236, 237t, 238f-239f
assessments, 237, 237b
consonant sounds, 236
error types, 237-240
kinesiologic positions, 237t, 238f
mechanisms, 235, 235f
phonation, 235
phonemes, 235-236
production, 235, 236f
unvoiced sounds, 235
vocal tract variables, 236
voiced sounds, 235
vowel sounds, 236
swallowing mechanisms, 232-235, 233f-234f
assessments, 235
first stage, 233
in normal adult, 232
in normal child, 232-233, 234f
processes of, 232
repetitive swallowing cycle, 233
second stage, 233-235
third stage, 235
velopharyngeal function effects, 254-259, 255f-258f
assessments, 259
velopharyngeal insufficiency (VPI), 254-259, 255f-258f
Masticatory mucosa, 174-175, 174f-175f
Mathematics, definitions of, 338
Maxilla
reorientation, 417
segmentation, 417
Maxillary arch
constriction, 640-685, 1198f-1200f, 1265-1271, 1266f-1269f
expansion, 290f, 297-301
Maxillary deficiencies, 686-757. *See also* Deficiency patterns.
assessments for, 755
camouflage approaches
orthodontic, 688-689
surgical, 689
clinical characteristics of, 687
frontal view, 687
profile view, 687
definitive reconstruction for, 689-753, 690f-693f, 696f-753f
complications of, 754
informed consent and, 754
malocclusions after, 754
patient education for, 754
preorthodontic periodontal checklist, 689-694
Maxillary deficiencies *(Continued)*
suboptimal facial aesthetics after, 754
temporomandibular disorders (TMDs) and, 754
timing of, 689
dental characteristics of, 687
etiology of, 686-687
functional aspects of, 686-757
growth modification roles, 688
hereditary considerations for, 686
preadolescent patients, 688
prevalence of, 686
radiologic findings for, 687
Maxillary dysplasia, 1443, 1444f-1445f
Maxillary excess, vertical, 758-806. *See also* Excess patterns.
Maxillary hypoplasia, 1373-1428
Maxillary incisor crown
horizontal position, 363-367
vertical position, 362-363
Maxillary-mandibular chin advancement (MMCA), 992-1000
Maxillary/nasal base aesthetic unit, 1230
Maxillary vertical excess, 758-806. *See also* Long face patterns.
clinical characteristics of, 759
complications of, 802
definitive reconstruction for, 761-801
dental findings of, 759
diagnostic criteria for, 758
etiology of, 759-760
functional aspects of, 758-759
growth modification roles, 760
informed consent considerations, 802
orthodontic camouflage approach to, 760
orthodontic molar intrusion approach to, 760-761
patient education for, 802
postsurgical malocclusion, 802-803
postsurgical orthodontic maintenance, 802
prevalence of, 758, 803
radiographic findings of, 759
surgical planning for, 801-802
Maxillofacial complex malformations, 1261-1296
comparisons of, 1261, 1288
Down (Trisomy 21) syndrome, 1265-1271
historical perspectives of, 1265-1271
macroglossia in, 1270-1271
reconstruction in, 1271
hemihyperplasia, facial, 1276-1277
characteristics of, 1276-1277, 1278f-1279f
vs. hemimandibular hyperplasia (HMH), 1277
historical perspectives of, 1276
neoplasms, associated, 1276-1277
reconstruction in, 1277
Maxillofacial complex malformations *(Continued)*
Klippel-Feil anomaly (KFA), 1261-1265
characteristics of, 1261-1262
historical perspectives of, 1261
reconstruction in, 1262-1265, 1263f-1265f
neurofibromatosis, 1271-1274
acoustic type (type II), 1271
characteristics of, 776f-779f, 1271-1274
historical perspectives of, 1271
orbitotemporal, 1274
reconstruction in, 1274
von Recklinghausen type (type I), 1273-1274
prevalence of, 1261
vascular, 1277-1288
arteriovenous, 1280-1282, 1286f-1287f
capillary, 1288
characteristics of, 1277, 1280f-1287f
complications of, 1277
historical perspectives of, 1277
lymphatic, 1277-1279, 1282f-1283f
venous, 1279-1280
velocardiofacial syndrome (VCFS), 1288
characteristics of, 1288
pathophysiology of, 1288
prevalence of, 1288
reconstruction in, 1288
Maxillofacial deficiency, 866-896. *See also* Short face patterns.
Class II
Division I, 893
Division II, 893
clinical characteristics of, 867
clinical examinations for, 758, 866
definitive reconstruction, 892-894
dental findings for, 867
functional aspects of, 866-867
growth modification roles, 867-892
postsurgical orthodontic maintenance, 894
prevalence of, 866
radiographic findings for, 867
treatment indications and objectives for, 867, 868f-892f, 895
Maxillofacial trauma
secondary jaw deformities after, 1471-1529. *See also* Secondary jaw deformities.
posttraumatic orthognathic deformities, 1478-1521
posttraumatic saddle-nose deformity, 1475-1478
posttraumatic segmental dentoalveolar defects, 1521, 1522f-1526f
posttraumatic temporomandibular joint ankylosis, 1471-1475
prevalence of, 1471, 1521-1527
Maxillomandibular complex, 995, 995f
Maxillomandibular surgical planning, detailed, 360-368, 364f-365f. *See also* Planning techniques.
anterior nasal spine morphology, 367-368
A-point to B-point relationships, 367
chin morphology, baseline, 368
eight critical decisions, 353f-359f, 360-368
facial views, natural head position, 367
jaw reorientations, 368
maxillary cant (upper face reference), 361-362
maxillary dental midline (upper face reference), 361
maxillary incisor crown horizontal position (upper face reference), 363-367
maxillary reorientation, 367-368
maxilla segmentation, 368
nose floor, baseline pyriform, 367-368
osseous genioplasty considerations, 368
pitch orientation, 367
pogonion locations, 368
rims, baseline pyriform, 367-368
roll orientation, 361-362
yaw orientation, 361
Maximum tooth movement, 142. *See also* Tooth.
MC/FB osteotomies. *See* Monobloc/facial bipartition (MC/FB) osteotomies.
Mean arterial pressure (MAP), 309, 318
Mental health, baseline, 218
Mental nerve injuries, 500-502, 500f-501f, 987. *See also* Nerve injuries.
Mesenchymal cells, 136-137, 136f, 138f
Mesial tooth surfaces, 138f, 172
Metal alloy arch wires, 162
Methyl methacrylate alloplastic materials, 614
Michelangelo, 342
Michelet, Francois, 50
Microgap healing, 48-50
Microsomia, hemifacial (HFM), 1095-1158
approach comparisons, 1152
case studies of, 339f, 365b, 430b, 430f-436f, 433b
characteristics of, 1095
classification patterns of, 1096
dysmorphology with, 1097-1099
audiologic findings, 1098-1099
cranio-orbito-zygomatic region, 1099
ear structures, inner, 1098-1099
ear structures, middle, 1098-1099
facial soft tissues, 1097-1098, 1098f-1099f

Microsomia, hemifacial (HFM) *(Continued)*
maxillomandibular region, 1098f, 1099
facial growth potential with, 1099-1110
importance of, 1099-1100, 1101f-1102f
longitudinal radiographic maxillofacial studies, 1100
long-term facial growth studies, 1100
mandibular growth studies, 1100
metallic jaw implant studies, 1100
progressive mandibular asymmetry studies, 1100-1110, 1103f-1110f, 1112f
historical perspectives of, 1177
in infancy and early childhood, 1096-1097, 1097f
inheritance patterns of, 1095-1096
temporomandibular joint (TMJ)-mandibular malformation classifications, 1101f-1102f, 1110-1111
bone-anchored hearing aid (BAHA) options, 1151-1152
comparisons of, 1111
costochondral graft reconstruction, mandibular, 1122-1150, 1123f-1149f
external auditory canal reconstruction, 1151-1152
external ear reconstruction, 1151
facial soft tissue reconstruction, 1150-1151
first-stage mandibular reconstruction (during mixed dentition), 1114-1150, 1115f-1120f
maxillofacial reconstruction, 1113-1114
middle ear reconstruction, 1151-1152
mixed dentition, 1114-1150, 1115f-1120f
orbital reconstruction, 1111-1113, 1112f
osteodistraction reconstruction, mandibular, 1121-1122
osteodistraction, residual mandibular deformity, 1150
zygomatic reconstruction, 1111-1113, 1112f
Midface aging, 984-985
Midline
ridging, 1766
tissue structure deficiencies, 73
Milestones. *See also* Historical perspectives.
corticotomies, maxillary, 44
craniomaxillary surgery, 47-53, 48f-49f
future innovations, impacts on, 56
mandibular ramus, transoral sagittal splitting of, 34-37, 35f
orthodontia, first use of, 18
osteotomies. *See also* Osteotomies.
dento-osseous segmental (one tooth), 45
Le Fort I (Down-fracture), 44
Le Fort I (type), 38-41, 40f
Le Fort I (Down-fracture) with segmentation, 47
mandibular, anterior segmental, 42-43, 43f
mandibular sagittal split, 45
mandibular vertical oblique, 44
maxillary, 30-32
maxillary, posterior segmental, 43-44
oblique, chin, 45-47
plate and screw fixation for, 47-53, 48f-49f
retrognathism, mandibular advancement for, 29-30
revascularization and bone healing, 44
standard orthognathic procedures, biologic basis studies, 41-47
transoral sliding osseous genioplasty, 37, 37f-38f
Mini-plates, 166
Mini-screws, 166
Misdiagnoses considerations, 807, 808f
Mixed dentition, 1114-1150, 1115f-1120f
MMCA. *See* Maxillary-mandibular chin advancement (MMCA).
Mobilization, inadequate, 567
Model-planning, analytic
mandible-first. *See also* Mandible-first analytic model-planning.
advantages of, 424, 425f-426f
case studies, hemifacial microsomia, 339f, 365b, 430b, 430f-436f, 433b
centric relation (CR), 424-427
clinical settings for, 439
execution of, 428-429
technical aspects of, 428-429
techniques for, 429
standard, 374-423. *See also* Standard analytic model-planning.
accuracy of, 389f-393f, 406-407
alginate impressions, 380f-385f, 415
casts, impression, 380f-385f, 415
components of, 374, 375f-404f
controversies of, 405-408
Model-planning, analytic *(Continued)*
direct visual examinations, 408
Erickson Model Table for, 389f-393f, 406-407
face-bow registration and transfer, 375f-385f, 408-416, 409f-415f
goals of, 374-405, 420
intermediate splint contraction, maxilla positioning, 417-418
key measurements, 408
key steps for, 375f-393f, 407
reorientation. mandibular cast, 418
reorientation, maxilla, 417
segmentation, maxilla, 417
semi-adjustable articulators, 408-416
spatial relationship captures, 408-416
standard facial records, 408
studies of, 419-420
Modified Le Fort I osteotomies, 1430-1431. *See also* Le Fort I osteotomies.
Modulor (Le Corbusier), 349-350, 350f
Molar intrusion approach, 760-761
Mona Lisa (da Vinci), 348, 348f
Monobloc/facial bipartition (MC/FB) osteotomies, 465f, 1237-1246, 1238f-1247f
Morphologic differentiation, 985-986
Motivational considerations, 210, 211f-217f
Mouth-opening ability, 317
Movement, tooth. *See* Tooth.
Multifactorial analyses, 272
Multisource models, 25
Multi-strand arch wires, 162
Myopathic face, 115-118
Myopathy effects, congenital, 115-118, 116f-117f, 119f-120f

N

Nasal airstream obstruction, 236
Nasal airway considerations, 287-307. *See also* Airways.
chronic obstructive nasal breathing, 287-295
causes of, 287-295, 288f-295f, 298f-300f, 302f-304f
comprehensive approaches to, 301
long face patterns and, 287-288, 290f
management of, 296
maxillary deformities and, 287-288, 291f-292f
nasal seroplasty and, 287-288
septal deformities, 287-288, 291f-292f
studies of, 288-295
turbinates and, 287-288, 289f-290f
Nasal airway considerations *(Continued)*
Le Fort I down-fracture osteotomies, 287-288, 293f-295f, 296-297
maxillary arch expansion, 290f, 297-301
nasal floor recontouring, 290f, 295f, 297-301, 298f-300f
quality-of-life issues, 301
residual postoperative nasal obstruction, 297
studies of
case study, 298f-300f, 302-304
COG-ENT chronic rhinosinusitis study, 301
historical perspectives, 287
Nasal Obstruction Symptom Evaluation (NOSE) survey, 296
Nasal floor recontouring, 290f, 295f, 297-301, 298f-300f
Nasal obstructions, 1373-1428, 1443, 1444f-1445f
Nasal Obstruction Symptom Evaluation (NOSE) survey, 296
Nasolacrimal apparatus dysfunction, 505
Nasomaxillary hypoplasia, 1159. *See also* Binder syndrome.
National Health and Nutrition Examination Survey III (NHANES III), 64
Nausea management, 324
Neck and lower face soft tissues, 1746-1783. *See also* Aesthetics.
complications and unfavorable results, 1766
contour irregularities, 1766
digastric muscle ridging, 1766
hematoma, 1766
infection, 1766
midline ridging, 1766
nerve injury, 1766
skin irregularities, 1766
skin necrosis, 1766
submandibular gland, 1766
submental fullness, residual, 1766
submental hollowing, residual, 1766
facial envelope, aging of, 70f-71f, 1746-1749
characteristics of, 1747
factors of, 1746-1747
superficial muscular aponeurotic system (SMAS), 1746, 1749-1754
midface and neck rejuvenation, approaches to, 1749-1767
anterior neck approach, 1758-1766
cervical fascia management, 1762-1763
cervical flap elevation, 1762-1763
face-lift approach, traditional, 1749-1758, 1755f-1761f

Neck and lower face soft tissues *(Continued)*
neck defatting, 1762-1763
patient selection for, 1762
platysma muscle management, 1762-1763
submental approach, 1758-1766
midface and neck rejuvenation, principles of, 1747-1749
baseline maxillomandibular skeletal disharmony, 1748f, 1749, 1750f-1754f
degenerative skin changes, 1749
facial harmony, 1747
fat atrophy *vs*. hypertrophy, 1749
volume redistribution *vs*. skin tension, 1749
patient education materials for, 1773
treatment pitfalls, 1766-1767
dental-only approach, 1767-1768
segmental soft-tissue approach, 1768-1769, 1768f-1769f
soft tissue-only approach, 1767
Neck assessments, global, 351-360. *See also* Global head and neck assessments.
Neck development, 69-72
Neck extension, limited, 317-318
Necrosis, 180
Necrosis with skin loss, 1739
Neovascular age-related macular degeneration, 12
Nerve injuries
facial, 503
inferior alveolar, 500-502, 500f-501f, 987
infraorbital, 987-988
lingual, 502-503, 503f
in lower face and neck soft tissues, 1766
mental, 500-502, 500f-501f, 987
Neural crest origin deficiencies, 73-77, 76f-77f
Neurofibromatosis, 1271-1274. *See also* Complex malformations.
acoustic type (type II), 1271
characteristics of, 776f-779f, 1271-1274
historical perspectives of, 1271
orbitotemporal, 1274
reconstruction in, 1274
von Recklinghausen type (type I), 1273-1274
Neuromotor effects, 105-118, 109f-112f
Neuromuscular masticatory system, 227, 228f-229f, 230t. *See also* Mastication-swallowing-speech considerations.
central pattern generators (CPGs) and, 227
dynamic balance of, 227
muscles of, 227, 230t
processes of, 227, 228f-229f
NHANES III. *See* National Health and Nutrition Examination Survey III (NHANES III).
Nickel-titanium
arch wires, 162
brackets, 142
Nitinol arch wires, 162
Non-keratinized tissue, 174-175
Non-nonsteroidal anti-inflammatory analgesics, 142-143
Nonsteroidal anti-inflammatory drugs (NSAIDs), 142-143
Normal ears *vs*. prominent ears, 1703, 1704f, 1740. *See also* Prominent ears.
Nose floor, baseline pyriform, 367-368
NOSE survey. *See* Nasal Obstruction Symptom Evaluation (NOSE) survey.
Nutrition considerations, 324

O

Obesity-related considerations
bariatric surgery, 1002-1008
obstructive sleep apnea (OSA), 995-996, 995f. *See also* Obstructive sleep apnea (OSA).
Obstructions
functional sites, apnea, 995f, 997f, 1000
nasal, 297
obstructive sleep apnea (OSA), 992-1058. *See also* Obstructive sleep apnea (OSA).
Obstructive nasal breathing, chronic, 287-295
Obstructive sleep apnea (OSA), 992-1058
airway management and, 1002
in childhood, 1000-1002
clinical characteristics of, 993-1000, 995f, 997f-998f
anatomy, 995, 995f
apnea-hypopnea index (AHI) and, 992-1000
associated syndromes, 996
body mass index (BMI) and, 995-996, 995f
central sleep apnea, 1000
mandibular prognathism and, 996
pathophysiology, 993-997
sleep electroencephalograms (EEGs) and, 994
upper airway resistance syndrome (UARS), 994
clinical evaluations for, 997-999
apnea-hypopnea index (AHI) and, 992-1000
attended polysomnography, 999
cephalometric upper airway length (UAL), 999
Epworth Sleepiness Scale (ESS), 998
overnight polysomnography (PSG), 992
severity assessments, 993-1000
diagnoses of, 993-1000
etiology of, 992
Obstructive sleep apnea (OSA) *(Continued)*
functional obstruction sites, 995f, 997f, 1000
intranasal, 1000
retroglossal, 1000
retropalatal, 1000
prevalence of, 992
public health burden of, 1049
risk factors for, 996-997
severity of, 993-1000
studies of, 992
treatment options for, 1002-1049, 1011f-1045f
bariatric surgery, 1002-1008
continuous positive airway pressure (CPAP), 1009
maxillary-mandibular chin advancement (MMCA), 992-1000
oral appliances, 1009-1010
spectrum of, 1010-1045
studies of, 1002
upper airway surgery, 1010-1045
treatment rationales for, 993
Obwegeser, Hugo L., 32-41, 32f
Occlusal function, 137, 139f-140f
Occlusal slides
centric relation-centric occlusion (CR-CO), 271
limited, 271
Occlusal tooth surfaces, 138f, 172
Occlusal trauma, primary *vs*. secondary, 137
Occlusion trauma, 179-180
Oculo-auriculo-vertebral dysplasia, 1095. *See also* Hemifacial microsomia (HFM).
Oculo-auriculo-vertebral spectrum, 1095. *See also* Hemifacial microsomia (HFM).
Open joint procedures, 278
Operating room team preparation, 478
Operative surgery, 22
Oral epithelium, 174-175, 174f-175f
Oral musculature kinesiologic positions, 237t, 240f
Orbital reconstruction, 1111-1113, 1112f
Orbito-naso-zygomatic aesthetic unit, 1230
Orbitotemporal neurofibromatosis, 1274
Organ function effects, 309-310
Orientation
roll, 361-362
yaw, 361
Oronasal fistula, residual, 1373-1428, 1443, 1444f-1445f
Orthodontia, first use of, 18
Orthodontic considerations, 135-170
accelerated osteogenic orthodontics, 164-165
appliances, fixed, 156-163
arch wires, 162
auxiliary attachments, 161
banding *vs*. bonding, 161-162
bracket materials of, 161-162
cements and adhesives, 162
clear brackets, 161
Orthodontic considerations *(Continued)*
design of, 161-162
edgewise appliances, 156-161
historical perspectives of, 156-161
intra-oral bracket axillaries, 162-163
prescriptions for, 161
rotational control of, 161
appliances, removable, 163-164
clinical judgment, importance of, 164
functional appliances, 163-164
historical perspectives of, 163
Invisalign, 164
retention appliances, 164
vs. temporary skeletal anchorage (TADs), 166
diagnoses, accurate, 135
occlusal function, 137, 139f-140f
periodontium responses to, 137
tooth responses to, 137
orthodontic forces, negative sequelae of, 144-156
alveolar ridge height effects, 144-156
biotype considerations and, 156
cortical plate effects, 144-156
excessive mobility effects, 144
gingiva effects, 144-156
pain, 144
pulp effects, 144
root structure effects, 144, 145f-155f, 157f-160f
orthodontic tooth movement, 136-137
anchorage roles in, 143
applied orthodontic force, 142
biologic basis of, 136
bone responses to, 137-141
continuous *vs*. interrupted, 142
control of, 142
cortical wall roles in, 143-144
displacement resistance, 143
force duration and decay, 142
forms of, 142
ideal, 142
limiting factors of, 143-144
maximum, 142
mechanical techniques for, 156
medication effects on, 142-143
orthodontic force levels, 141-142
periodontal ligament (PDL) responses to, 137-141
rate of, 142-143
reciprocal, 143
rotation, 142
sustained force, 137-142
theories of, 137-141
tipping, 142
translation, 142
periodontal ligaments (PDLs)
orthodontic force effects on, 144
structure and function of, 136-137, 136f, 138f

Orthodontic considerations *(Continued)*
treatment philosophies, importance of, 135, 166
Orthodontic tooth movement, 136-137. *See also* Orthodontic considerations.
Orthognathic surgical principles and practice
cleft-orthognathic surgeries
bilateral lip/palate (BCLP), 1371-1440
isolated cleft palate (ICP), 1441-1470
unilateral lip/palate (UCLP), 1297-1370
fundamentals
blood loss and fluid replacement, 308-336
convalescence, 308-336
definition and prevalence, 61-68
facial perspectives, 1-17
formation influences, 69-134
historical perspectives, 18-60
mastication considerations, 227-263
nasal airway considerations, 287-307
orthodontic considerations, 135-170
periodontal considerations, 171-208
psychosocial considerations, 209-226
speech-related considerations, 227-263
swallowing considerations, 227-263
techniques, airway management, 308-336
techniques, anesthesia, 308-336
temporomandibular disorders (TMDs), 264-286
growth patterns and presentations
constriction, maxillary arch, 640-685
deficiency, mandibular (primary), 640-685
deficiency, maxillary, 686-757
deficiency, maxillomandibular, 866-896
deformities, mandibular, 758-806
dentofacial disharmony, adult, 945-991
dentofacial disharmony, teenage/young adult, 897-944
excess, mandibular (asymmetric), 807-865
excess, mandibular (relative), 686-757
excess, maxillary (vertical), 758-806
face, long, 758-806
face, short, 866-896
obstructive sleep apnea (OSA), 992-1058
protrusive, bimaxillary dental, 897-944
Orthognathic surgical principles and practice *(Continued)*
malformations
Binder syndrome, 1159-1181
complex, 1261-1296
craniosynostosis syndromes, 1182-1260
hemifacial microsomia (HFM), 1095-1158
Treacher Collins syndrome (TCS), 1059-1094
posttraumatic deformities
aesthetic alterations, chin, 1564-1612
aesthetic alterations, nose, 1613-1702
aesthetic alterations, prominent ears, 1703-1745
aesthetic alterations, soft tissues (lower face), 1746-1783
aesthetic alterations, soft tissues (neck), 1746-1783
idiopathic condylar resorption (ICR), 1530-1563
jaw, secondary, 1471-1529
secondary jaw deformities, 1471-1529
treatment planning techniques
analytic model-planning, mandible-first, 424-440
analytic model-planning, standard, 374-423
complication-related considerations, 475-542
fundamentals of, 337-373
grafts and grafting, 607-639
malocclusion prevention, 543-606
procedure sequencing, 441-474
OSA. *See* Obstructive sleep apnea (OSA).
Osseous genioplasty
biologic foundations of, 1571
pitfall avoidance strategies for, 1594f-1599f, 1601-1602
techniques, 1597-1600, 1597f-1599f
Osteoblasts, 141
Osteochondroma, mandibular condyle, 807. *See also* Asymmetric mandibular excess.
Osteoclast-mediated bone resorption, 142-143
Osteoclasts, 141
Osteoconduction, 607, 608f
Osteocytes, 136-137, 136f, 138f
Osteodistraction
mandibular reconstruction, 1121-1122
residual mandibular deformity, 1150
Osteogenesis, 607
Osteogenesis, distraction, 1373, 1436
Osteogenic orthodontics, accelerated, 164-165
Osteoinduction, 607
Osteotomies
chin, 452f, 464f-467f
dento-osseous segmental, 45
historical perspectives of, 30-32, 42-43, 43f. *See also* Historical perspectives.
Le Fort I, 563-567
down-fracture, 287-288, 293f-295f, 296-297
malocclusion and, 563-567. *See also* Malocclusion prevention.
maxillary, 446-448, 447f-448f
modified, 1358-1359
standard, 1358
two-segment, 1358-1359
mandibular, 42-43, 43f
mandibular, anterior segmental, 42-43, 43f
maxillary, 30-32
maxillary, posterior segmental, 43-44, 43f
monobloc/facial bipartition (MC/FB), 465f, 1237-1246, 1238f-1247f
oblique, chin, 525-527
sagittal split ramus osteotomies (SSROs), 486-504, 543-556, 544f-555f
saw-cut tibia, dog model, 48-50
sequencing techniques, 441-474. *See also* Sequencing techniques.
unilateral cleft lip/palate (UCLP), 1299f-1305f, 1307f-1312f, 1317f-1354f, 1357-1359
Oto-mandibular dysostosis, unilateral, 1095. *See also* Hemifacial microsomia (HFM).
Otoplasty procedures, 1708-1713. *See also* Prominent ears.
antihelical fold, creation *vs.* deepening, 1709f-1710f, 1712-1713
antihelical fold, marking of, 1712
conchal-mastoid suture placement, 1712f, 1713
conchal wall resection, 1241f, 1711f, 1712
corneal protection, 1709
draping, 1709
dressing placement, 1713, 1714f
general anesthesia intubation, 1709
local anesthesia, 1709
medications, 1709
postauricular flaps, elevation of, 1708f, 1710-1712
postauricular skin excision, 1707f, 1710
postoperative care, 1713
preparation, 1709
results, assessments, 1713-1714, 1715f-1716f, 1718f-1726f, 1728f-1738f
set-back
earlobe, 1713
lower third of ear, 1713
middle third of ear, 1712f, 1713
Otoplasty procedures *(Continued)*
resection, 1241f, 1711f, 1712
upper third of ear, 1713
skin closure, 1713
superior helix-mastoid suture placement, 1713
surgical markings, 1709-1710
Outcomes, patient-centered assessments, 220-222
Outpatient/home convalescence, 325. *See also* Convalescence.
anesthesia considerations and, 308-336. *See also* Anesthesia and related considerations.
outpatient regimens, 325
physical therapy, 328b-330b
Outstanding ears, 1703. *See also* Prominent ears.
Overgrowth syndromes, 102, 103f
Overjet, 271
Overlying soft-tissue envelope, 351-352
Overnight polysomnography (PSG), 992
Overoperated face, 1747
Oxygenation maintenance, 318

P

Pacioli, Luca, 345-346, 346f
Palatal fistula, residual, 1443, 1444f-1445f
Palate development, 72, 74f-75f
Palate/lip, cleft. *See also* Cleft-orthognathic surgeries.
bilateral (BCLP), 1371-1440
isolated cleft palate (ICP), 1441-1470
unilateral (UCLP), 1297-1370
Paracetamol, 142-143
Parathyroid hormones, 142-143
Partial condylectomy, high, 860-861
Passive self-ligating brackets, 161
Patient-centered assessments, 220-222
Patient education
for bimaxillary dental protrusive growth pattern procedures, 941
for convalescence, at home/outpatient and, 326
for long face pattern procedures, 802
for lower face and neck soft tissues procedures, 1773
for maxillary deficiency procedures, 754
for maxillary vertical excess procedures, 802
for prominent ear procedures, 1740
Patient positioning. *See* Positioning considerations.
Pattern generators, central (CPGs), 227
Patterns and presentations
constriction, maxillary arch, 640-685
deficiency
mandibular, primary, 640-685
maxillary, 686-757
maxillomandibular, 866-896

Patterns and presentations *(Continued)*
deformities, mandibular, 758-806
dentofacial disharmony
adult, 945-991
teenage/young adult, 897-944
excess
mandibular, asymmetric, 807-865
mandibular, relative, 686-757
maxillary, vertical, 758-806
face
long, 758-806
short, 866-896
obstructive sleep apnea (OSA), 992-1058
protrusive, bimaxillary dental, 897-944
PCR. *See* Progressive condylar resorption (PCR).
PDLs. *See* Periodontal ligaments (PDLs).
Periodontium tissues, 174-178, 174f-175f
alveolar bone, 177-178
cementum, 176-177
gingiva, 174-178, 175f
oral epithelium, 174-175, 174f-175f
periodontal ligaments (PDLs), 176
Periodontal considerations, 171-208
adult orthodontic treatments, 203-204
aging effects, 178
assessments, importance of, 204
degeneration factors, 171
health therapeutics establishment, 203
orthodontic camouflage maneuvers, detrimental effects of, 202-203
long face patterns, 202
maxillary deficiency, 202
primary mandibular deficiency, 202-203
relative mandibular excess, 202
periodontal disease etiology, 178-181
attachment apparatus lesions, 180
co-destructive factors, 180
dental restorations and, 181
gingival interface and, 181
gingivitis, 178-179, 178f-179f
occlusion trauma, 179-180
orthodontic appliance effects, 180-181
periodontitis, 178-179, 178f-179f
tooth movement forces effects, 180-181
risk factor assessments, biologic *vs*. anatomic, 181-202, 182f-200f
alveolar bone/dental root ratio, 200-201
biotypes and, 181
gingival biotype, 200
inadequate alveolar bone effects, 200-201
Periodontium tissues *(Continued)*
inadequate attached gingiva effects, 200
inadequate gingiva coverage, 201-202
inadequate incisor coverage, 201-202
lip incompetence, 201-202
malocclusion effects, 202
mouth breathing effects, 201-202
nasal obstruction effects, 201-202
vertical maxillary excess, 201-202
teeth, 171-174
anatomy of, 172-174, 173f
cementoenamel junction (CEJ), 172, 173f
cementum, 173f, 174
cervical lines, 172, 173f
cingulum, 173f, 174
crowns, 172, 173f
cusp, 173f, 174
dentin, 173f, 174
enamel, 172-174, 173f
pulp cavity, 173f, 174
root apex, 172, 173f
roots, 172, 173f
structures of, 136f, 171
surfaces of, 138f, 172
Periodontal ligaments (PDLs)
anatomy, 176
orthodontic force effects on, 144
responses of, 137-141
structure and function of, 136-137, 136f, 138f
Periodontitis, 178-179, 178f-179f
Periodontium tissues, 174-178, 174f-175f
alveolar bone, 177-178
cementum, 176-177
gingiva, 174-178, 175f
oral epithelium, 174-175, 174f-175f
periodontal ligaments (PDLs), 176
Perioperative airway management. *See also* Airways.
back-up plans, 318-319
decision factors for, 317, 318f
dentofacial deformity considerations for, 318f, 319-320
difficult airway guidelines, 319
extubation, benchmark airway parameters checklist for, 323
extubation, criteria for, 321-322
extubation, planning for, 322
extubation, strategies for, 321
face-mask ventilation, adequate, 319
head examinations, 317-318
immediate preintubation preparation, 320-321
intubation strategies, 319
lower jaw forward protrusion, inability to, 317
Mallampati test, 318
mouth-opening ability, 317
neck examinations and, 317-318
neck extension, limited, 317-318
Perioperative airway management *(Continued)*
oxygenation maintenance, 318
presurgical airway evaluations, 319
thyromental distance, 318
tracheal intubation, complication prevention and, 318-319
Periorbital aging, 984-985
Personal motivation, 210, 211f-217f
Pfeiffer syndrome, 1201, 1221f-1227f
Pharyngeal musculature kinesiologic positions, 237t, 240f
Pharynx, 995, 995f
Phidias, sculptures of, 339, 340f
Philosophy, definitions of, 338
Phonation, 235
Phonemes, 235-236
Physical therapy considerations, 328b-330b
Pierre Robin sequence, 996
Pioneers. *See also* Historical perspectives.
in craniofacial surgery, 53-56
in craniomaxillofacial surgery, 47-53
orthodontic, 18-26
Planetary symphony concept, 338
Planning techniques
analytic model-planning
mandible-first, 424-440
standard, 374-423
complication-related considerations, 475-542
fundamentals, 337-373
clinical evaluations, 351-369. *See also* Clinical evaluations.
ideal facial aesthetics, 337-351. *See also* Ideal facial aesthetics.
maxillomandibular surgical planning, detailed, 360-368
grafts and grafting, 607-639
malocclusion prevention, 543-606
procedure sequencing, 441-474
Plaque-induced inflammatory response, 180
Plasti-Pore alloplastic materials, 614
Plate removal needs, 479
Plato, 339-341, 340f-341f
Plosive/stop airstream obstruction, 236
Pogonion locations, 368
Polio, Marcus Vitruvius, 342, 342f
Polyacid-modified composite cements, 162
Polyamide-mesh alloplastic materials, 614
Polyclitus (Kanon of beauty), 368-369
Polyethylene augmentation implants, 636
Polykleitos, sculptures of, 338-350, 338f-339f
Polysomnography (PSG), 992
attended, 999
overnight, 992
Pont, Dal, 35-37
Porous
implants
hydroxyapatite bloc, 620-622, 621f-622f
polyethylene augmentation, 636
polyethylene alloplastic materials, 614
Port wine stains, 1288
Positioning considerations, 309
Postauricular flaps, elevation of, 1708f, 1710-1712
Postauricular skin excision, 1707f, 1710
Posterior cranial vault aesthetic unit, 1230
Posttraumatic deformities
aesthetic alterations
chin, 1564-1612
prominent ears, 1703-1745
soft tissues, neck *vs*. lower face, 1746-1783
idiopathic condylar resorption (ICR), 1530-1563
jaw, secondary, 1471-1529. *See also* Secondary jaw deformities.
posttraumatic orthognathic deformities, 1478-1521
posttraumatic temporomandibular joint ankylosis, 1471-1475
prevalence of, 1471, 1521-1527
saddle-nose deformity, 1475-1478
segmental dentoalveolar defects, 1521, 1522f-1526f
Practice and principles (orthognathic surgery)
cleft-orthognathic surgeries
bilateral lip/palate (BCLP), 1371-1440
isolated cleft palate (ICP), 1441-1470
unilateral lip/palate (UCLP), 1297-1370
fundamentals
blood loss and fluid replacement, 308-336
convalescence, 308-336
definition and prevalence, 61-68
facial perspectives, 1-17
formation influences, 69-134
historical perspectives, 18-60
mastication considerations, 227-263
nasal airway considerations, 287-307
orthodontic considerations, 135-170
periodontal considerations, 171-208
psychosocial considerations, 209-226
speech-related considerations, 227-263
swallowing considerations, 227-263
techniques, airway management, 308-336

Practice and principles (orthognathic surgery) *(Continued)*
techniques, anesthesia, 308-336
temporomandibular disorders (TMDs), 264-286
growth patterns and presentations
constriction, maxillary arch, 640-685
deficiency, mandibular (primary), 640-685
deficiency, maxillary, 686-757
deficiency, maxillomandibular, 866-896
deformities, mandibular, 758-806
dentofacial disharmony, adult, 945-991
dentofacial disharmony, teenage/young adult, 897-944
excess, mandibular (asymmetric), 807-865
excess, mandibular (relative), 686-757
excess, maxillary (vertical), 758-806
face, long, 758-806
face, short, 866-896
obstructive sleep apnea (OSA), 992-1058
protrusive, bimaxillary dental, 897-944
malformations
Binder syndrome, 1159-1181
complex, 1261-1296
craniosynostosis syndromes, 1182-1260
hemifacial microsomia (HFM), 1095-1158
Treacher Collins syndrome (TCS), 1059-1094
posttraumatic deformities
aesthetic alterations, chin, 1564-1612
aesthetic alterations, prominent ears, 1703-1745
aesthetic alterations, soft tissues (lower face), 1746-1783
aesthetic alterations, soft tissues (neck), 1746-1783
idiopathic condylar resorption (ICR), 1530-1563
jaw, secondary, 1471-1529
treatment planning techniques
analytic model-planning, mandible-first, 424-440
analytic model-planning, standard, 374-423
complication-related considerations, 475-542
fundamentals, 337-373
grafts and grafting, 607-639
malocclusion prevention, 543-606
procedure sequencing, 441-474
Preferred occlusion, 143
Preintubation preparation, 320-321
Premature suture closure, 70f-71f, 91-92, 92f-102f
Preorthodontic periodontal checklist, 689-694
Presentations and patterns
constriction, maxillary arch, 640-685
deficiency
mandibular (primary), 640-685
maxillary, 686-757
maxillomandibular, 866-896
deformities, mandibular, 758-806
dentofacial disharmony
adult, 945-991
teenage/young adult, 897-944
excess
mandibular, asymmetric, 807-865
mandibular, relative, 686-757
maxillary, vertical, 758-806
face
long, 758-806
short, 866-896
obstructive sleep apnea (OSA), 992-1058
protrusive patterns, bimaxillary dental, 897-944
Pressure-tension theory, 137-141
Presurgical orthodontic treatments, 861
Pretap concept, 50
Prevalence and definitions, 61-68
dental compensation for skeletal discrepancy, 61, 62f
dentofacial deformity definitions, 61, 66
diagnoses, accurate, 63-64
interdisciplinary approaches, 61
jaw deformity and malocclusion prevalence, 64-65
horizontal/sagittal dimension, 64
U.K. Index of Treatment Need, 65
U.S. Health and Nutrition Examination Survey III (NHANES III), 64
vertical dimension, 64
orthodontic-introduced dental compensation, 61
orthognathic procedures, 66
Prevention techniques, malocclusion, 543-606
in Le Fort I osteotomies, 563-567
early malocclusion, 544f-555f, 563-566
immediate malocclusion, 544f-555f, 563-566
late malocclusion, 564f-566f, 566-567, 568f-570f
technical errors, 567
orthodontic movement limitations and, 543
in orthodontic treatments, 567-595, 568f-592f
dental stability, 567-595
relapse, 567-595
specific dentofacial growth patterns, 593-595
Prevention techniques, malocclusion *(Continued)*
prevalence of, 599
in sagittal split ramus osteotomies (SSROs), mandibular, 543-556, 544f-555f
condylar resorption, 556, 557f-559f
continued growth, mandibular, 556, 560f-566f
early malocclusion, 543-555
immediate malocclusion, 543-555
late malocclusion, 555-556
malunion, ramus, 556
remodeling, ramus, 556
in transverse maxillary deficiency, 593-595
Primary mandibular deficiency, 640-685
camouflage approach, orthodontic, 642-643
clinical characteristics of, 641
definitive reconstruction, 302f-304f, 643-682, 643f-647f, 649f-669f, 674f-682f
basic approach, 682
complications of, 683
informed consent for, 683
objectives of, 641-642
patient education of, 683
postsurgical orthodontic maintenance, 682-683
temporomandibular disorders (TMDs) and, 683
timing of, 641-642
dental findings of, 641
functional aspects of, 640
growth modification roles, 642
historical perspectives of, 640
maxillary arch constriction, with/without, 640-685
in preadolescent patients, 642
prevalence of, 640, 683
radiographic findings of, 641
treatment indications for, 641-642
Primary occlusal trauma, 137
Principles and practice (orthognathic surgery)
cleft-orthognathic surgeries
bilateral lip/palate (BCLP), 1371-1440
isolated cleft palate (ICP), 1441-1470
unilateral lip/palate (UCLP), 1297-1370
fundamentals
blood loss and fluid replacement, 308-336
convalescence, 308-336
definition and prevalence, 61-68
facial perspectives, 1-17
formation influences, 69-134
historical perspectives, 18-60
mastication considerations, 227-263
nasal airway considerations, 287-307
Principles and practice (orthognathic surgery) *(Continued)*
orthodontic considerations, 135-170
periodontal considerations, 171-208
psychosocial considerations, 209-226
speech-related considerations, 227-263
swallowing considerations, 227-263
techniques, airway management, 308-336
techniques, anesthesia, 308-336
temporomandibular disorders (TMDs), 264-286
growth patterns and presentations
constriction, maxillary arch, 640-685
deficiency, mandibular (primary), 640-685
deficiency, maxillary, 686-757
deficiency, maxillomandibular, 866-896
deformities, mandibular, 758-806
dentofacial disharmony, adult, 945-991
dentofacial disharmony, teenage/young adult, 897-944
excess, mandibular (asymmetric), 807-865
excess, mandibular (relative), 686-757
excess, maxillary (vertical), 758-806
face, long, 758-806
face, short, 866-896
obstructive sleep apnea (OSA), 992-1058
protrusive patterns, bimaxillary dental, 897-944
malformations
Binder syndrome, 1159-1181
complex, 1261-1296
craniosynostosis syndromes, 1182-1260
hemifacial microsomia (HFM), 1095-1158
Treacher Collins syndrome (TCS), 1059-1094
posttraumatic deformities
aesthetic alterations, chin, 1564-1612
aesthetic alterations, prominent ears, 1703-1745
aesthetic alterations, soft tissues (lower face), 1746-1783
aesthetic alterations, soft tissues (neck), 1746-1783
idiopathic condylar resorption (ICR), 1530-1563
jaw, secondary, 1471-1529

Principles and practice (orthognathic surgery) *(Continued)*
treatment planning techniques
analytic model-planning, mandible-first, 424-440
analytic model-planning, standard, 374-423
complication-related considerations, 475-542
fundamentals, 337-373
grafts and grafting, 607-639
malocclusion prevention, 543-606
procedure sequencing, 441-474
Procedure planning techniques
analytic model-planning
mandible-first, 424-440
standard, 374-423
complication-related considerations, 475-542
fundamentals, 337-373
grafts and grafting, 607-639
malocclusion prevention, 543-606
sequencing, 441-474
Procrit, 313
Production, speech, 235, 236f
Profitt, William R., 25-26, 26f
Progressive condylar resorption (PCR)
case studies of, 1534b-1538b, 1534f-1537f, 1548b, 1549f-1553f
vs. idiopathic condylar resorption (ICR), 1548b, 1549f-1553f. *See also* Idiopathic condylar resorption (ICR).
Prolonged vector-controlled forces, 137
Prominent ears, 1703-1745
aesthetic considerations for. *See* Aesthetics.
anatomy, 1704f, 1705
binding, 1739-1740
complications and suboptimal results, 1714-1739
abscesses, stitch, 1739
antihelical folds, too-sharp, 1714
chondritis, 1739
conchal hypertrophy, inadequate management of, 1714
ear asymmetry, residual, 1731-1739
external auditory canal, pinching of, 1714
flat ear appearance, postsurgical, 1739
infection, cartilage, 1739
necrosis with skin loss, 1739
prevalence of, 1714
prominent earlobe, excessive, 1730
scar formation, abnormal, 1739
suture granulomas, 1739
upper helix failure *vs*. relapse, 1730-1731
embryology of, 1705
historical perspectives of, 1704-1705
Prominent ears *(Continued)*
non-surgical correction, newborns, 1739-1740
vs. normal ears, 1703, 1704f, 1740
objectives for, 1706
ear axis, 1706
ear position, 1706
ear protrusion, 1706
ear size, 1706
otoplasty procedures for, 1708-1713
antihelical fold, creation *vs*. deepening, 1709f-1710f, 1712-1713
antihelical fold, marking of, 1712
conchal-mastoid suture placement, 1712f, 1713
conchal wall resection, 1241f, 1711f, 1712
corneal protection, 1709
draping, 1709
general anesthesia intubation, 1709
local anesthesia, 1709
medications, 1709
postauricular flaps, elevation of, 1708f, 1710-1712
postauricular skin excision, 1707f, 1710
postoperative care, 1713
preparation, 1709
results, assessments, 1713-1714, 1715f-1716f, 1718f-1726f, 1728f-1738f
set-back, earlobe, 1713
set-back, lower third of ear, 1713
set-back, middle third of ear, 1712f, 1713
set-back, resection, 1241f, 1711f, 1712
set-back, upper third of ear, 1713
skin closure, 1713
superior helix-mastoid suture placement, 1713
surgical markings, 1709-1710
patient education materials for, 1740
prevalence of, 1183, 1703-1704
surgical goals for, 1704
surgical principles for, 1706-1707
goals, 1704
techniques, 1707-1708, 1707f-1712f
timing, 1705-1706
Proplast alloplastic materials, 614
Proportion fundamentals, 337
Prosopagnosia, 12-15, 15f
Prostaglandin E, 141
Protrusion, ear, 1706
Protrusive growth patterns, bimaxillary, 897-944
characteristics of, 942
clinical characteristics of, 898, 942
dental findings for, 898
functional aspects of, 898
orthodontic approach
historical perspectives of, 899
studies of, 899-940
techniques, 900f-939f
Protrusive growth patterns, bimaxillary *(Continued)*
prevalence of, 897
radiologic findings for, 898-899
surgical approaches, 940-941
complications of, 941
informed consent and, 941
malocclusion after, 941-942
patient education for, 941
presurgical considerations, 941
temporomandibular disorders (TMDs) and, 942
treatment pitfalls of, 899
Protrusive patterns, bimaxillary, 897-944
PSG. *See* Polysomnography (PSG).
Psychiatric genetics, 15-16, 15t
Psychological considerations, 987
Psychosocial considerations, 209-226
body dysmorphic disorder (BDD), 219
convalescence, educational perspectives, 220
decision-making processes, patient *vs*. family, 219
outcomes, patient-centered assessments, 220-222
presurgical, 210-219
baseline eating disorders, 218
baseline mental health, 218
high-risk social behaviors, 218
personal motivation, 210, 211f-217f
social interaction issues, 218-219
psychological functioning, 219
quality of life, health-related, 220-222
reviews of, 223
successful outcomes, controversies of, 222-223
Puckering, ear, 1708
Pulp effects, 144
Pyriform rim, recontouring, 451f, 464f
Pythagorus, 338-350, 338f

Q

Quality of life, health-related, 220-222

R

Ramus defects, ascending, 623-626
Range-of-motion (ROM)
cervical spine, 360
mandibular, 269, 360
neck, 360
Ratio
extreme and mean (Euclid), 337
Golden, 337, 342
Rear undermining resorption, 180
Reciprocal tooth movement, 143. *See also* Tooth.
Recombinant human erythropoietin, 313
Recontouring, nasal floor, 290f, 295f, 297-301, 298f-300f
Registration, face-bow, 375f-385f, 408-416, 409f-415f
Rehabilitation, comprehensive, 982
Reinforced resin composite brackets, 161
Rejuvenation
facial, 983-986
craniofacial morphologic changes, 985-986
mandibular skeletal changes, 986
midface aging and, 984-985
morphologic differentiation, 985-986
periorbital aging and, 984-985
special considerations for, 983-984
midface and neck, 1749-1767
Relative mandibular excess patterns, 686-757. *See also* Excess patterns.
Removable appliances, 163-164. *See also* Appliances.
clinical judgment, importance of, 164
functional appliances, 163-164
historical perspectives of, 163
Invisalign, 164
retention appliances, 164
vs. temporary skeletal anchorage (TADs), 166
Reorientations
jaw, 368
mandibular cast, 418
maxilla, 417
maxillary, 367-368
Repetitive swallowing cycle, 233
Replacement, fluid. *See* Fluid replacement and blood loss.
Resin composite brackets, 161
Resorbable fixation, 479-481
Resorption
cementum, 180
condylar idiopathic (ICR), 1530-1563. *See also* Idiopathic condylar resorption (ICR).
case studies of, 1534
clinical perspectives of, 1531-1532
etiology theories of, 1530-1531, 1560
favorable results of, 1560
jaw deformity correction, clinical approaches to, 1533
morphologic findings of, 1530
prevalence of, 1530
osteoclast-mediated, 142-143
undermining, 141
undermining, frontal *vs*. rear, 180
Respiration pattern effects, 115, 116f-117f
Retention appliances, 164. *See also* Appliances.
Retinal cell transplantation, 12
Retroflossal region, upper airway, 995, 995f
Retrognathism, 29-30
Retropalatal region, upper airway, 995, 995f
Revascularization, 44
Reverse Trendelenburg position, 309
Rhythmos principle, 338-339
Ribbon arch, 21-22

Rib donor sites, autogenous, 623-626. *See also* Donor sites.
Rickett's dividers, 350, 350f
Ridging
  digastric muscle, 1766
  midline, 1766
Robin sequence, 91
Rod-cone dystrophies, 12
Roll orientation, 361-362
ROM. *See* Range-of-motion (ROM).
Root
  apex, 172, 173f
  structure effects, 144, 145f-155f, 157f-160f
Roots
  anatomic, 172, 173f
Rosen, Harvey, 50-53
Rotational control, fixed appliances, 161. *See also* Fixed appliances.
Rubella virus, 75

S

Saethre-Chotzen syndrome, 1201
Sagittal split ramus osteotomies (SSROs)
  bleeding, significant, 503-504
  mandibular, 486-504
  nerve injuries
    facial, 503
    lingual, 502-503, 503f
    mental, 500-502, 500f-501f
Saw-cut tibia osteotomies, dog model, 48-50
Schange, J.M. Alexis, 18-20
Schmid, Edward, 33-34
Schuchardt, Hans, 47-48
Schuchardt, Karl, 30-31
Screw removal needs, 479
Secondary jaw deformities, 1471-1529
  posttraumatic orthognathic deformities, 1478-1521
    clinical characteristics of, 1478-1480
    condylar fractures, 1480-1521, 1484f-1521f
    late secondary consequences, 1480-1521
    malunion after midface (Le Fort I) fracture, 1480, 1481f-1483f
  posttraumatic saddle-nose deformity, 1475-1478
    bone and cartilage vault involvement, 1475, 1476f-1478f
    cartilage vault only involvement, 1475, 1476f-1478f
    clinical characteristics of, 1475, 1476f-1479f
  posttraumatic segmental dentoalveolar defects, 1521, 1522f-1526f
  posttraumatic temporomandibular joint ankylosis, 1471-1475
    controversies of, 1472-1475
    observations for, 1475
    in pediatric patients, 1471-1475
    recommendations for, 1475
Secondary jaw deformities *(Continued)*
    studies of, 1471-1472, 1473f-1474f
    unresolved issues of, 1472-1475
  prevalence of, 1471, 1521-1527
Secondary occlusal trauma, 137
Segmental soft-tissue approach, 1768-1769, 1768f-1769f
Segmentation, 368, 417
Self-correction, 91
Self-etching primers, 162
Self-ligating brackets, 142, 161-162
Semi-adjustable articulators, 408-416
Sensory loss, 987-988
Sensory recovery, 988
Septal deflections, 287-288
Septal thickenings, 287-288
Septoplasty, 451-452, 453f-454f
Sequencing techniques, 441-474
  approaches to, 441, 473-474
  historical perspectives of, 441
  orthognathic and simultaneous procedures, 441-473
    anesthesia, emergence from, 471-473
    anterior nasal spine, recontouring, 451f, 464f
    anterior neck soft tissue management, 471
    cortical cuts, 442-446, 444f-445f
    inferior turbinates, reduction of, 451-452, 453f-454f
    mandibular wisdom teeth, removal of, 467-468
    maxillary wisdom teeth, removal of, 455, 455f
    maxilla, segmentation of, 448-449, 449f, 455-458, 456f-458f
    nasal floor, recontouring, 451f, 464f
    osteotomies, chin, 452f, 464f-467f
    osteotomies, maxillary (Le Fort I), 446-451, 447f-448f, 450f-453f, 458-461, 459f-463f
    osteotomies, sagittal split ramus (SSROs), 442-446, 444f-445f, 465-471, 468f-470f, 472f-473f
    preparation and draping, 441-442, 442f-444f
    pyriform rim, recontouring, 451f, 464f
    septoplasty, 451-452, 453f-454f
    wound closure, 471, 473f
Set-back
  earlobe, 1713
  lower third of ear, 1713
  middle third of ear, 1712f, 1713
  resection, 1241f, 1711f, 1712
  upper third of ear, 1713
Set back/setting back concept, 28
Sharpey's fibers, 175-176, 175f
Short face patterns, 866-896. *See also* Face.
  Class II
    Division I, 893
    Division II, 893
  clinical characteristics of, 867
  clinical examinations for, 758, 866
  definitive reconstruction, 892-894
    complications of, 894
    informed consent of, 894
    malocclusion after, 894-895
    orthodontic preparation, 893
    patient education for, 894
    presurgical planning, 894
    surgical approaches, 893-894
    temporomandibular disorders (TMDs) and, 895
    timing of, 892-893
  dental findings for, 867
  functional aspects of, 866-867
  growth modification, preadolescent patients, 867-892
  postsurgical orthodontic maintenance, 894
  radiographic findings for, 867
  treatment indications and objectives for, 867, 868f-892f, 895
Significant bleeding, 503-504. *See also* Blood replacement and related considerations.
Silicon alloplastic materials, 614
Silver splints, 32
Sinus blockage, 1373-1428, 1443, 1444f-1445f
Six Elements of Orofacial Harmony (Andrews), 25
Six Keys of Optimal Occlusion (Andrews), 25
Skeletal
  augmentation, face, 636
  changes, mandibular, 986
  growth, influences of, 105-118, 109f-112f
  mobilization, inadequate, 567
  reconstruction staging, 1072-1087, 1073f-1079f
    bone-anchor hearing aid (BAHA) options, 1090-1091
    external auditory canal reconstruction, 1090-1091
    external ear reconstruction, 1089-1090
    facial soft tissue reconstruction, 1089
    mandibular reconstruction, first-stage (during mixed dentition), 1088-1089
    mandibular reconstruction, first-stage (in newborns), 1087-1088
    maxillomandibular reconstruction, 1072-1087
    middle ear reconstruction, 1090-1091
    nasal reconstruction, 1074f-1076f, 1089
Skeletal *(Continued)*
    orbital reconstruction, 1072, 1073f-1083f
    zygomatic reconstruction, 1072, 1073f-1083f
  stability, post-reconstruction, 1177
    method comparisons, 1177
    patient comparisons, 1177
    result comparisons, 1177
Skull
  comparisons, 5-6, 6f-8f
  development of, 69-72, 70f-71f
Sleep apnea, obstructive (OSA), 992-1058
  airway management and, 1002
  in childhood, 1000-1002
  clinical characteristics of, 993-1000, 995f, 997f-998f
    anatomy, 995, 995f
    apnea-hypopnea index (AHI) and, 992-1000
    associated syndromes, 996
    body mass index (BMI) and, 995-996, 995f
    central sleep apnea, 1000
    mandibular prognathism and, 996
    pathophysiology, 993-997
    sleep electroencephalograms (EEGs) and, 994
    upper airway resistance syndrome (UARS), 994
  clinical evaluations for, 997-999
    apnea-hypopnea index (AHI) and, 992-1000
    attended polysomnography, 999
    cephalometric upper airway length (UAL), 999
    Epworth Sleepiness Scale (ESS), 998
    overnight polysomnography (PSG), 992
    severity assessments, 993-1000
  diagnoses of, 993-1000
  etiology of, 992
  functional obstruction sites, 995f, 997f, 1000
    intranasal, 1000
    retroglossal, 1000
    retropalatal, 1000
  prevalence of, 992
  public health burden of, 1049
  risk factors for, 996-997
  severity of, 993-1000
  studies of, 992
  treatment options for, 1011f-1045f
    bariatric surgery, 1002-1008
    continuous positive airway pressure (CPAP), 1009
    maxillary-mandibular chin advancement (MMCA), 992-1000
    oral appliances, 1009-1010
    spectrum of, 1010-1045
    studies of, 1002
    upper airway surgery, 1010-1045
    uvulopalatopharyngoplasty (UPPP), 1010-1045
  treatment rationales for, 993

Slides, occlusal
centric relation-centric occlusion (CR-CO), 271
limited, 271
SMAS. *See* Superficial muscular aponeurotic system (SMAS).
Social behaviors, high-risk, 218
Social interaction issues, 218-219
Social-psycho considerations, 209-226
Soft tissue-only approach, 1767
Soft tissues, face *vs*. neck, 1746-1783. *See also* Aesthetics.
complications and unfavorable results, 1766
contour irregularities, 1766
digastric muscle ridging, 1766
hematoma, 1766
infection, 1766
midline ridging, 1766
nerve injury, 1766
skin irregularities, 1766
skin necrosis, 1766
submandibular gland, 1766
submental fullness, residual, 1766
submental hollowing, residual, 1766
facial envelope, aging of, 70f-71f, 1746-1749
characteristics of, 1747
factors of, 1746-1747
superficial muscular aponeurotic system (SMAS), 1746, 1749-1754
midface and neck rejuvenation approaches
anterior neck approach, 1758-1766
cervical fascia management, 1762-1763
midface and neck rejuvenation, approaches to, 1749-1767
anterior neck approach, 1758-1766
cervical fascia management, 1762-1763
cervical flap elevation, 1762-1763
face-lift approach, traditional, 1749-1758, 1755f-1761f
neck defatting, 1762-1763
patient selection for, 1762
platysma muscle management, 1762-1763
submental approach, 1758-1766
midface and neck rejuvenation, principles of, 1747-1749
baseline maxillomandibular skeletal disharmony, 1748f, 1749, 1750f-1754f
degenerative skin changes, 1749
facial harmony, 1747
fat atrophy *vs*. hypertrophy, 1749
volume redistribution *vs*. skin tension, 1749
Soft tissues, face *vs*. neck *(Continued)*
mobilization, inadequate, 567
patient education materials for, 1773
treatment pitfalls, 1766-1767
dental-only approach, 1767-1768
segmental soft-tissue approach, 1768-1769, 1768f-1769f
soft tissue-only approach, 1767
Spatial orientation
articulator-mounted mandibular unit, 416
articulator-mounted maxillary unit, 416
Spatial relationship captures, 408-416
Specialized mucosa, 174-175, 174f-175f
Speech-mastication-swallowing considerations, 227-263
functional mechanisms of, 227
interdisciplinary evaluations, 259
jaw deformities with malocclusion effects, 240-253
cause-effect relationships, 252-253
historical perspectives of, 240-252
studies of, 240-252, 241f-251f
mastication mechanisms, 230-232, 231f
neuromuscular masticatory system, 227, 228f-229f, 230t
central pattern generators (CPGs) and, 227
dynamic balance of, 227
muscles of, 227, 230t
processes of, 227, 228f-229f
orthodontics effects, 253-259
orthognathic surgery effects, 253-259
patient satisfaction, 259
physiologic mechanisms of, 227
speech, 235-240
air stream obstruction categories, 236
articulation, 235
articulators, individual, 235-236, 237t, 238f-239f
assessments, 237, 237b
consonant sounds, 236
error types, 237-240
kinesiologic positions, 237t, 238f
mechanisms, 235, 235f
phonation, 235
phonemes, 235-236
production, 235, 236f
unvoiced sounds, 235
vocal tract variables, 236
voiced sounds, 235
vowel sounds, 236
swallowing mechanisms, 232-235, 233f-234f
assessments, 235
first stage, 233
in normal adult, 232
Speech-mastication-swallowing considerations *(Continued)*
in normal child, 232-233, 234f
processes of, 232
repetitive swallowing cycle, 233
second stage, 233-235
third stage, 235
velopharyngeal function effects, 254-259, 255f-258f
assessments, 259
velopharyngeal insufficiency (VPI), 254-259, 255f-258f
Spiessel, Bernd, 50
Split strength, inadequate, 567
SSROs. *See* Sagittal split ramus osteotomies (SSROs).
Stainless steel arch wires, 162
Standard analytic model-planning, 374-423. *See also* Analytic model-planning.
accuracy of, 389f-393f, 406-407
alginate impressions, 380f-385f, 415
casts, impression, 380f-385f, 415
components of, 374, 375f-404f
controversies of, 405-408
articular device options, 405
bite registration materials, 379f-380f, 406
capturing, 406
centric relation (CR) capture, 379f-380f, 405-406
computer-aided software design, 408
face-bow device options, 405
model-planning techniques, accuracy of, 406-407
site measurements, inaccurate, 406
direct visual examinations, 408
Erickson Model Table for, 389f-393f, 406
reorientation, maxillary casts, 417
site measurements, maxilla reorientation, 406
spatial orientation, articulator-mounted mandibular unit, 416
spatial orientation, articulator-mounted maxillary unit, 416
face-bow registration and transfer, 375f-385f, 408-416, 409f-415f
goals of, 374-405, 420
intermediate splint contraction, maxilla positioning, 417-418
key measurements, 408
key steps for, 375f-393f, 407
reorientation
mandibular cast, 418
maxilla, 417
segmentation, maxilla, 417
semi-adjustable articulators, 408-416
spatial relationship captures, 408-416
Standard analytic model-planning *(Continued)*
splints
final contraction, mandible positioning, 418
prefabricated acrylic, finishing of, 418-419
standard facial records, 408
studies of, 419-420
materials and methods, 419
planned *vs*. actual procedural comparisons, 420f, 421t
radiographic analyses, 419, 420f
results, 419-420
Standard facial records, 408
Step-by-step sequencing techniques, 441-474
approaches to, 441, 473-474
historical perspectives of, 441
orthognathic and simultaneous procedures, 441-473
anesthesia, emergence from, 471-473
anterior nasal spine, recontouring, 451f, 464f
cortical cuts, 442-446, 444f-445f
inferior turbinates, reduction of, 451-452, 453f-454f
mandibular wisdom teeth, removal of, 467-468
maxillary wisdom teeth, removal of, 455, 455f
maxilla, segmentation of, 448-449, 449f, 455-458, 456f-458f
nasal floor, recontouring, 451f, 464f
osteotomies, chin, 452f, 464f-467f
osteotomies, maxillary (Le Fort I), 446-451, 447f-448f, 450f-453f, 458-461, 459f-463f
osteotomies, sagittal split ramus (SSROs), 442-446, 444f-445f, 465-471, 468f-470f, 472f-473f
preparation and draping, 441-442, 442f-444f
pyriform rim, recontouring, 451f, 464f
septoplasty, 451-452, 453f-454f
wound closure, 471, 473f
Sterile freeze-dried iliac grafts, 622-623. *See also* Grafts and grafting.
Stickler syndrome, 85f-89f, 91
Straight-wire appliances, 25
Strong *vs*. weak chin, 1564-1571. *See also* Chin.
Struge-Weber syndrome, 1282-1288
Submandibular gland, 1766
Submental
fullness, residual, 1766
hollowing, residual, 1766
Substitutes, grafts, 608-609. *See also* Grafts and grafting.
Substitution speech errors, 237-240
Sulcular epithelium, 174-175, 174f-175f

Summetria concept, 338-339
Superelastic arch wires, 162
Superficial muscular aponeurotic system (SMAS), 1746, 1749-1754
Superior helix-mastoid suture placement, 1713
SureSmile system, 161
Surgery and Diseases of the Mouth and Jaw (Blair), 19f, 29
Surgical principles and practice
  cleft-orthognathic surgeries
    bilateral lip/palate (BCLP), 1371-1440
    isolated cleft palate (ICP), 1441-1470
    unilateral lip/palate (UCLP), 1297-1370
  fundamentals
    blood loss and fluid replacement, 308-336
    convalescence, 308-336
    definition and prevalence, 61-68
    facial perspectives, 1-17
    formation influences, 69-134
    historical perspectives, 18-60
    mastication considerations, 227-263
    nasal airway considerations, 287-307
    psychosocial considerations, 209-226
    speech-related considerations, 227-263
    swallowing considerations, 227-263
    techniques, airway management, 308-336
    techniques, anesthesia, 308-336
    temporomandibular disorders (TMDs), 264-286
  growth patterns and presentations
    constriction, maxillary arch, 640-685
    deficiency, mandibular (primary), 640-685
    deficiency, maxillary, 686-757
    deficiency, maxillomandibular, 866-896
    deformities, mandibular, 758-806
    dentofacial disharmony, adult, 945-991
    dentofacial disharmony, teenage/young adult, 897-944
    excess, mandibular (asymmetric), 807-865
    excess, mandibular (relative), 686-757
    excess, maxillary (vertical), 758-806
    face, long, 758-806
    face, short, 866-896
    obstructive sleep apnea (OSA), 992-1058
    protrusive, bimaxillary dental, 897-944
  malformations
    Binder syndrome, 1159-1181
    complex, 1261-1296
Surgical principles and practice *(Continued)*
    craniosynostosis syndromes, 1182-1260
    hemifacial microsomia (HFM), 1095-1158
    Treacher Collins syndrome (TCS), 1059-1094
  posttraumatic deformities
    aesthetic alterations, chin, 1564-1612
    aesthetic alterations, prominent ears, 1703-1745
    aesthetic alterations, soft tissues (lower face), 1746-1783
    aesthetic alterations, soft tissues (neck), 1746-1783
    idiopathic condylar resorption (ICR), 1530-1563
    jaw, secondary, 1471-1529
  treatment planning techniques
    analytic model-planning, mandible-first, 424-440
    analytic model-planning, standard, 374-423
    complication-related considerations, 475-542
    grafts and grafting, 607-639
    malocclusion prevention, 543-606
    procedure sequencing, 441-474
Suture closure, premature, 70f-71f, 91-92, 92f-102f
Suture granulomas, 1739
Swallowing-mastication-speech considerations, 227-263
  functional mechanisms of, 227
  interdisciplinary evaluations, 259
  jaw deformities with malocclusion effects, 240-253
    cause-effect relationships, 252-253
    historical perspectives of, 240-252
    studies of, 240-252, 241f-251f
  mastication mechanisms, 230-232, 231f
  neuromuscular masticatory system, 227, 228f-229f, 230t
    central pattern generators (CPGs) and, 227
    dynamic balance of, 227
    muscles of, 227, 230t
    processes of, 227, 228f-229f
  orthodontics effects, 253-259
  orthognathic surgery effects, 253-259
  patient satisfaction, 259
  physiologic mechanisms of, 227
  speech, 235-240
    air stream obstruction categories, 236
Swallowing-mastication-speech considerations *(Continued)*
    articulation, 235
    articulators, individual, 235-236, 237t, 238f-239f
    assessments, 237, 237b
    consonant sounds, 236
    error types, 237-240
    kinesiologic positions, 237t, 238f
    mechanisms, 235, 235f
    phonation, 235
    phonemes, 235-236
    production, 235, 236f
    unvoiced sounds, 235
    vocal tract variables, 236
    voiced sounds, 235
    vowel sounds, 236
  swallowing mechanisms, 232-235, 233f-234f
    assessments, 235
    first stage, 233
    in normal adult, 232
    in normal child, 232-233, 234f
    processes of, 232
    repetitive swallowing cycle, 233
    second stage, 233-235
    third stage, 235
  velopharyngeal function effects, 254-259, 255f-258f
    assessments, 259
    velopharyngeal insufficiency (VPI), 254-259, 255f-258f
Symbolic transformation, 1
Symbolism, 1
Symmetry fundamentals, 337
Syndromes
  Apert, 1184-1200, 1190f-1193f, 1707f-1709f
  Binder, 1159-1181
  brachial arch, 73-77, 76f-77f
  Carpenter, 1226
  craniosynostosis, 1182-1260
  Crouzon, 1201, 1202f-1207f, 1213f-1220f, 1247f-1249f, 1249-1251, 1711f
  Down (Trisomy 21), 1265-1271
  formation influences on, 73-102. *See also* Formation influences.
  Goldenblat, 1095. *See also* Hemifacial microsomia (HFM).
  Goldenhar, 73-77, 76f-77f
  influences on, 73-102
  overgrowth, 102, 103f
  Pfeiffer, 1201, 1221f-1227f
  Saethre-Chotzen, 1201
  Stickler, 85f-89f, 91
  Struge-Weber, 1282-1288
  Treacher Collins (TCS), 73-77, 76f-77f, 1059-1094
  Treacher Collins-Franceschetti, 55
  upper airway resistance (UARS), 994
  van der Wound, 77-91, 84f
  velocardiofacial (VCFS), 1288

T

TADs. *See* Temporary anchorage devices (TADs).
TCS. *See* Treacher Collins syndrome (TCS).
Teenage/young adult dentofacial disharmony, 897-944. *See also* Dentofacial disharmonies.
  bimaxillary dental protrusive growth patterns, 897-944
  characteristics of, 942
  clinical characteristics of, 898
  dental findings for, 898
  functional aspects of, 898
  orthodontic approach, 898
    historical perspectives of, 899
    studies of, 899-940
    techniques, 900f-939f
  prevalence of, 897
  radiologic findings for, 898-899
  surgical approaches, 940-941
    complications of, 941
    informed consent and, 941
    malocclusion after, 941-942
    patient education for, 941
    postsurgical considerations, 941
    temporomandibular disorders (TMDs) and, 942
  treatment pitfalls of, 899
Teflon alloplastic materials, 614
Telephone ear, 1703-1704
Telephone ear appearance, postsurgical, 1739
Templin-Daley Screening Test of Articulation, 237, 237b
Temporary anchorage devices (TADs), 142-143
Temporomandibular disorders (TMDs), 264-286
  approaches to, 264-269, 265f-269f
  asymmetric mandibular excess and, 862-863
  bimaxillary dental protrusive growth patterns and, 942
  complications and, 478
  global head and neck assessments and, 360
  incidence of, 264-269
  jaw deformity effects on, 274-277
  mandibular range-of-motion (ROM), 269
  maxillary deficiencies and, 754
  National Institutes of Health (NIH) treatment guidelines for, 278
  occlusal conditions associated with, 271-272
    centric relation-centric occlusion (CR-CO) slides, 271
    multifactorial analyses of, 272
    overjet, 271
    posterior teeth, missing, 272
    unilateral maxillary (lingual) crossbite, 271-272
  occlusal features and factors of, 269-271, 270b
  occlusion effects on, 277-278
  open joint procedures and, 278

Temporomandibular disorders (TMDs) *(Continued)*
orthodontic treatment effects on, 272-274
comparisons of, 273-274
historical perspectives of, 272-273
preventative-specific treatments, 273
relief-specific treatments, 273
studies of, 272-273
orthognathic correction effects on, 274-277
comparisons of, 277
historical perspectives of, 274-277
studies of, 274-277
prevalence of, 278
temporomandibular joint (TMJ) diagnostic categories and, 269-271, 270b
Temporomandibular joint (TMJ)-mandibular malformation classifications, 1069-1072, 1070f-1071f
bone-anchored hearing aid (BAHA) options, 1151-1152
comparisons of, 1111
costochondral graft reconstruction, mandibular, 1122-1150, 1123f-1149f
external auditory canal reconstruction, 1151-1152
external ear reconstruction, 1151
facial soft tissue reconstruction, 1150-1151
first-stage mandibular reconstruction, 1114-1150, 1115f-1120f
maxillofacial reconstruction, 1113-1114
mixed dentition, 1114-1150, 1115f-1120f
orbital reconstruction, 1111-1113, 1112f
osteodistraction mandibular reconstruction, 1121-1122
osteodistraction, residual mandibular deformity, 1150
zygomatic reconstruction, 1111-1113, 1112f
Tessier intracranial approach, 55
Tessier, Paul Louis, 33-34, 34f, 53-56, 55f-56f
Thalidomide, 75
Thermally-induced superelastic arch wires, 162
Thickenings, septal, 287-288
Third molar management, 478-479, 480f
Three-segment maxilla segmentation, 455-458, 456f-458f
Thyromental distance, 318
Thyroxin, 142-143
Tipping, 142
Tissues, soft. *See* Soft tissues, face *vs*. neck.
Tissue structure deficiencies, 73
Titanium
fixation, 479-481
titanium-nickel alloy arch wires, 162
brackets, 142
TMDs. *See* Temporomandibular disorders (TMDs).
TMJ. *See* Temporomandibular joint (TMJ)-mandibular malformation classifications.
Too-sharp antihelical folds, 1714
Tooth
anatomy, 173f, 174
movement, orthodontic, 136-137. *See also* Orthodontic considerations.
anchorage roles in, 143
applied orthodontic force, 142
biologic basis of, 136
bone responses to, 137-141
continuous *vs*. interrupted, 142
control of, 142
cortical wall roles in, 143-144
displacement resistance, 143
force duration and decay, 142
forms of, 142
ideal, 142
limiting factors of, 143-144
maximum, 142
mechanical techniques for, 156
medication effects on, 142-143
orthodontic force levels, 141-142
periodontal ligament (PDL) responses to, 137-141
rate of, 142-143
reciprocal, 143
rotation, 142
sustained force, 137-142
theories of, 137-141
tipping, 142
translation, 142
Traditional face-lift approach, 1749-1758, 1755f-1761f
Trainer, R., 32-34
Tranexamic acids, 311
Transfer, face-bow, 375f-385f, 408-416, 409f-415f
Transformations, evolutionary, 1, 4-6
craniofacial evolution, 4-5
definition of, 1
hominid species, 6, 8f-9f
mammalian brain, 4, 5f
molecular components, 4-5
primate brain, 4-5
skull comparisons, 5-6, 6f-8f
Transoral sliding osseous genioplasty, 37, 37f-38f
Transverse maxillary deficiency, 593-595
Traumatic occlusion, 137
Treacher Collins-Francescherri syndrome, 55
Treacher Collins syndrome (TCS), 1059-1094
approach comparisons, 1091
characteristics of, 1059-1061, 1060f
Treacher Collins syndrome (TCS) *(Continued)*
dysmorphology with, 1062-1063
audiologic findings, 1063
auditory canal, external, 1063
cranio-orbita-zygomatic region, 1062
ear structures, middle and inner, 1063
facial soft tissues, 1063
maxillomandibular region, 1062-1063
facial growth potential with, 1063-1069, 1064f-1069f
formation influences, 73-77, 76f-77f. *See also* Formation influences.
historical perspectives of, 1177
in infancy and early childhood, 1061-1062
inheritance and genetic markers, 1061, 1061f
obstructive sleep apnea (OSA) and, 996. *See also* Obstructive sleep apnea (OSA).
skeletal reconstruction staging, 1072-1087, 1073f-1079f
bone-anchor hearing aid (BAHA) options, 1090-1091
external auditory canal reconstruction, 1090-1091
external ear reconstruction, 1089-1090
facial soft tissue reconstruction, 1089
mandibular reconstruction, first-stage (during mixed dentition), 1088-1089
mandibular reconstruction, first-stage (in newborns), 1087-1088
maxillomandibular reconstruction, 1072-1087
middle ear reconstruction, 1090-1091
nasal reconstruction, 1074f-1076f, 1089
orbital reconstruction, 1072, 1073f-1083f
zygomatic reconstruction, 1072, 1073f-1083f
temporomandibular joint (TMJ)-mandibular malformation classification, 1069-1072, 1070f-1071f
testing for, 1061
Treatise on Oral Deformities as a Branch of Mechanical Surgery, A (Kingsley), 18, 19f
Treatise on the Irregularities of the Teeth and Their Corrections, A (Farrar), 20, 20f
Treatment of Malocclusion of the Teeth (Angle), 20-21, 21f
Treatment of Malocclusion of the Teeth and Fractures of the Mandible: Angle's System (Angle), 22-23
Treatment planning techniques
analytic model-planning
mandible-first, 424-440
standard, 374-423
complication-related considerations, 475-542
fundamentals, 337-373
clinical evaluations, 351-369. *See also* Clinical evaluations.
ideal facial aesthetics, 337-351. *See also* Ideal facial aesthetics.
maxillomandibular surgical planning, detailed, 360-368
grafts and grafting, 607-639
malocclusion prevention, 543-606
procedure sequencing, 441-474
Trisomy 21 (Down) syndrome, 1265-1271. *See also* Complex malformations.
characteristics of, 1198f-1200f, 1265-1271, 1266f-1269f
historical perspectives of, 1265-1271
macroglossia in, 1270-1271
obstructive sleep apnea (OSA) and, 996. *See also* Obstructive sleep apnea (OSA).
reconstruction in, 1271
Tupper, P., 26
Turbinates
hypertrophic, 287-288, 290f
inferior, 287-288, 289f
Tweed, Charles, 24, 24f
Two-jaw surgery needs, 801
Two-segment maxilla segmentation, 456f-458f, 458

U

UAL. *See* Upper airway.
UARS. *See* Upper airway.
UCLP. *See* Unilateral cleft lip/palate (UCLP).
U.K. Index of Treatment Need, 65
Undermining resorption, 141, 180
Unilateral cleft lip/palate (UCLP), 1297-1370. *See also* Cleft-orthognathic surgeries.
coordinated team approach, 1302-1304
facial growth implications, 1297-1302
historical perspectives of, 1297
in infants, 1297-1302
jaw deformities and, 1356-1357
orthodontic approaches, 1356-1357
orthognathic approaches, 1299f-1305f, 1307f-1312f, 1317f-1354f, 1357-1359
controversies of, 1363-1365
distraction osteogenesis (DO) and, 1365
evolution of, 1357-1358
initial surgical healing, 1360
Le Fort I osteotomies, modified, 1358-1359
Le Fort I osteotomies, standard, 1358
Le Fort I osteotomies, two-segment, 1358-1359

Unilateral cleft lip/palate (UCLP) *(Continued)*
maxillary reconstruction, staging of, 1363-1364
mixed dentition and, 1364
pitfalls of, 1359-1361
skeletal relapse and, 1364-1365
skeletal stability and, 1362-1363
studies of, 1361-1363
unresolved issues of, 1363-1365
velopharyngeal (VP) function and, 1364
presurgical assessments, 1357
treatment protocols, 1299f-1305f, 1304-1306, 1307f-1312f, 1317f-1354f
Unilateral condylar hyperactivity, 860-861
Unilateral maxillary crossbite, 271-272
Unilateral oto-mandibular dysostosis, 1095. *See also* Hemifacial microsomia (HFM).
Unstable *vs*. unreliable centric relation (CR), 427. *See also* Centric relation (CR).
Unvoiced sounds, 235
Upper airway, 995, 995f
length (UAL), 999
resistance syndrome (UARS), 994
surgery, 1010-1045
Upper face reference
maxillary cant, 361-362
maxillary dental midline, 361
maxillary incisor crown vertical position, 362-363
maxillary incisor crown horizontal position, 363-367
Upper helix, failure *vs*. relapse, 1730-1731
Upper midface deformities, 1232
UPPP. *See* Uvulopalatopharyngoplasty (UPPP).
U.S. National Health and Nutrition Examination Survey III (NHANES III), 64
Uvulopalatopharyngoplasty (UPPP), 1010-1045

V
Valium, 75
van der Wound syndrome, 77-91, 84f
Vascular complex malformations, 1277-1288. *See also* Complex malformations.
arteriovenous, 1280-1282, 1286f-1287f
capillary, 1288
characteristics of, 1277, 1280f-1287f
complications of, 1277
historical perspectives of, 1277
lymphatic, 1277-1279, 1282f-1283f
venous, 1279-1280
Vascular thrombosis, 180
VCFS. *See* Velocardiofacial syndrome (VCFS).
Vector-controlled forces, 137
Vectored force, 406
Velocardiofacial syndrome (VCFS), 1288. *See also* Complex malformations.
characteristics of, 1288
pathophysiology of, 1288
prevalence of, 1288
reconstruction in, 1288
Velopharyngeal (VP) dysfunction, 1428, 1443, 1444f-1445f
Velopharyngeal insufficiency (VPI), 254-259, 255f-258f
Ventilation, face-mask, 319
Vertical dimensions, 64
Vertical discrepancy, chin, 1565f-1571f, 1571-1572, 1573f-1593f. *See also* Chin.
Vertical maxillary excess patterns, 758-806. *See also* Long face patterns.
clinical characteristics of, 759
complications of, 802
definitive reconstruction for, 761-801
dental findings of, 759
diagnostic criteria for, 758
etiology of, 759-760
functional aspects of, 758-759
growth modification, preadolescent patients, 760
informed consent considerations, 802
orthodontic camouflage approach to, 760
orthodontic molar intrusion approach to, 760-761
patient education for, 802
postsurgical malocclusion, 802-803
postsurgical orthodontic maintenance, 802
prevalence of, 758, 803
radiographic findings of, 759
surgical planning for, 801-802
Virenque, Maurice, 53-55
Vision restoration, 12, 15f
Visual distortion speech errors, 237-240
Visual examinations, direct, 408
Vitamin D, 75
Vitamin D_3, 142-143
Vitruvian Man (da Vinci), 344f
Vitruvius, Marcus Polio, 342, 342f
Vocal tract variables, 236
Vomiting management, 324
Von Recklinghausen type neurofibromatosis, 1273-1274
Vowel sounds, 236
VP dysfunction. *See* Velopharyngeal (VP) dysfunction.
VPI. *See* Velopharyngeal insufficiency (VPI).

W
Walker, Robert V., 41-42, 41f
Wassmund, Martin, 30-32, 39
Weak *vs*. strong chin, 1564-1571. *See also* Chin.
Wet bonding, 162
Whipple, J.W., 26-28
Width, biologic, 175-176
Window-period donations, 313
Wires, arch, 162
Wound healing risk factors, 982

X
Xylocaine, 1710

Y
Yaw orientation, 361
Young adult/teenage dentofacial disharmonies, 897-944. *See also* Dentofacial disharmonies.
characteristics of, 942
clinical characteristics of, 898
dental findings for, 898
functional aspects of, 898
orthodontic approach, 898
historical perspectives of, 899
studies of, 899-940
techniques, 900f-939f
prevalence of, 897
radiologic findings for, 898-899
surgical approaches, 940-941
complications of, 941
informed consent and, 941
malocclusion after, 941-942
patient education for, 941
postsurgical considerations, 941
temporomandibular disorders (TMDs) and, 942
treatment pitfalls of, 899

Z
Zeising, Adolf, 349, 349f
Zygomatic reconstruction, 1072, 1073f-1083f, 1111-1113, 1112f